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A HANDBOOK OF
MEDICAL DIAGNOSIS

IN FOUR PARTS

I. MEDICAL DIAGNOSIS IN GENERAL
II THE METHODS AND THEIR IMMEDIATE RESULTS
III. SYMPTOMS AND SIGNS
IV. THE CLINICAL APPLICATIONS

*FOR THE USE OF PRACTITIONERS
AND STUDENTS*

BY

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Physician to its Hospital; Physician to the Pennsylvania Hospital; Physician
in Chief to the German Hospital, Philadelphia.

427 TEXT ILLUSTRATIONS AND 14 FULL PAGE PLATES
FOURTH EDITION, THOROUGHLY REVISED

“The whole Art of Medicine is in Observation.”

PHILADELPHIA & LONDON
J. B. LIPPINCOTT COMPANY

To
THE MEMORY OF MY
FATHER
ELLWOOD WILSON, A.M., M.D.

PREFACE TO THE FOURTH EDITION

Three years have elapsed since the last edition of this work was issued. Many minor changes and the addition of much new matter have been necessary. To present recent advances in diagnostic methods and their practical application, several sections have had to be rearranged and many parts recast.

The consideration of the electrocardiograph has been transferred to its proper place among instruments of precision, the section of Diagnosis by the Röntgen rays has been entirely rewritten by Prof. W. F. Manges with new radiograms, and the nosological classification of Brill's Disease as a sporadic form of typhus fever has been accepted in accordance with the results of the most recent clinical and laboratory findings. The macroscopic agglutination test in enteric fever, the luetin test in the diagnosis of tertiary syphilis, the Abderhalden diagnosis of pregnancy, the functional diagnosis of renal efficiency and the complement-fixation test in the diagnosis of gonorrhœa have been duly noted. In the section on Diseases of the Ductless Glands, the subject of Dyspituitarism and Cushing's arrangement of its clinical types have been added and attention called to the importance of the polyglandular syndrome. The paragraphs relating to the etiology of acute poliomyelitis have been rewritten in accordance with the results of the experimental work of Flexner and his associates at the Rockefeller Institute.

A number of new illustrations have been inserted. The endeavor to bring this edition into close correspondence with present-day knowledge and experience has given me constant pleasure and satisfaction.

I am still unwilling to yield to the suggestion to make larger use of diagrams and parallel column tables. Really useful knowledge of clinical phenomena for the purposes of diagnosis must be, like the phenomena themselves, not rigidly conformed to type but adaptable and plastic. The diagram that does not and cannot be made to correspond to the facts of nature and the parallel columns of symptoms and signs that do not indicate the variants from type in disease are snares and pitfalls to the reader, and the fewer of such devices that are obtruded upon his attention the better.

The index, rightly called by Fuller (1650) a most important part of a book, "if not for need for speed of what they desire to find," has been again remade, with many additional references but with little increase in the number of pages.

My thanks are due to Dr. Creighton H. Turner for efficient assistance in seeing the work through the press and to Messrs. J. B. Lippincott Company for untiring and generous coöperation.

J. C. Wilson.

Philadelphia, January, 1915.

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PREFACE TO THE FIRST EDITION

THIS volume has been written partly in response to the wishes of some of my professional colleagues, partly to meet the urgent demands of many successive classes of pupils, but chiefly in the hope that at this time a convenient and practical presentation of the subject of Medical Diagnosis will prove useful to the profession at large.

The treatment of the subject-matter under four main headings has been adopted with the view of simplifying the arrangement of the topics in a department of medicine which has attained large scope and insistent importance. It is the confident expectation of the author that this plan will fulfil the twofold requirement, that, within the compass of a single book, clinical phenomena, on the one hand, and, on the other, those complexes of clinical phenomena which constitute diseases, are brought into correlation in such a manner that the practitioner who seeks information upon an obscure case may at once turn to the discussion of the methods available to clear it up, and the student may find the definite clinical applications of the same methods and their results in descriptive medicine.

Practical rather than theoretical considerations have been held constantly in view alike in the treatment of the clinical and the laboratory subjects. To attain this end a degree of positiveness of assertion not warranted under other circumstances and the avoidance of the discussion of moot and unsettled questions have seemed proper.

The Medical Diagnosis of J. M. Da Costa was published in 1864. That brilliant contribution to the literature marked an epoch in the progress of internal medicine. From the time of its appearance the traditional conception of diagnosis by intuition—a gift of the favored few—ceased to occupy the thoughts of medical men, and the subject ranged itself among the arts based upon scientific facts. It maintained in successive editions during the life of its distinguished author its position in the forefront of the progress of applied medicine during a period of extraordinary advancement in the collateral sciences upon which the practice of medicine rests. The continuing rapid development of knowledge relating to the facts of medicine in the last decade has rendered necessary fresh presentations of the subject, and from time to time excellent works have appeared. These differ greatly among themselves, according to the views of their several authors, in method and detail. To add to this honorable list demands the justification of something different in method, new arrangement of detail, and the presentation of the whole subject in accordance with the requirements of contemporary medicine. It is hoped that in the present volume these demands are fulfilled. It is the outcome of many years devoted to work in the wards, with the controlling side-lights upon bedside diagnosis afforded by the clinical laboratory, revelations at the hands of surgical colleagues in the operating theatre and confrères in pathology in the post-mortem room, the frequent opportunity of seeing unusual and grave cases in consultation, and long experience as a teacher. Such a career arouses enthusiasm but begets caution. It does not encourage in any way the belief that diagnosis in medicine is an easy matter, but forces the conclusion that it is often difficult and in rare instances impossible. For this reason and because we are always eager to extend the boundaries of our knowledge, this art is as absorbing as it is useful.

In the making of a handbook of this kind it is necessary to draw at every step upon the great fund of acquired information which has become the common property of the profession. To those whose contributions have formed that fund and to those who are daily adding to it I tender grateful acknowledgment for its use. I have mentioned by name those to whose work I have especially referred, but, as a general rule, it has been impracticable for want of space to append systematic references to the literature.

The illustrations are in large part drawn from personal observations. They have been selected solely with the view to elucidate the subject in hand. Diagrams have been employed when this method of presentation has appeared desirable, and the free use of clinical charts constitutes an important feature of the work.

To the friends and fellow-workers who have rendered—some small, some larger, but all generous and willing—assistance, I desire to express my thanks. The list includes many colleagues in the hospitals with which I am connected, some who were and others who still are resident physicians. It includes also Mr. Wilbert and Drs. Bachmann, Manges, Rosenberger, Rowntree, Royer, White, W. R. Wilson, and J. Leslie Davis. To Drs. de Schweinitz, Welch and Schamberg, T. M. Rotch, Packard, Piersol, Young, Emerson, Dudley Fulton, and many others, together with their publishers, I am indebted for permission to use illustrations. The pages on the diagnosis of diseases of the eye were written by Dr. Sweet; those on the stomach and intestines mainly by Dr. Gwyn; those on the nervous system by Drs. James Hendrie Lloyd and the late William Pickett; those on X-ray diagnosis by Dr. Moore, and those on the examination of the blood, urine, sputum, and other fluids by Dr. F. J. Kalteyer. The excellent drawings, plates, and other illustrations made by Messrs. Schmidt and Faber add much to the usefulness of the book.

I am under special and lasting obligation to Dr. Kalteyer for his most able and untiring aid while the work was in press, and to the publishers for their generous coöperation at every stage in its making.

J. C. WILSON.

PHILADELPHIA, September, 1909.

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A HANDBOOK
of
MEDICAL DIAGNOSIS

PART I.

OF MEDICAL DIAGNOSIS IN GENERAL.

I.

GENERAL CONSIDERATIONS.

DIAGNOSIS in medicine is the art or process of distinguishing between different diseases. It occupies a position related on the one hand to etiology—that science which has for its object the study of the causes of disease—and on the other to therapeutics—the art of healing. To recognize a disease involves the consideration of its causes, and if they can be corrected or removed, points the way to a cure—*causa sublata tollitur effectus.* Even when the causes are beyond our control or the lesions which they have produced are permanent, a knowledge of the true nature of the malady may enable us to select judiciously the therapeutic measures by which are brought about those adjustments which relieve suffering and prolong life. The maxim, "*qui bene diagnoscit bene curat,*" is not without truth. Finally, a correct diagnosis is essential to a reasonable prognosis, since by this means only can we foretell the probable course of a disease, whether it tends to recovery, to continuing disability, or to death.

Diagnosis is of fundamental importance in scientific medicine. The prevention of disease and the healing of the sick constitute the goal of medicine, but diagnosis is the course by which that goal is to be reached. Empirical systems ignore alike the causal and the pathological basis of disease and content themselves with the study and treatment of symptoms, and all practice tends to degenerate into charlatanism in proportion as it allows itself to be betrayed into this delusion. Rational medicine, on the other hand, regards symptoms primarily as clues to a diagnosis, only secondarily as indications for treatment; and treatment itself as efficient when it is causal or radical, and as a makeshift when it is simply palliative or symptomatic. When pain is present we seek by the methods of diagnosis to find the cause of it and to relieve it by the removal of the cause, and are not content simply to relieve the pain without regard to the underlying condition which produced it.

The art of diagnosis is important not only because of its practical utility, but also because it deals with the facts of nature. Hypotheses and theories in regard to disease come and go, nosological arrangements change and shift like the colors in the kaleidoscope, therapeutic fashions rise and fall, but the facts gained by close and constant observation belong to science and are changeless, and these are the facts with which diagnosis is concerned. It has been said that the whole art of medicine is in observation. It is certainly true that the art of diagnosis is in observation. Errors occur far more commonly from incomplete observation than from want of knowledge. A systematic, patient, painstaking study of the facts is essential to success.

The requirements of this branch of medicine are most varied and exacting. A knowledge of anatomy, and especially of visceral and regional anatomy, is essential. The variations in the size and position of the organs within the limits of health must be known. The structure and relation of the parts entering into the formation of the nervous system must be mastered. The physiological functions of the complex human organism are to be familiar knowledge. The causes of disease, both those belonging to the outside world and those developed within the body itself, and the susceptibilities which vary at different periods of life and under different circumstances, must be thoroughly understood. Changes produced by pathogenic factors must be clearly known. In truth, the facts of pathology and semeiology and the natural history of the diseases constitute the basis of diagnosis.

Hence, in the arrangement of medical studies, diagnosis is properly taken up after the student has made advanced progress in the fundamental branches, and the success of the practitioner in this field of medicine is dependent upon close habits of observation, accurate knowledge, and large experience. A judicial temperament and the ability to weigh evidence and assign due relative value to the factors in clinical problems are essential. Not less important are patience and a systematic procedure in all cases. Equally essential are correct habits of reasoning, since without these a faulty conclusion may follow accurately observed facts. The diagnostician in the broad field of clinical medicine must frequently turn for assistance to his professional colleague, who is familiar with the facts of the more restricted specialties and has mastered their technic, and he is becoming with advancing knowledge more and more dependent for accurate results upon instruments of precision and the clinical laboratory. Finally, the diagnostician should not be without imagination. Making use of his knowledge of anatomy and morbid anatomy he should cultivate the habit of picturing to himself the changes in the organs of the body by which clinical phenomena are brought about, such as the consolidated lung in pneumonia, the fibrinous exudate or effusion in pleurisy, the impacted gall-stone in biliary fever, the thrombus in phlebitis, the clot and its location in cerebral apoplexy; and in order that this habit of forming at the bedside, by a process of projective imagination, mental pictures of structural conditions hidden from the eye may be developed to the greatest extent, he should avail himself of every opportunity of witnessing operations involving the cranium, thorax, and abdomen, and of being present at post-mortem examinations.

The object of diagnosis is not merely to find a name for a morbid condition or symptom-complex. This it does, it is true, but in doing so it determines the condition of the patient as an individual, the intensity of the pathological process, the importance of prominent symptoms, the presence or absence of complications or intercurrent diseases, and in acute maladies the ability of the organism to withstand the attack. A correct diagnosis enables us to determine whether the condition of the patient is due to causes still operative or the result of influences that have ceased to act; whether or not his malady is self-limited, and, by collating the facts of any given case with the general knowledge of the profession, to form an opinion as to the probable duration of the sickness and its ultimate outcome. It informs us whether the prominent symptoms are the direct manifestation of an independent morbid process, as in gonorrhœal arthritis, the expression of a constitutional susceptibility, as in rheumatic fever, or an acute outbreak of a persistent condition, as in podagra. It enables us to recognize primary and secondary morbid processes and to distinguish between them, as in appendicitis and peritonitis, and to perceive the relation between associated visceral lesions due to the same cause, or to an extension to the neighboring organs, as in the case of left-sided pleurisy with pericarditis. It takes into consideration the hereditary tendencies of the patient, his age, surroundings, occupation, mode of life and habits. Diagnosis is clearly the only basis for rational therapeutics and reasonable prognosis. The medical sciences deal with diseases, the art of diagnosis with individuals. Disease is not an entity, but the sum of the phenomena of the reaction of the organism to pathogenic influences.

There are various methods of diagnosis, all of which may be included under the two general groups of DIRECT and INDIRECT DIAGNOSIS.

Direct Diagnosis.

A direct diagnosis is made when the history of the case and the clinical phenomena are sufficient to warrant a positive conclusion. The history of a violent prolonged chill, followed by high fever and pain in the chest, with cough, rusty sputum containing pneumococci, dulness upon percussion in the affected area, crepitant râles, and bronchial breathing, justify a direct diagnosis of croupous pneumonia. The previous history of the attack is not always necessary, the foregoing associated symptoms and signs being sufficient for the diagnosis of pneumonia even when the patient is delirious or too ill to give an account of himself.

The direct method is sometimes described as the semeiological method. The diagnosis is based upon the clinical phenomena of the disease and is reached by analysis and induction. When the data are adequate it is altogether the most scientific and satisfactory method.

Indirect Diagnosis.

The indirect method must be employed when the clinical phenomena are obscure or insufficient for a direct diagnosis. The results are not always conclusive and the diagnosis may remain for a time one of probability. This method includes differential diagnosis and diagnosis by exclusion.

THE DIFFERENTIAL METHOD is based upon the recognition of the essential phenomena by which one disease may be discriminated from others of a group presenting similar manifestations. A young person may present himself complaining of the following symptoms: Loss of flesh and strength, occasional irregular chills, followed by fever and sweating, shortness of breath upon exertion, cough and pain in the chest with scanty expectoration. Upon inspection the respiratory movement of the right side is diminished. The right thorax is found to be enlarged and altered in contour. There is faint cyanotic discoloration with œdema in the infra-axillary region. The heart is displaced to the left and the lower border of the liver downward. Vocal fremitus is enfeebled. There is marked dulness upon percussion over the lower part of the chest, continuous with the liver dulness, while the percussion note over the upper portion has a slightly tympanitic quality. Upon auscultation the vesicular murmur is faint and distant. Neither râles nor friction sounds are heard. The greater number of these symptoms and physical signs may be encountered in (a) abscess of the right lobe of the liver, (b) malignant disease of the pleura, (c) serofibrinous pleurisy, (d) empyema.

(a) Abscess of the right lobe of the liver is comparatively rare. There is frequently a history of dysentery or other disease of the abdominal viscera. The pus collection is rarely sufficiently large to displace the heart.

(b) Malignant disease of the pleura is likewise a rare affection. It usually develops insidiously without pain. It is not attended by chills or fever and does not displace the heart or liver until the growth has attained unusual proportions. It produces a profound cachexia and usually involves rather than compresses the lung, so that tympany in the upper part of the lung is absent and irregular patchy dulness is elicited over the seat of the growth.

(c) Serofibrinous pleurisy does not usually give rise to fever or, even when massive, to disturbance of the circulation of the wall of the chest or œdema.

(d) The essential phenomena by which, when present, empyema may be discriminated from the foregoing affections, in addition to the signs of compression of the lung and displacement of adjacent organs, are chills, fever, sweating, and cyanosis and œdema of the chest wall.

DIAGNOSIS BY EXCLUSION differs from differential diagnosis only in its scope. It seeks to establish the nature of the disease by the negative process of showing what it is not. The various diseases presenting similar clinical phenomena are compared in turn with the case under consideration, and one after another excluded, the diagnosis of that disease being finally made to which the malady most closely conforms. In the above example we should first set aside abscess of the liver, then malignant disease of the pleura, then serofibrinous pleurisy, and by exclusion arrive at the diagnosis of empyema. Diagnosis by exclusion is a tedious and inconvenient method, not, however, without value in difficult and obscure cases. It may be employed with advantage in clinical teaching. Other methods are:

CAUSAL OR ETIOLOGICAL DIAGNOSIS.—The nature of an obscure malarial disease with or without fever may be determined by the discovery of the æstivo-autumnal parasite in the blood, or the tuberculous basis of impaired health with cough and obscure physical signs may be revealed by an examination of the sputum. When such a diagnosis concerns germ diseases it is spoken of as Bacteriological Diagnosis.

HÆMATOLOGICAL DIAGNOSIS.—This may depend upon (a) the specific agglutinating properties of the serum, as in enteric fever or dysentery; (b) the morphology, as in pernicious anæmia or leukæmia; (c) the presence of parasites, as in malaria or trypanosomiasis; or (d) the result of cultures, as in enteric fever or septic conditions.

A PROVISIONAL DIAGNOSIS is that which best accords with the sum of the probabilities when the data are insufficient, or pending a further investigation of the facts. Such a diagnosis may serve as a working hypothesis for therapeutic purposes and the general management of the patient. It can be revised or confirmed.

A SURGICAL DIAGNOSIS is made from the stand-point of the surgeon, and may in proper cases be confirmed or set aside during the life of the patient by an exploratory operation.

FUNCTIONAL DIAGNOSIS is the determination of the degree of the impairment of the functions of organs caused by local affections or the extent of the interference with physiological processes resulting from general disease, and the bearing of such impairment or interference upon the future of the individual as regards health and prolongation of life. Functional diagnosis is closely allied to prognosis.

A THERAPEUTIC DIAGNOSIS is that procedure by which in obscure cases the nature of the disease is determined by the results of treatment. This method is of very limited application. A provisional diagnosis of malaria having been reached by the process of exclusion, the patient may be put at rest and quinine administered in proper doses. Should the symptoms promptly disappear, the diagnosis of malaria becomes probable. A similar diagnosis of syphilis having been reached by analogous methods, the subsidence of symptoms upon the administration of mercurials or the iodides may in some cases confirm the diagnosis. In almost all such cases there are other and better methods of diagnosis which may be employed concurrently with the treatment. In grave or urgent cases it is, however, better to give the patient at once the benefit of the doubt.

CLINICAL DIAGNOSIS is the diagnosis made at the bedside.

ANATOMICAL DIAGNOSIS is the diagnosis made by the pathologist in the post-mortem room.

It is not in all cases possible to make a positive diagnosis at once. Time may be required for a more thorough investigation of the history of the case, a closer study of the patient's surroundings, repeated observation, or for the report of examinations conducted in the clinical laboratory. Information bearing upon the previous history of the patient or the beginning of his illness cannot always be obtained. He may be delirious, unconscious, or may have lost the power of speech. The history communicated by his friends is often uncertain and misleading. Persons of the lower classes are very commonly indifferent to symptoms which are not painful or disabling and lack the ability to describe their sensations. Many persons, on the other hand, often intentionally, sometimes unconsciously, make false statements in regard to their past life and present symptoms. Some parts of the narrative are exaggerated, others suppressed. Symptoms may be imitated and superficial lesions artificially produced. Hence a group of feigned diseases, against which the physician must be upon his guard.

MALINGERING.—The term malingerer is used to describe one who intentionally simulates a disease. Malingering occurs in every grade of life and under various circumstances. It is to be suspected when a simulated disease lacks essential symptoms or its picture is overdrawn, and when there is lack of correspondence between the alleged symptoms and the actual signs or the obvious general health; it is to be detected by close study of the case under various conditions, by the use of instruments of precision, and in some cases by the application of powerful faradic currents or an examination under anæsthesia. The over-indulged child, to avoid his lessons or escape punishment, may feign an illness; an older person, to excite compassion or from mere love of deception. It is common among beggars, sailors and soldiers, those improperly seeking pensions, and claimants against corporations for accidental damages. The simulation of disease is, however, not always intentional. Hysterical and neurasthenic individuals sometimes exaggerate symptoms or imitate the manifestations of disease without purpose or intention—the unconscious mimicry of disease. There are those, on the other hand, who from motives of delicacy or shame, or in consequence of natural reserve, or from fear of having their apprehensions confirmed, refuse to consult the physician, or when forced to do so give a garbled and incomplete history of their sickness. This may occur among those suffering from venereal diseases or chronic diseases popularly regarded as incurable, as tuberculosis and cancer.

The diagnosis of an obscure case occasionally demands an investigation of the surroundings of the patient at the time of the development of the illness. Time may be required to ascertain etiological conditions relating to his food, drink, occupation, endemic influences, or exposure to transmissible diseases locally epidemic. Questions of this kind frequently arise at a period like the present, when facilities for commercial intercourse are increasing and when military operations and the exigencies of trade have greatly extended travel to all parts of the world.

Repeated examinations may be necessary in order to obtain accurate impressions when the physical signs are obscure or ill defined. Excessive subcutaneous fat, local œdema, or general anasarca may interfere with the physical exploration; or local tenderness, intense pain, great restlessness, or an unwillingness on the part of the patient to submit to an examination may give rise to delay. In other cases the unusual character of the symptoms or an association of clinical phenomena not previously encountered may render repeated examinations necessary. During the stage of invasion in the acute febrile infections a positive diagnosis is often impossible.

The advances of modern medicine have enormously increased our knowledge of diseases and the precision of diagnosis. In all departments of clinical medicine, scientific accuracy has taken the place of probability. The every-day routine examinations of the clinical laboratory cannot be made off-hand. The more elaborate investigations involved in obscure cases demand technical skill and a reasonable time. The reports are necessary to a final diagnosis. An immediate diagnosis is not only not necessary, it is very often not possible. Haste involves the risk of error. Conclusions cannot be reached until the premises are established. A provisional diagnosis may serve to meet the immediate requirements of the situation. Treatment may be instituted in response to urgent indications. When in the period of invasion of an acute illness there is reason to suspect a transmissible disease, such as scarlatina or variola, the same measures of prophylaxis should be instituted pending the evolution of the process that would be employed if the suspected disease were actually present.

There are cases in which diagnosis in a broad sense is impossible. A name may be given to some prominent symptom or group of symptoms, but the essential pathological process may remain obscure until its nature is revealed upon the post-mortem table.

When possible a positive diagnosis should be made at once; in all cases as soon as practicable. The student is, however, warned against making any but a provisional diagnosis upon insufficient data. To ask for delay is by no means a confession of ignorance; on the contrary, it is the course dictated by knowledge and experience. Intelligent people, who seek the best professional advice, fully understand this. It is only the ignorant who are satisfied with a phrase for diagnosis, a prescription dashed off at sight and no directions whatever, who insist upon being told what is the matter at once.

II.

MEDICAL TOPOGRAPHY.

MEDICAL TOPOGRAPHY is that branch of diagnosis which has for its object the consideration of the boundaries and relations of the external parts and internal organs of the body. Various points, lines, and regions or areas, some artificial, others natural, serve the purposes of this method of clinical investigation.

THE HEAD.

The head is divided by anatomists into two parts, the cranium and the face.

The Cranium.

The skull encloses and protects the brain. It is divided into regions corresponding with the superficial bones which enter into the formation of the skull,—namely, occipital, parietal, frontal, and temporal. These regions are separated by the cranial sutures. Opposite the angles of the parietal bones are spaces called fontanelles,—*fons*, a fountain,—which remain unossified after the bony growth of the skull is elsewhere completed. Of these, two in the median line, the anterior and posterior fontanelles, are important.

The regions of the skull serve for the localization of subjective sensations, as pain or headache, and superficial lesions, as craniotabes, nodes, nævi, injury, or suppuration. The mastoid process of the temporal bone is an important landmark, as indicating the extension of middle-ear disease. The greatest convexity in the frontal region on either side is known as the frontal eminence. It is separated by a slight depression below from the superciliary ridge, at the level of which in the median line is the nasal eminence or glabella. About the inner third of the orbital arch is the supra-orbital notch or foramen, a point of tenderness in supra-orbital neuralgia.

Sutures.—Failure on the part of the cranial bones to unite, with persistent wide sutures, may be due to hydrocephalus, cretinism, or in very rare instances to antenatal rickets.

Fontanelles.—VARIATIONS IN PROMINENCE.—Bulging of the fontanelles is a common symptom in infants and young children. It is much more marked in the anterior fontanelle. When persistent it indicates organic diseases of the brain, as hydrocephalus, meningitis, or intracranial hemorrhage, which is in infants far more commonly meningeal than cerebral. When transient it is usually pulsating and associated with high temperature and other symptoms of an acute febrile infection.

Retraction of the fontanelles occurs in chronic wasting diseases, as tuberculosis, infantile atrophy or marasmus, and colitis, and in acute diarrhœal affections, as enterocolitis and cholera infantum.

Variations in Size.—The posterior fontanelle is normally obliterated about the sixth week. The anterior remains patulous as at birth or even slightly increases in size up to about the ninth month, and closes before the end of the second year. Delay in closing beyond this period is commonly associated with wide and ununited sutures and occurs in rickets and hydrocephalus. The diameter of the anterior fontanelle at the end of the first year is normally about 2.5 centimetres. A greater width occurs in rickets and some cases of congenital syphilis. A very wide fontanelle is characteristic of hydrocephalus.

The Face.

The regions of the face are the orbital, nasal, buccal, and oral. They contain the muscles of expression and are of great importance in the diagnosis of local and constitutional disease, as well as in the recognition of mental and emotional conditions. The facies in various conditions will be described in a later chapter. Changes caused by nervous and ocular disorders will be considered under their appropriate headings.

DEFORMITIES OF THE HEAD IN THE NEWBORN.

Caput Succedaneum.—A swelling of the scalp caused by pressure during parturition. The lesion consists of passive congestion with extravasation of blood and œdema of the tissues of the scalp at the area of absence of pressure, namely, the part of presentation. The tumor is irregularly circumscribed and does not fluctuate. It disappears without treatment in the course of a few days. This condition is to be distinguished from—

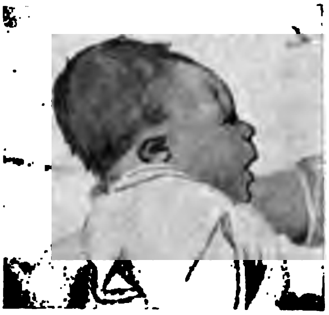


Fig. 1.—Caput succedaneum. Male, 2 hours old.—Rotch.

Cephalhæmatoma.—A tumor formed during labor by hemorrhage into the space between (a) the occipitofrontalis aponeurosis and the periosteum, or between the periosteum and the skull—external cephalhæmatoma—or (b), between the skull and the dura mater—internal cephalhæmatoma.

(a) External Cephalhæmatoma.—The most common variety is subperiosteal. It occurs in the form of an irregular, circular, flat tumor over one or, in rare instances, both parietal bones. There is distinct fluctuation, but the overlying skin is not discolored. Slight elevation of the bone at the border of the swelling may be felt in a few days, with obscure crepitus. The condition is to be distinguished from caput succedaneum by its location, fluctuation upon palpation, and the examination of fluid withdrawn by aspiration. The bony rim is diagnostic at a later period. It is not to be confounded with a depressed fracture, which is irregular in outline and lacks the distinct tumor formation with fluctuation and the rim-like bony circumference characteristic of hæmatoma.

(b) Internal Cephalhæmatoma.—A very rare condition which ends in the death of the child. There are pressure symptoms. It is sometimes associated with the external form. It has occurred in breech presentations.

Fluctuating tumors arising from the course of the cranial sutures are usually situated in the occipital region or at the glabella. Three varieties are described.

Meningocele.—This term is used to designate a hernial protrusion of the meninges through an opening in the bony cranium resulting from defective ossification or failure in suture formation. It may result from intra-uterine hydrocephalus. The tumors usually contain cerebrospinal fluid, and are translucent, with large veins upon the surface. In some instances an impulse may be felt upon crying and the tumor can be reduced by gentle pressure.

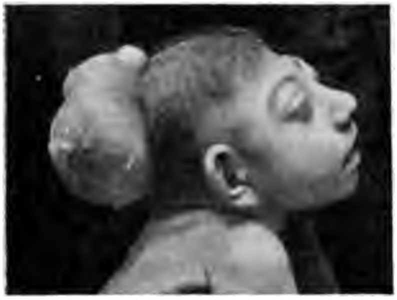


FIG. 2.—Hydro-encephalocele.—Rotch.

Encephalocele.—This form of cerebral hernia is more common. The tumor contains brain substance in addition to the membranes.

Hydro-encephalocele. — The hernial contents consist of the membranes, brain tissue surrounding one of the ventricles, and a portion of the ventricle itself distended with cerebrospinal fluid. These tumors vary in size from a walnut to a large orange and tend to increase in size. They are usually pedunculated. The prognosis is unfavorable, though remarkable recoveries have occurred after operation.

Anencephalia.—This developmental defect is rarely complete. Partial anencephalia is the usual form. In accordance with a recognized pathological law, the deficiency of contents causes microcephalic deformity of the skull.

Hydrocephalus.—Congenital internal hydrocephalus is a common cause of deformity of the skull in the newborn. The head is markedly enlarged; the cranial bones are thinned and displaced outwards; the sutures widely separated and the fontanelles prominent and fluctuating. In marked cases the temporal and parietal bones flare outward so that the cranium is more or less pear-shaped, the greatest diameter being in the upper part. The face is usually normal in size, but it looks abnormally small, being dwarfed by the great size of the head.

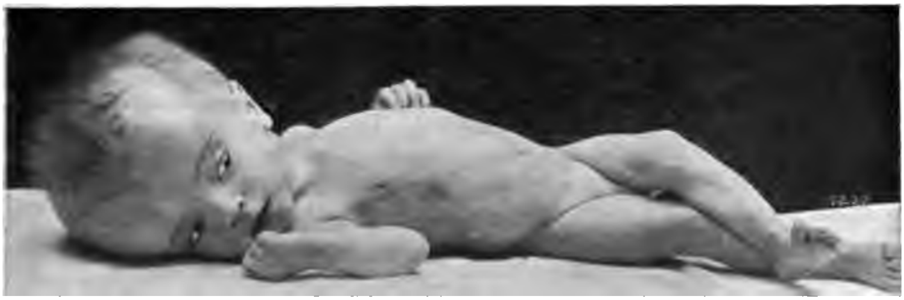


FIG. 3.—Congenital internal hydrocephalus. Male, 7 months old.—Rotch.

THE NECK.

Length and Thickness.

In early infancy the neck appears short on account of the large size of the head and its tendency to fall forward, and the relatively high position of the sternum and clavicles. The neck appears to be broad in comparison with its length also by reason of the large amount of subcutaneous fat. In fact at all periods of life the thick neck of obese persons appears short, an appearance heightened by the accumulation of fat known as the double chin.

A short thick neck and stout plethoric body constitute the chief structural factors in the so-called habitus apoplecticus. On the other hand, a long slender neck with a prominent larynx, and narrow flat chest with projecting scapulæ, are characteristic of the habitus phthisicus. But both these designations are misleading, since apoplexy is dependent upon a condition of the arteries and frequently occurs in spare persons with long thin necks, and pulmonary tuberculosis is the result of infection and not rarely selects its victims among those who have well-formed chests and necks, and occasionally among those who are stout, with short thick necks.

Contour.

Larynx.—In lean persons the larynx is prominent and forms the projection anteriorly in the median line known as the Adam's apple. In fat persons this organ is much less noticeable. Descent of the larynx upon inspiration occurs in all forms of severe dyspnœa, and especially in the spasmodic respiration which often precedes death in respiratory diseases attended with stenosis of the larynx or œdema, collapse or extensive consolidation of the lungs. Pressure displacements of the larynx and trachea sometimes result from the presence of aneurismal or other tumors of the neck. They are usually lateral. Some degree of lateral displacement may also result from pleural adhesions and the traction of a contracting lung in neglected pleurisy or fibroid phthisis. Moderate bilateral prominence and enlargement of the neck without spastic contraction occur in the habitual dyspnœa of severe chronic bronchitis, emphysema, bronchial asthma, cardiac disease and certain cases of chronic uræmia—so-called renal asthma. Rigidity of the neck is sometimes due to myalgia of the cervical muscles, spondylitis deformans involving the cervical vertebræ, or caries. It may be caused by painful inflammatory processes, as acute adenitis, parotid bubo, mumps boils, or carbuncles.

Thyroid Body.—This gland is situated in the lower part of the neck and embraces the trachea in its upper part, reaching up to the larynx on each side. It consists of two lateral lobes united by an isthmus. The right lobe is usually slightly longer and wider than the left. Both the larynx and the thyroid body which is in relation with it rise with the act of swallowing. Enlargement of the thyroid body usually affects the isthmus and both lobes, but one—very often the right—to a greater extent than the other. The enlargement may be vascular, parenchymatous, fibroid, cystic, or due to adenoma, carcinoma, tuberculosis, or gumma. Vascular enlargement of the thyroid body may be physiological, occurring during menstruation or pregnancy and subsiding at the termination of these events, or pathological, as in exophthalmic goitre, when it is often variable in size and attended with marked pulsation, thrill, and murmur. Venous hyperæmia may be due to the pressure of an aneurism or mediastinal tumor. Parenchymatous enlargement or simple goitre may be of moderate size, but in some instances attains enormous dimensions, protruding beyond the chin and hanging over the sternum. Cystic goitre when multiple may be recognized by the smooth, hemispherical, close set, elastic nodules upon the surface; when single and larger, by fluctuation. Thyroid abscess is rare and usually accompanied by local inflammatory œdema and grave constitutional symptoms. Cancer, tuberculosis, and gumma rarely involve the thyroid and may be recognized by their local characters and the associated constitutional phenomena. An underlying aneurism or mediastinal growth may displace the thyroid upwards and forwards or to either side. In aneurism of the innominate, the displacement is towards the left. An aneurism sometimes imparts its movements to the overlying thyroid. Atrophy of the thyroid may give rise to flattening of the surface. More commonly it can be recognized only upon palpation. It is usually accompanied by the symptoms of cretinism or myxœdema.

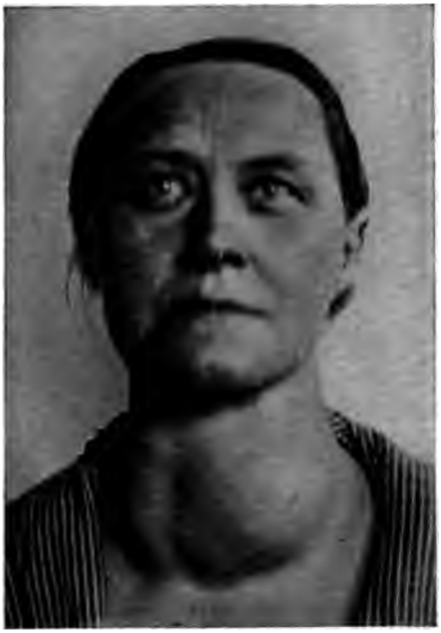
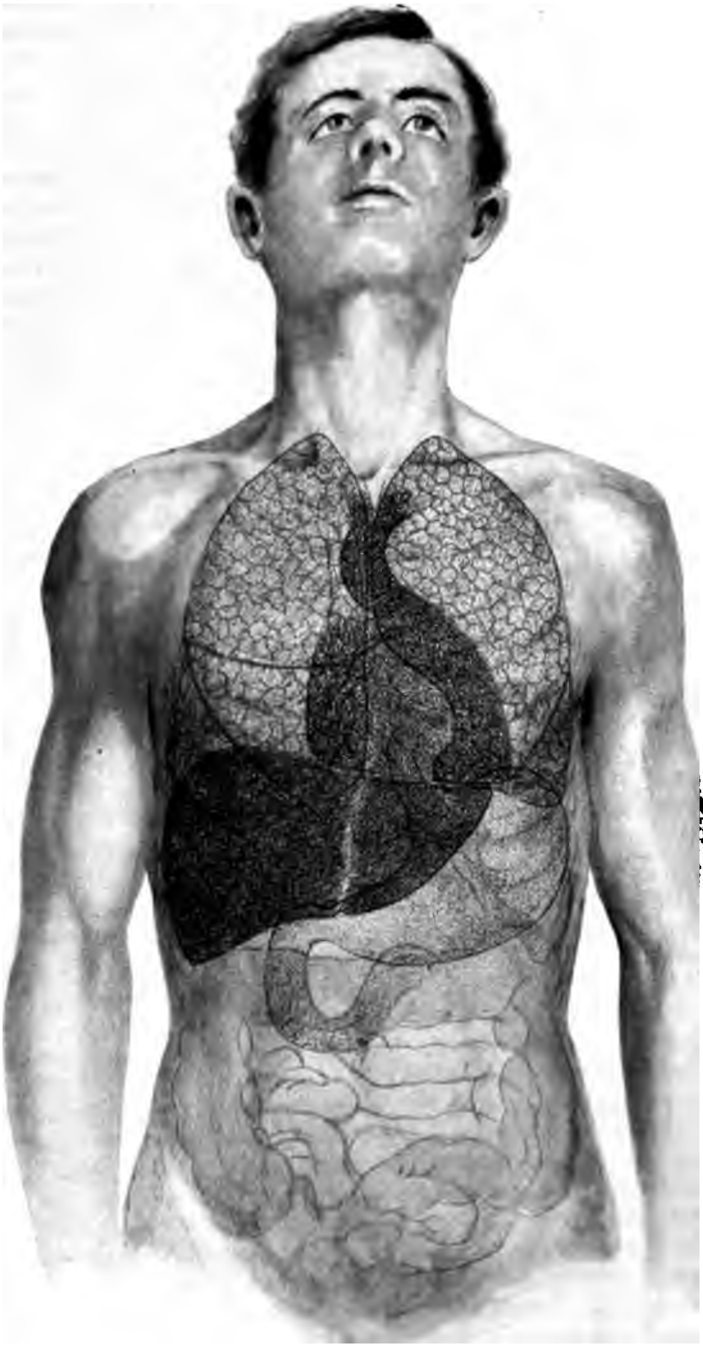


FIG. 4.—Cystic goitre.

Muscles.—One or both sternomastoid muscles may be hypertrophied and prominent. In torticollis or wry-neck the contraction is usually unilateral, and the neck is rotated so that the mastoid is drawn towards the inner end of the clavicle, the chin raised and the face turned towards the unaffected side. In rare cases wry-neck is bilateral,—retrocollic spasm, —the head retracted and the face turned upward. The spasm in both forms of torticollis may be tonic or clonic. The disease is sometimes congenital.

The Clavicles.—The position of these bones has much to do with the appearance of the neck as regards length. They are high in deep-chested persons with large lungs and in emphysema; low in flat-chested individuals with small lungs and in phthisis and pulmonary fibrosis from any cause. These bones are deformed after fracture and sometimes present nodes and irregularities of the surface caused by syphilitic periostitis. Prominence in the retroclavicular space sometimes occurs in emphysema



General anatomical outlines and relations of the thoracic and abdominal organs.

of high grade, but as a rule the clavicles in this condition are prominent and both the retro- and infraclavicular spaces are retracted. The subcutaneous fat pads of myxœdema are frequently seen above the clavicles and sometimes at the root of the neck posteriorly. The neck is occasionally the seat of extensive inflammatory œdema with violaceous discoloration—collar of brawn—especially in scarlet fever, erysipelas, and infected wounds, and sometimes much distorted by subcutaneous emphysema, such as follows rupture of the pleura or wounds or operations involving the upper air-passages.

THE THORAX.

The thorax is of conical shape with convex walls. Its truncated upper end is narrow and bounded by the first dorsal vertebra, the first pair of ribs, and the manubrium of the sternum. Its expanded base is filled in by the vault of the diaphragm. The anterior border of the base curves downwards and backwards on each side from the xyphoid cartilage to the twelfth rib. Its transverse diameter greatly exceeds its anteroposterior diameter, which is further shortened in the middle line by the projection of the spinal vertebræ forwards into the cavity of the thorax. This space contains the heart and great vessels together with the pericardium, the lungs and pleuræ, the trachea, the greater part of the œsophagus, and the thymus gland or its remnant.

Anatomical Landmarks of the Thorax.

ANTERIOR SURFACE.

The Chest.—The clavicles, sternum, ribs, and interspaces constitute natural surface conformations to which, for purposes of study and description, clinical phenomena may be referred.

The Clavicles.—The part immediately above these bones on either side is known as the supra- or retroclavicular space; that immediately below them as the infraclavicular space. Into the supraclavicular spaces the apex of the lung enters to a slight extent, usually a little further on the right than on the left side. In well-developed lungs these spaces are not retracted, but in ill-developed lungs and in pathological states characterized by contraction of lung tissue they are more or less strongly depressed.

The Sternum.—The upper border of this bone is marked by a large incurvation known as the episternal notch, which limits the root of the neck anteriorly and in which can be felt at times the pulsating aorta—dilatation, dynamic pulsation, aneurism of the transverse arch. At the line of juncture of the manubrium and gladiolus or body is a more or less prominent transverse line or prominence, better developed in the male—the angle of Ludovicius. At the lower end is the xyphoid or ensiform appendix, variable in size and shape and sometimes having its tip everted in such a manner as to form an infrasternal depression or fossa.

The Ribs and Intercostal Spaces.—THE RIBS.—In spare persons the ribs may be counted with ease. When, however, there is much subcutaneous fat, the recognition of any particular rib is sometimes difficult. The first rib may be known by the articulation of its cartilage with the sternum at a point immediately below the articulation of the clavicle. The articulation of the second costal cartilage is directly opposite the junction of the first and second pieces of the sternum—angulus Ludovici. The ribs slope downwards from their spinal articulations in such a manner that their chondrosternal articulations lie at a much lower level, the articulation of the first rib anteriorly being in quiet breathing on the horizontal plane of the fourth rib at the back and so on to the seventh rib. In the expiratory type of chest this oblique position of the ribs is somewhat increased; in the inspiratory type it is much diminished.

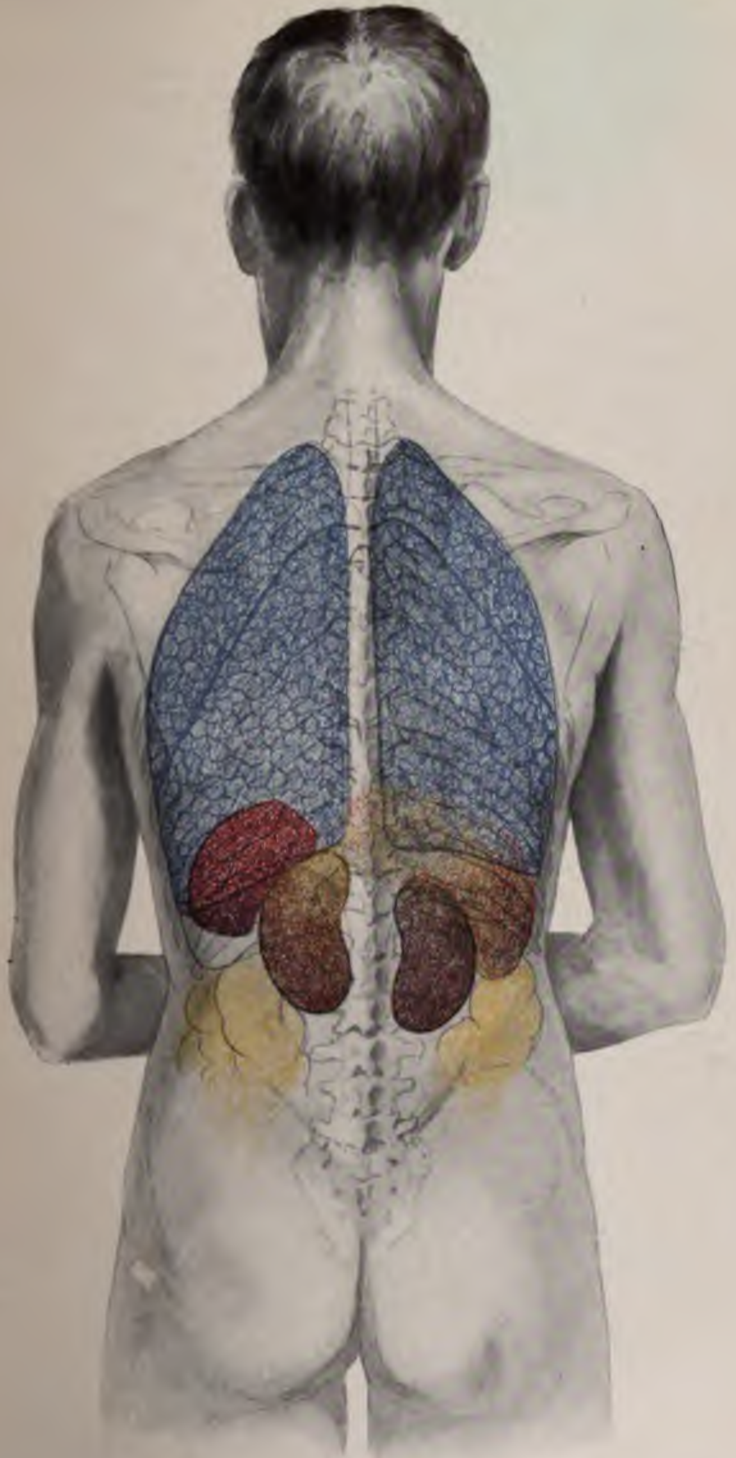
THE INTERCOSTAL SPACES.—These spaces correspond to the ribs and cartilages immediately above them—that is, the first space lies immediately below the first rib. They are wider in front than behind. In expiration the upper spaces are increased in width and the lower narrowed, while in inspiration these conditions are relatively reversed. It is in accordance with this fact that the upper spaces are wider and the lower narrower in the expiratory, while the upper are narrower and the lower wider in the inspiratory form of chest. In fat persons the intercostal spaces cannot be made out, but in those who are lean they appear as shallow, parallel, oblique furrows symmetrically arranged upon each side of the chest. They are deeper upon inspiration than on expiration or quiet breathing, and conspicuously so in obstructive dyspnœa. These furrows are obliterated in massive pneumonia and in pleural effusions and the spaces may actually bulge in old cases of large empyema. Local protrusion of the chest wall such as occurs in large hypertrophy of the heart in early life causes widening of the overlying intercostal spaces. The unilateral flattening of the chest which accompanies fibroid phthisis or follows a neglected pleural effusion, crowds the ribs together, even in some instances to overlapping, and in this manner obliterates the spaces in whole or in part.

Normal Cardiac Pulsation.—The apex beat is seen in the fifth intercostal space to the left of the parasternal line, while undulatory pulsation in several spaces occurs in dilatation of the heart, and heaving pulsation over a large area in marked hypertrophy of that organ. In rare instances intercostal pulsation is due to a neglected empyema. The pulsation is almost always in the anterolateral aspect of the chest upon the left side. In mitral and aortic stenosis, aortic insufficiency, cases of congenital malformation of the heart, and aneurism of the aorta, thrills may be felt upon palpation.

The Nipple.—This organ is not without value as a topographical landmark in children and spare males, but in women and obese persons of both sexes its position is extremely variable. When there is little fat and the mammæ are undeveloped the nipple is situated about the fourth intercostal space, sometimes over the fourth, sometimes over the fifth rib, and in a vertical line intersecting the middle of the clavicle -the mammillary line. It is obvious that the nipple — mammilla — is not a satisfactory anatomical landmark. The midclavicular line is much more useful.

POSTERIOR SURFACE.—THE BACK.

The Spine.—In children and lean persons the spinous processes are prominent. In muscular adults and fat people they are situated in the middle of a shallow longitudinal groove formed by the prominence of the



General anatomical outlines and relations of the thoracic and abdominal organs.

erector spinæ muscles on either side. They become more prominent when the patient bends strongly forward. Owing to the denseness of the overlying musculotendinous tissues, the spines of the upper five cervical vertebræ cannot as a rule be recognized upon palpation. The sixth may be felt and seen in many persons, and the seventh—vertebra prominens — is usually conspicuous and forms a point of departure from which the thoracic and lumbar spines may be counted. The eighth and ninth thoracic spines are normally somewhat more prominent than the others. Marked prominence of one or more vertebral spines, with tenderness upon pressure and pain upon rotary movements of the spine or jarring, is significant of spinal caries, usually tuberculous, rarely syphilitic. There is frequently angular curvature.

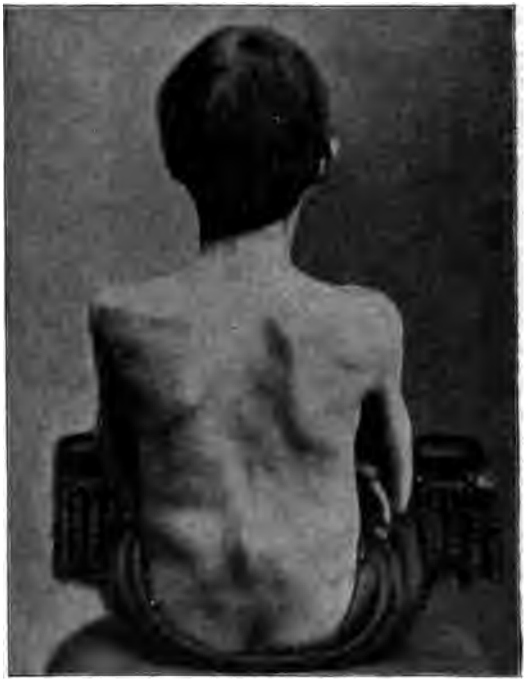


FIG. 5.—Spinal caries. Lumbar region.—Young

Kyphosis.—The curvature is in the sagittal plane with the concavity anterior. It is chiefly thoracic, sometimes cervicothoracic, and may constitute nothing more than one of the skeletal changes due to old age. It occurs also in those who habitually carry heavy burdens on the head and shoulders, in emphysema, rickets, osteitis deformans, and acromegaly. This rounded curvature is to be distinguished from the sharper, often angular curvature of vertebral caries or mollities ossium.

Scoliosis. — A rotary-lateral curvature usually involving the upper thoracic spine with compensating curvature in the lower thoracic and lumbar regions. Less commonly it affects the cervical or lumbar regions. Scoliosis is very common in school-girls in consequence of poor muscular development and faulty desk attitudes. It may result from the habitual carrying of heavy weights on the same arm, inequality in the length of the legs, deformity of a foot, tilting of the pelvis, old sciatica, the arrested growth of a limb following infantile palsy, hemiplegia, and mollities ossium. The deformity of the chest following long-neglected pleural effusion, serofibrinous or purulent, includes dorsal scoliosis, the concavity looking towards the affected side.

Lordosis.—An exaggeration of the normal lumbar curve occurs in advanced pregnancy, large abdominal tumors and ascites, progressive muscular atrophy, and pseudohypertrophic muscular paralysis. The attitude in the last condition is characteristic. The legs are separated, the head is thrown back, the spine strongly curved, and the abdomen thrust forward.

Spina Bifida.—This is a developmental fault consisting of failure on the part of the laminæ of the vertebræ to unite. The usual site is in the lumbar or lumbosacral region. The protruding tumor is in the middle line, sometimes covered with normal skin, sometimes with a thin, translucent membrane. There are two varieties:

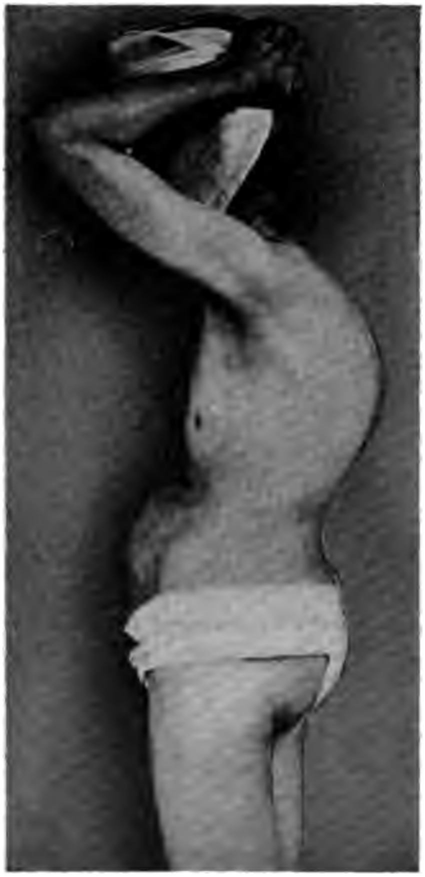


FIG. 6.—Kyphosis.

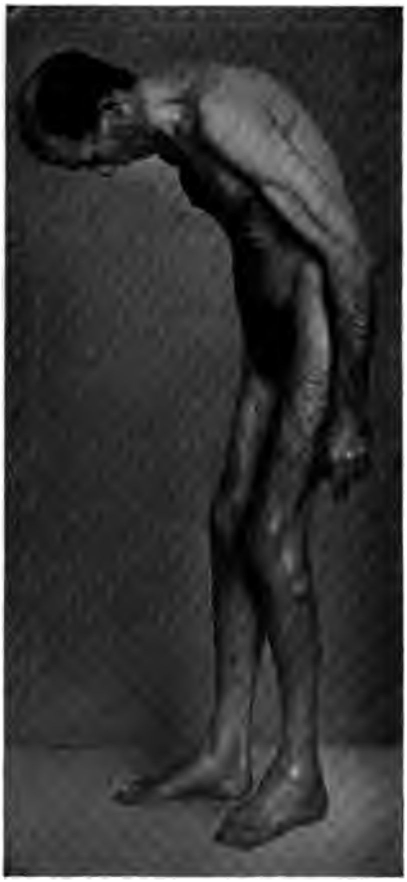


FIG. 7.—Senile kyphosis.

SPINA BIFIDA OCCULTA, in which the sac is walled off from all connection with the spinal canal, and—

SPINA BIFIDA VERA, in which the cyst is filled with cerebrospinal fluid and increases in size during violent crying, and can be diminished by gentle pressure.

Three subvarieties are recognized:

Spinal Meningocele.—The protruding membranes contain only cerebrospinal fluid.

Meningomyelocele.—The sac contains not only fluid but also substance of the cord. This is the most common form.

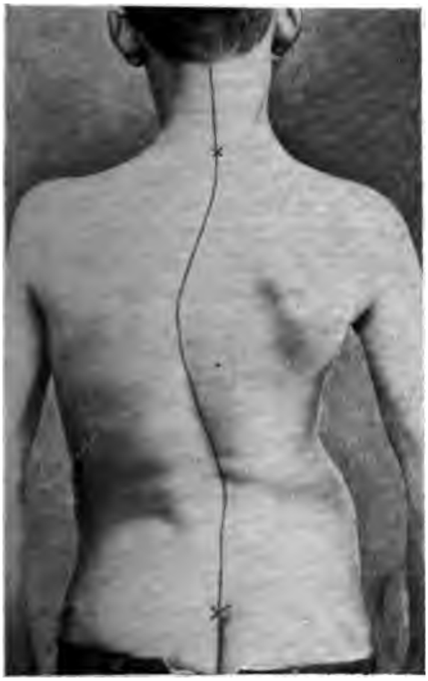


FIG. 8.—Primary left dorsal scoliosis.—Young.

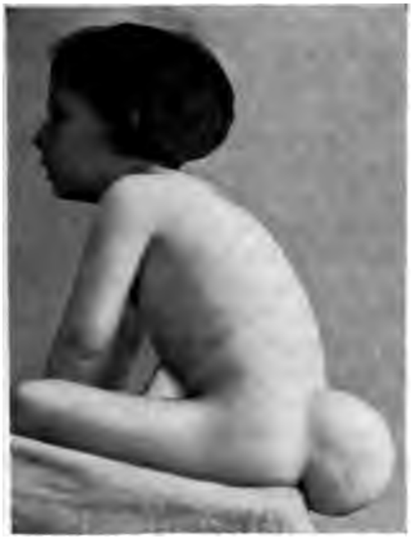


FIG. 10.—Spina bifida of lumbar region Male, 5 years old.—Rotch.

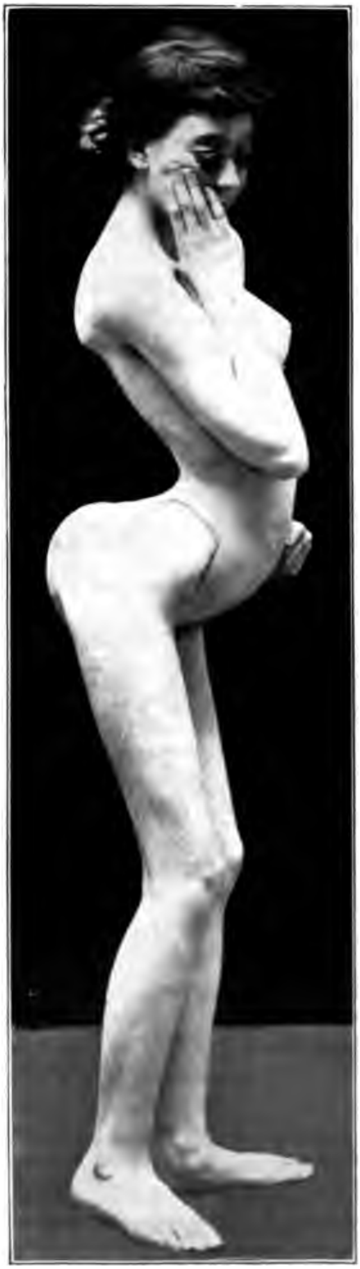


FIG. 9.—Extreme lordosis in progressive muscular atrophy.—Young.

Syringomyelocele.—The sac is formed of the membranes and a protruding portion of the cord, the central canal being dilated to form the cavity of the tumor.

This group of deformities is commonly associated with other developmental defects. Exceptionally spina bifida occurs in children otherwise healthy and well developed.

The Scapulæ.—These flat, triangular, trowel-like bones are placed symmetrically upon the upper and back part of the thorax and extend, when the arms hang by the sides in the erect posture, from the second to the seventh ribs. They are attached to the skeleton by the clavicle and the humerus and are therefore freely movable. When the arms are folded and the body is bowed forward, the interscapular space is much increased, an important fact in physical diagnosis. The inner borders of the scapulæ project in consequence of muscular weakness, palsy, and changes in the contour of the chest. Combinations of these causative conditions may occur in the same case. Both inner borders project in the alar or pterygoid chest and in the progressive muscular dystrophies affecting the shoulder girdle. The abnormal mobility of the shoulder-blades arising from loss of muscular tone permits the inner borders to project like budding wings. The inner border stands out upon the affected side in contraction of the chest from pulmonary fibrosis; in associated serratus and trapezius paralysis, especially when the arms are held out in front in the horizontal plane; in scoliosis due to various causes, and sometimes upon the left side in large aneurism of the descending portion of the arch of the aorta.

Immobility of the Spine.—Flexion, extension, and lateral and rotary movements may be restricted or wholly prevented by various pathological conditions, as (1) those giving rise to pain in movement, among which the more common are traumatism, myalgia—lumbago—abscess, carbuncle, meningeal hemorrhage; (2) those involving spasm, as cerebrospinal fever and the spastic form of myalgia; (3) those affecting the joints and bones, most of which terminate in ankylosis, as traumatic, gonorrhœal, or tuberculous disease and spondylitis deformans; and (4) certain neuroses, as many of the cases of so-called typhoid spine, railway spine, hysterical spine, irritable spine, and so on.

LATERAL SURFACES.

The landmarks are the axilla—armpit—above, the anterior and posterior axillary folds, the ribs and interspaces and the upper border of hepatic, on the right, and of splenic dulness on the left side, below. Enlarged lymphnodes, which frequently undergo suppuration or may be tuberculous, carcinomatous, leukæmic or pseudoleukæmic, are common in the axillary space.

Artificial Lines and Spaces of the Thorax.

The following conventional imaginary lines and spaces serve a useful purpose in the examination and description of thoracic lesions. For convenience of demonstration the lines may be marked upon the surface with a dermatographic pencil. The subject is in the erect posture with his arms symmetrically disposed.

A. Vertical Parallel Lines. — With the exception of the first and last they are double—bilateral.

(a) The mesial or midsternal line.
(b) The line of the sternal border.
(c) The parasternal line, midway between the line of the sternal border, and—
(d) The midclavicular line, sometimes spoken of as the mammillary line because in individuals with undeveloped mammæ it passes through or near the nipple.

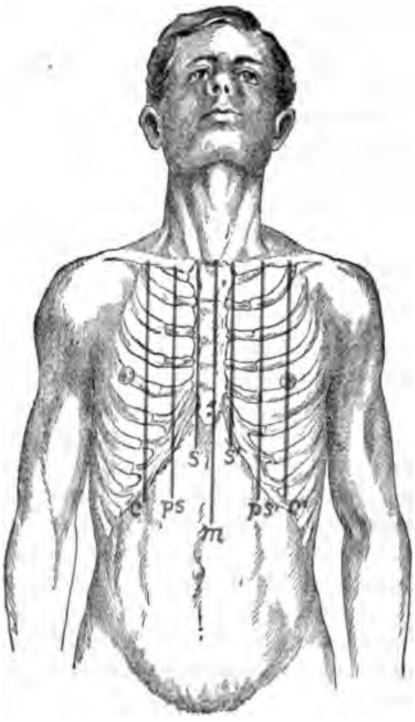


Fig. 11.—Lines of reference: Anterior.—*m*, middle line; *s*, *s'*, right and left lines of the sternal border; *ps*, *ps'*, parasternal lines; *c*, *c'*, midclavicular or mammillary lines.

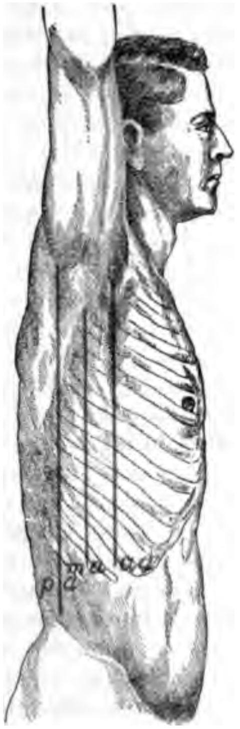


Fig. 12.—Lines of reference: Lateral.—*aa*, anterior axillary line; *ma*, midaxillary line; *pa*, posterior axillary line.

(e) The line of the anterior axillary fold.
(f) The midaxillary line.
(g) The line of the posterior axillary fold.
(h) The scapular line, passing vertically through the inferior angle of the scapula—a very movable and uncertain landmark.
(i) The posterior mesial line, corresponding to the line of the spinous processes.

B. Horizontal Parallel Lines.—These are anteriorly:

(a) A line touching the lower border of the cricoid cartilage.
(b) A line passing through the clavicles.
(c) A line passing through the third chondrosternal articulation.
(d) A line passing through the sixth chondrosternal articulation.

And posteriorly:

(a) A line touching the upper border of the scapulæ.
(b) A line passing through the spines of the scapulæ.
(c) A line passing through the inferior angles of the scapulæ.
(d) A line touching the upper border of the spine of the twelfth dorsal vertebra.

Regional Divisions of the Thorax.

By the intersection of certain of the above-described lines the following arbitrary regions are formed:

(a) THE SUPRASTERNAL REGION. — This region overlies the thyroid body, the trachea, and more deeply the œsophagus. The transverse aorta, when dilated, extends into it and may be felt pulsating above the level of the sternal incisura.

(b) THE UPPER STERNAL REGION. — Beneath the breastbone lie the remnants of the thymus, the mesial borders of the upper lobes of the lungs, and more deeply the transverse arch of the aorta.

(c) THE LOWER STERNAL REGION.—Within the limits of this space lie the mesial border of the right lung, the termination of the fissure forming the upper boundary of the middle lobe, and that part of the right heart which constitutes the area of superficial dulness.

On each side:

(d) THE SUPRACLAVICULAR REGION.—This space lies above the upper edge of the collar-bone and contains the apex of the corresponding lung.

(e) THE CLAVICULAR REGION.—A space of no great moment in diagnosis. It corresponds to the boundaries of the inner half of the bone. The clavicle may be used as a pleximeter in direct percussion.

(f) THE INFRACLAVICULAR REGION.—A most important area of the chest. It is bordered above by the line of the clavicles, internally by the line of the sternal border, externally by the line of the anterior axillary fold projected upward to the acromion process, and below by the horizontal line passing through the third chondrosternal articulation. It contains on either side that part of the upper lobe of the lung in which tuberculous bronchopneumonia is as a rule first recognizable.

(g) THE MAMMARY REGION.—From the lower border of the preceding to the line passing through the sixth chondrosternal articulation. This space contains on the right side a part of the upper and middle lobes and the fissure separating them, together with the right auricle near the sternal border, and more deeply in the vault of the diaphragm the convexity of the right lobe of the liver. It overlies on the left side the extrasternal area of superficial dulness, the apex of the right and of the left ventricle, and the mesial border of the left lung with the lingula. Into the mammary region on each side extend the interlobar fissures of the lungs.

(h) THE INFRAMAMMARY REGION.—This area, which extends from a horizontal line through the sixth chondrosternal articulation downwards, overlies the liver on the right side, and upon the left a portion of the left lobe of the liver, the fundus of the stomach, the transverse colon, and the spleen. On the left is Traube's semilunar space.

(i) THE AXILLARY REGION.—This space is bounded by the lines of the axillary folds and the armpit above. It is a diagnostic territory of some importance.

(j) THE INFRA-AXILLARY REGION.—The upper boundary is the line which passes through the sixth chondrosternal articulation; its lower is the base of the chest. In this region, upon the left, the upper border of splenic dulness may be demonstrated upon percussion. The interlobar fissure traverses the axillary and infra-axillary spaces.

(k) THE SUPRASCAPULAR REGION.—An area of importance on account of the early manifestations of phthisis.

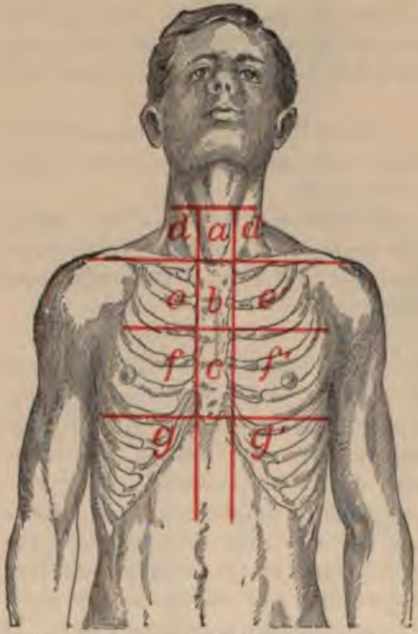


FIG. 13.—Regional divisions of the thorax: Anterior.—*a*, suprasternal region; *b*, upper sternal; *c*, lower sternal; *d,d'*, right and left supraclavicular; *e,e'*, right and left infraclavicular; *f,f'*, mammary; *g,g'*, inframammary.

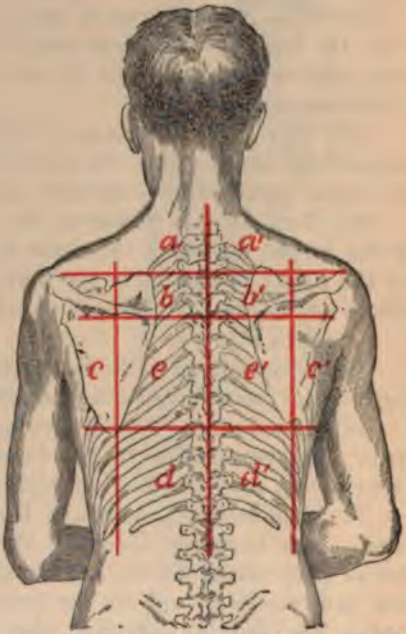


FIG. 14.—Regional divisions of the chest: Posterior.—*a,a'*, supraclavicular regions; *b,b'*, supraspinous; *c,c'*, infraspinous; *d,d'*, infrascapular; *e,e'*, interscapular regions.

(l) THE SUPRASPINOUS REGION.—That space lying between the upper border of the scapula and the spine of the scapula, and occupied by the thick supraspinous muscle.

(m) THE INFRASPINOUS REGION.—From the spine of the scapula to the level of the inferior angle. The infraspinous and infrascapular regions are traversed by the interlobar fissures. This fact is of importance in the recognition of the signs of the extension of a tuberculous infiltration to the apex of the lower lobe.

(n) THE INFRASCAPULAR REGION.—From the angle of the scapula, namely, about the level of the seventh rib, to the base of the chest.

(o) THE INTERSCAPULAR REGION.—The space lying between the inner borders of the two scapulæ. It extends across the spinal column and is much widened when the arms are folded and the body bent forward.

THE ABDOMEN.

The abdomen is the great cavity of the body extending from the diaphragm above to the levator muscles of the anus below. It is subdivided by an oblique plane at the brim of the pelvis into two portions, the abdomen proper and the pelvis. For the purpose of exact reference to the position and condition of the organs contained in the cavity of the abdomen in health and disease, certain lines, as in the case of the thorax, are recognized upon the surface. These dividing lines are natural, and artificial or conventional.

The Natural Lines of the Abdomen.

(a) THE LINEA ALBA in the middle line from the ensiform cartilage to the symphysis pubis.

(b) THE LINEÆ SEMILUNARES, one upon either side, passing from the ninth costal cartilage to the pubic bone and following the outer border of the rectus abdominis muscle.

(c) THE LINEÆ TRANSVERSÆ, of which there are three, the upper being at the level of the tip of the ensiform cartilage, the middle at a level midway between the first and the navel, and the third at the level of the navel.

(d) In fat persons A DEEP TRANSVERSE SULCUS or furrow crosses the abdomen a short distance above the pubic arch and a second similar but less marked groove is sometimes seen about the level of the umbilicus. These grooves vary in depth according to the amount of fat in the belly wall and are deeper in the erect than in the recumbent posture.

The Imaginary or Conventional Lines.

(a) THE MESIAL LINE, passing through the tip of the ensiform cartilage, the umbilicus, and the symphysis pubis, and corresponding to the linea alba.

(b) THE PROLONGATION DOWNWARD of the midclavicular line, which passes through the eighth costal cartilage to the middle of Poupart's ligament upon each side.

(c) THE INFRACOSTAL LINE, passing around the body in the horizontal plane of the tenth costal cartilages.

(d) THE BI-ILIAC LINE, which corresponds to the plane of the most prominent part of the iliac crests.

These two lines (c) and (d) divide the abdominal surface into three zones: an upper or epigastric, a middle or umbilical, and a lower or hypogastric. The two vertical lines dropped from the middle of the clavicle to the middle of Poupart's ligament again divide each of those zones into three regions, as follows:

(a) AN EPIGASTRIC REGION OR UPPER CENTRAL REGION.--This overlies a portion of the right and left lobes of the liver and a large part of the anterior wall of the stomach, with the pylorus, the aorta, the cœliac axis, the semilunar ganglia, and at a greater depth the pancreas.

(b) A Right and Left Hypochondriac Region.—The right hypochondriac region overlies the right lobe of the liver and the gall-bladder, the duodenum, the hepatic flexure of the colon, and the upper part of the right kidney; the left the greater curvature of the stomach, the spleen, the tail of the pancreas, the splenic flexure of the colon, and the upper part of the left kidney.

(c) An Umbilical or Middle Central Region.—In this space lie the greater curvature of the stomach, the mesentery, the great omentum, coils of the small intestine, and the transverse colon.

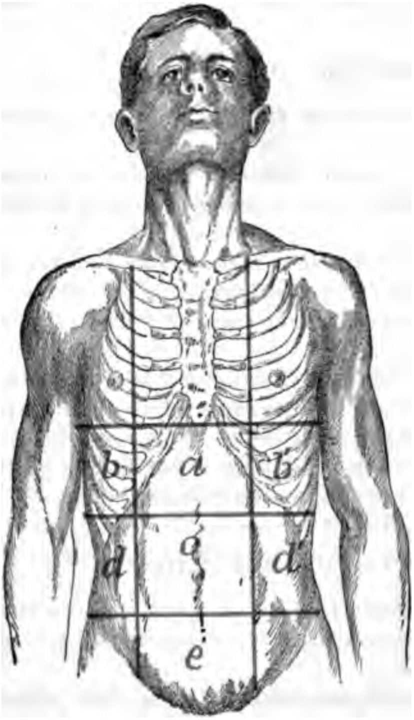


Fig. 15.—Regional divisions of the abdomen: *a*, epigastric or upper central region; *b,b'*, right and left hypochondrium; *c*, umbilical or middle central region; *d,d'*, right and left lumbar regions; *e*, hypogastric or middle lower region.

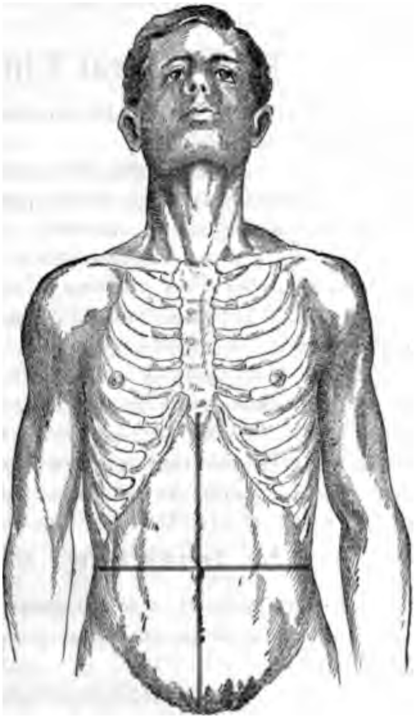


Fig. 16.—Quadrants of the abdomen.

(d) A Right and Left Lumbar Region.—The right contains the lower part of the right kidney, the ascending colon, and coils of small intestine; the left the lower part of the left kidney, descending colon, and small intestine.

(e) A Hypogastric, Suprapubic, or Middle Lower Region.—This space overlies coils of the small bowel, at its lower portion the fundus of the urinary bladder when distended, and the gravid womb.

(f) A Right and Left Iliac or Inguinal Region.—The right contains the cæcum and the base of the appendix or frequently the whole of it, the ileocæcal valve and the right ureter; the left the descending colon and left ureter.

The Quadrants of the Abdomen.

A simpler division of the surface of the abdomen into regions may be made by a vertical and a transverse line intersecting at the umbilicus. The four spaces thus defined are known respectively as the RIGHT and LEFT UPPER and LOWER QUADRANTS.

The Visceral Regions.

Certain important viscera give their names to the surface areas corresponding to the situation in which they are normally found. Thus we speak of:—

(a) THE PRECORDIAL AREA; THE PRECORDIA.—That part of the chest wall which overlies the heart, including the areas of superficial and deep dulness and increasing in extent in cardiac dilatation and hypertrophy.

(b) THE REGION OF THE APEX.—A more circumscribed space immediately above and around the normal apex and shifting as the apex shifts in enlargement and displacement of the heart.

(c) THE GASTRIC AREA, which corresponds to the normal situation of the stomach. The limits of this region are not strictly defined, since the organ varies in size when empty or distended with food or gas, and has some degree of mobility.

(d) THE HEPATIC AREA.—The lower border of this region is usually sharply defined both in normal and pathological conditions. Its upper border rounds away from the chest wall from which the upper surface of the liver is separated by the edge of the lung, and its left border is obscured by the tympany of the stomach and colon.

(e) THE REGION OF THE GALL-BLADDER.—The notch for the gall-bladder lies in the under border of the liver, slightly internal to the ninth right costal cartilage and near the outer border of the right rectus muscles. The fundus of the organ when distended and enlarged occupies a considerable area on both sides of this point as well as below it.

(f) THE ILEOCÆCAL AREA.—The part of the abdominal surface lying in the right lower quadrant of the abdomen and the seat of the local manifestations in appendicitis. Here lies the spot of focal tenderness described as McBurney's point.

(g) THE SPLENIC AREA.—The region which occupies the left hypochondrium extending towards the infra-axillary region. An enlarged spleen frequently transcends the normal borders of the splenic area, and a dislocated spleen occupies an entirely different position, in such a manner that the normal dulness in the splenic area is replaced by tympany.

(h) THE SIGMOID AREA.—The left inguinal region and the parts bordering upon it toward the median line, which are so designated because new growths and other pathological conditions involving the sigmoid flexure of the colon give rise to tumors or other clinical manifestations in this portion of the abdomen. It corresponds with the left lower quadrant.

(i) THE PELVIC AREA.—The designation sometimes employed to describe the suprapubic area because it is the region of the abdomen in which enlargements and new growths of the pelvic viscera are frequently manifest.

The extent of the various regions of this group is neither constant nor well defined. Their borders are often shifting and overlapping. Nevertheless they serve a useful purpose in the diagnosis of diseases of the abdominal organs.

Large accumulations of fat in the belly wall or within the peritoneal cavity, pregnancy, meteorism, dropsy and ascites, visceral displacements and enlargements, new growths and extra- and intraperitoneal cysts and abscesses distend the abdomen, modify its contour, and disarrange, often to an extreme degree, the relations between the above-described areas and the internal organs.

The foregoing anatomical and conventional lines and areas enable us definitely to fix the position of clinical phenomena for purposes of description and record.

The signs or symptoms of a lesion may be referred to a given region, as episternal pulsation, infraclavicular dulness, or precordial pain. More exactly the location of a given phenomenon may be indicated by the rib or interspace in which it is found and the distance from the midsternal line or its relation to one of the other vertical lines described, as, for example, the signs of a small cavity in the second interspace, a measured distance to the right—or left—of the median line; a presystolic thrill in the fifth interspace, to the left of the left parasternal line; an undulatory impulse in the fourth, fifth, and sixth interspaces, extending to a point midway between the left midclavicular line and the line of the anterior axillary fold.

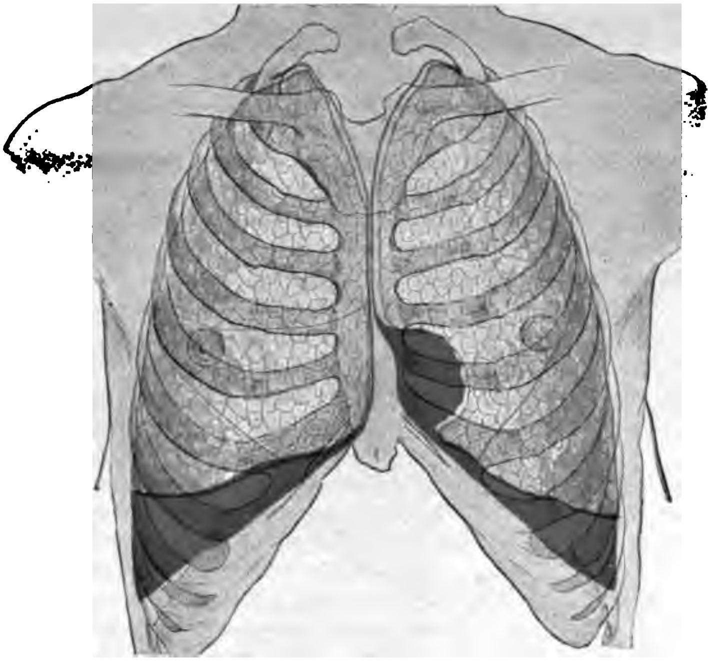
A tumor or painful spot in the abdomen may be located in one of the nine regions described as the epigastric, right iliac, hypogastric, and so on, or in one of the quadrants of the abdomen.

If greater accuracy is desired, the position of a lesion, physical sign, or tender spot may be stated to be a measured distance to the right or left, as the case may be, of the middle line at the level of the umbilicus, or a measured distance above or below the level of the umbilicus. Or, again, the anterior superior spine of the ilium may be taken as the point of departure for similar measurements.

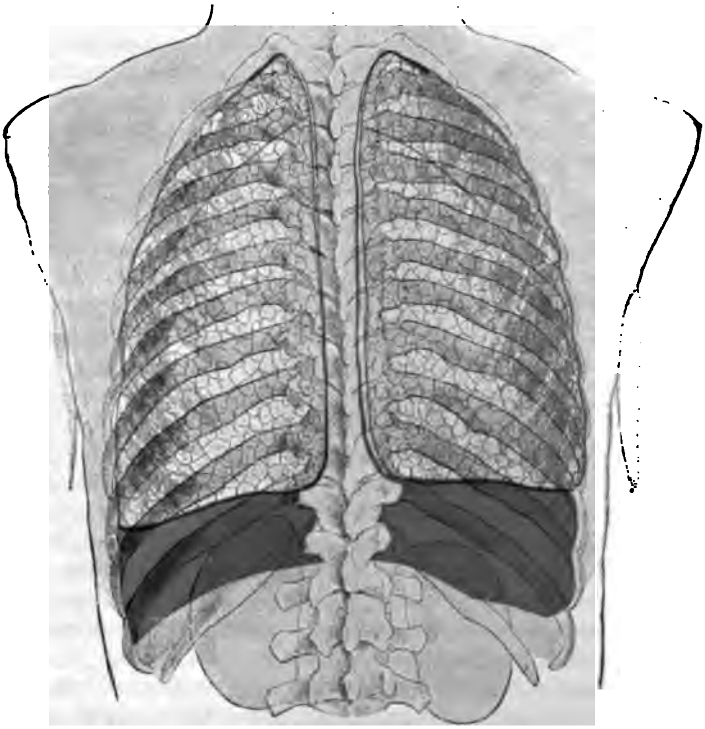
In the back the spinous processes may be taken as points of departure for the measurements. Thus a lesion may be a measured distance from the middle line on a level with the eighth dorsal spine or over a numbered interspace or rib.

The unit of measurement may be the centimetre, or the inch, if preferred, or the finger's breadth which equals about 2 centimetres or ¾ inch, or the hand's breadth, which varies from about 9 to 11 centimetres or 3½ to 4½ inches.

It is customary to indicate the extent of a lesion or the size of a tumor by less accurate but significant anatomical measurements; thus we say of a splenic tumor that it extends to the crest of the ilium or to the symphysis pubis or beyond the median line, or of a distended bladder or enlarged uterus that it reaches halfway from the pubis to the umbilicus or to the level of that anatomical landmark.



17.—Semidiagrammatic reconstruction, showing relations of pleural sacs (blue) and lungs (red) to thoracic wall; anterior aspect.



. 18.—Semidiagrammatic reconstruction, showing relations of pleural sacs (blue) and lungs (red) to body-wall: posterior aspect.

THE TOPOGRAPHICAL ANATOMY OF THE THORACIC ORGANS.

The Thymus Gland and its Remnants.

This temporary organ attains its maximum development about the end of the second year. It then undergoes a gradual involution process until it is reduced to a mere vestige. When fully developed it appears as a narrow elongated body lying in the anterior mediastinal space immediately behind the manubrium sterni and extending into the episternal region of the neck. Its size varies according to the degree of development. At birth it is about 6 centimetres in length, 2.5 centimetres in width, and .75 centimetre in thickness. The thymus is occasionally persistent and may then undergo hypertrophy. In this case and when enlarged as the result of tuberculous, syphilitic, or cancerous disease, or hemorrhagic or purulent infiltration, pressure symptoms, namely, paroxysmal dyspnœa—so-called thymic asthma—persistent dyspnœa, spasm of the glottis, or venous hyperæmia and local œdema arise.

The Trachea or Windpipe.

This tubular organ extends in the median line from the larynx to a point opposite the third dorsal vertebra, where it is crossed in front by the arch of the aorta, and there or immediately below this level it bifurcates into the right and left bronchi. Its length is variable, being in the adult about 9 to 11 centimetres, its width from 2 to 2.5 centimetres. It is both wider and longer in the male than in the female. The trachea is movable and may be displaced as well as compressed by an aneurism or a new growth. Its posterior membranous part is in relation with the œsophagus behind, and the recurrent laryngeal nerves ascend in the groove between these two organs. The manubrium sterni overlies the trachea, which traverses the posterior mediastinum.

The Primary Bronchi.

The right and left bronchi arise at the bifurcation of the trachea and diverge to the corresponding lung upon each side, which they respectively enter at the root to form by successive subdivisions the ramifications of the bronchial tree. The right bronchus—the wider and shorter of the two—passes obliquely downwards and outwards to the lung at the level of the fourth dorsal vertebra, and behind the aorta; the left, smaller in diameter but much greater in length, runs obliquely downwards and outwards below the arch of the aorta to the root of the left lung, into which it passes at the level of the body of the fifth dorsal vertebra. The length of the right bronchus is about 2.5, that of the left nearly 5 centimetres.

Irregular stenosis of the trachea or a main bronchus, from an aneurismal or neoplastic tumor or from a tenacious and adherent exudate, causes tracheal stridor and the accumulation of an abundant liquid exudate, as in some forms of bronchitis, and the pulmonary œdema that precedes death gives rise to coarse tracheal râles.

Elasticity of the Tracheobronchial Structures.—That these organs have a high degree of pliability, analogous to that of the vesicular structure of the lung, is shown by the manner in which they accommodate themselves to the displacing and distorting pressure of effusions, aneurism, and new growths of various kinds without great impairment of their function. That they possess equally remarkable capacity of elongation and contraction has been recently demonstrated by X-ray examination and the bronchoscope of Chevalier Jackson.

The Œsophagus: Gullet.

This tubular organ extends from the pharynx at the lower border of the fifth cervical vertebra—the level of the cricoid cartilage—along the anterior surface of the borders of the vertebræ, to pass through the diaphragm about the level of the ninth dorsal vertebra and end in the cardiac orifice of the stomach. Its length is about 23 centimetres. In the thorax it lies posterior to the lower part of the trachea, the upper part of the left bronchus, and the posterior surface of the pericardium. The œsophagus may be the seat of simple or syphilitic cicatricial stricture; stenosis from cancerous growth involving its wall or pressing upon it from without or from the external pressure of an aneurism. Spasmodic stricture occurs in neurotic and hysterical persons, and the impaction of a foreign body, as an artificial denture, a large piece of meat, or a bone, may cause mechanical obstruction, an accident that occasionally occurs among the insane. It is sometimes the seat of a diverticulum. The œsophagus is accessible to examination by the sound, the œsophagoscope, and X-rays. The time occupied in swallowing and the nature of the accompanying sounds may be studied by auscultation.

The Lungs and Pleuræ.

The lungs occupy the greater part of the cavity of the chest, enclosing between their concave inner surfaces the heart and great vessels. Each lung is attached to the inner wall of the thorax in the region of the bodies of the fourth and fifth dorsal vertebræ by a comparatively small pedicle called the root, and a narrow membranous fold continued downwards from it. Elsewhere the surface of the lung is free and covered by a serous membrane, the pleura, which is also reflected upon the inner wall of the chest. The root of each lung is composed of the respective main bronchus together with large blood-vessels, lymphatic vessels, chains of lymphatic glands, held together by connective tissue and enclosed in the pleura.

THE PLEURÆ.

Each pleura is a closed serous sac, lining the lateral cavity of the thorax to which it belongs, enclosing the lung and its root and forming by the aid of its fellow of the opposite side the mediastinum. That part of the pleura which encloses and covers the lung and its root is called the visceral or pulmonary pleura; that which is reflected upon the ribs and intercostal spaces, covers the upper convex surface of the diaphragm, and passes to the sides of the pericardium, thus forming the mediastinum,

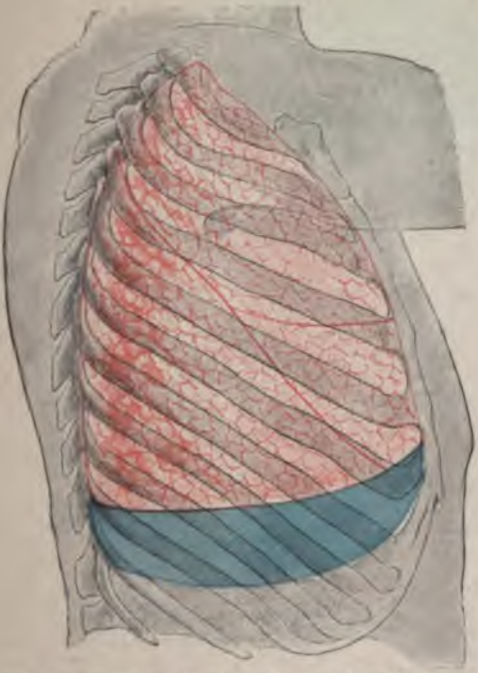


FIG. 19.—Semidiagrammatic reconstruction, showing relations of right pleural sac (blue) and lung (red to thoracic wall; lateral aspect.

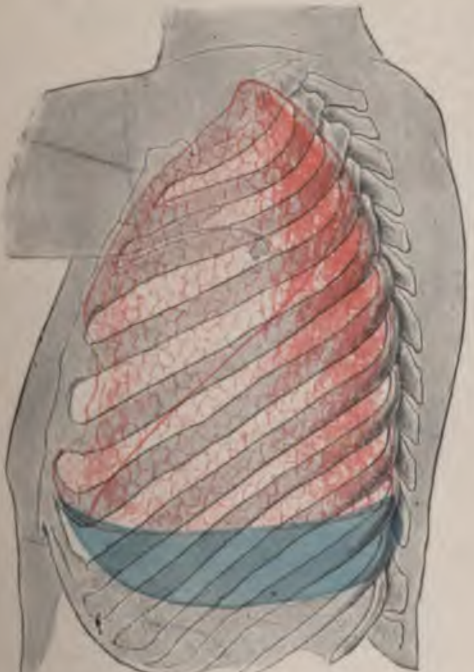


FIG. 20.—Semidiagrammatic reconstruction, showing relations of left pleural sac (blue) and lung (red) to thoracic wall; lateral aspect.

is called the parietal pleura, or — as to its different parts — the costal, diaphragmatic, and mediastinal pleura; and these two parts—namely, the visceral and the parietal pleura—are continuous with each other at the root of the lung.

The upper part of the pleura on each side passes upward beyond the clavicle into the neck, and contains the apex of the lung, which reaches from 2.5 to 4 centimetres above the margin of the first rib, usually a little higher upon one side than upon the other, but not constantly higher upon the right side as is often stated. Beneath the sternum the pleural sacs of the two sides come nearly or quite into contact in the upper part, but in the lower part the right pleura passes to or even beyond the middle line and the left pleura recedes from it to a variable distance beyond the sternal border. At the base of the chest the pleuræ do not reach to the attachments of the diaphragm, but they are reflected from the inner wall of the chest to the rising vault of the diaphragm in such a manner that, on quiet respiration or on full expiration, the parietal and visceral pleuræ are not in apposition, but the costal and diaphragmatic surfaces of the parietal pleura are opposed. The higher position of the right diaphragmatic vault, due to the high position of the right lobe of the liver, renders the right pleura somewhat shorter than the left, while the smaller portion of the heart upon the right side of the median line renders the right pleura somewhat wider than the left.

THE LUNGS.

Each lung is cone-shaped—with its blunt apex extending into the root of the neck, its anterior surface flattened, its lateral and posterior convex surfaces strongly convex, and its inner and inferior surfaces concave. The contour resulting from this conformation gives rise to sharp, well-defined anterior margins, the horizontal sections of which are acutely angular, and to a similar, sharply angular, circumferential border at the base, which fits into the corresponding re-entrant angle between the thoracic wall and the diaphragm—a fact of no little importance in physical diagnosis. Each lung is divided by a long, deep fissure, beginning about the level of the spine of the scapula and proceeding obliquely downward and outward to the sixth rib in the midaxillary line, into an upper and a lower lobe. The right lung is further divided by a second, shorter fissure, which passes inward either straight or in an upward or downward direction through the anterior margin, thus forming a third or middle lobe. Upon the inner anterior border of the left lobe is situated a deep notch into which the heart, enveloped in its pericardium, is received, and at the inferior part of this border of the lung is situated a tongue-like projection which passes in front of the apex of the heart—lingula.

The lungs completely fill the chest, and the surfaces of the visceral and parietal pleuræ are accurately in contact except along the anterior and inferior margins of the lungs. In these situations the sharp wedge-like borders of the lung advance between the reflected layers of the parietal pleura during inspiration and recede during expiration, as above stated.

The Mediastinum.

This space lies between the layers of an anteroposterior septum formed by the inner or mesial portions of the right and left pleuræ which pass upon the surface of the pericardium from the anterior and posterior walls of the chest to the root of the lung upon either side. It is subdivided into an anterior, middle and posterior mediastinum.

The anterior is narrow and of little depth, lying directly behind the inner surface of the sternum. At its upper part it contains the atrophied thymus. Behind the gladiolus the right and left pleuræ are in contact, and the anterior mediastinum consists merely of the connective-tissue layer by which they are joined. Lower down, while still shallow, it is widened, by the departure of the left pleura from the midsternal line, into a triangular space which lies between the anterior portion of the right ventricle and the wall of the thorax—the area of superficial cardiac dulness.

The middle mediastinum is the large space between the mesial layers of the two pleuræ which contains the pericardium and its contents.

The posterior mediastinum lies in front of the vertebral bodies and contains the trachea, the œsophagus, the thoracic duct, the descending aorta, the azygos vein, lymphatic vessels and the pneumogastric nerves.

THE PERICARDIUM.

This membranous sac, which occupies the middle mediastinum and contains the heart and the roots of the great blood-vessels, is conical in shape, its base resting upon the diaphragm and its apex extending upwards upon the walls of the blood-vessels as far as their first subdivisions. It consists of two layers, an external fibrous layer, which is attached below to the central tendon of the diaphragm, and above to the surface of the large blood-vessels which it embraces, and an inner serous layer, which lines the fibrous sac in which the heart is contained and is reflected upon the surface of that viscus in such a manner as to form a parietal and a visceral portion. The latter is sometimes described as the epicardium. The fibrous pericardium is furthermore firmly attached to the structures by which it is surrounded, namely, the sternum in front, the mediastinal pleuræ laterally, and the trachea, œsophagus, and main bronchi behind.

The Heart and Great Vessels.

THE HEART.

This central organ of the circulation is situated in the cavity of the thorax in the middle mediastinum. It lies unattached within the pericardium except by the great vessels which spring from its cavities at the base, and it rests upon the convexity of the diaphragm. Its base is directed upward, backward, and toward the right, and extends from the level of the fourth to that of the eighth dorsal vertebra, while its apex points downward, forward, and toward the left, coming into relation with the chest wall in the fifth intercostal space a little to the left of the parasternal line. It projects farther to the left of the median line than to the right in the average ratio of nearly 2 to 1.

Orthodiagraphic measurements have shown that the average oblique diameter of the heart from the true apex to the angle at the upper right border of the auricle and the great vessels is between 13 and 14 centimetres; the horizontal distance from the midsternal line to the most distant point of the border of the heart on the right, 3.5 to 4.5 centimetres; to the most distant point on the left, 7.5 to 8.5 centimetres.

The Relation of the Heart to the Anterior Wall of the Chest.—In general the normal heart in the adult may be said to extend from the level of the second intercostal space on the right side to the fifth interspace on the left. Investigations conducted to ascertain the exact relations of the viscus to the chest wall by thrusting long needles through it immediately after death, by means of sections of frozen bodies, and by the X-rays have not yielded constant nor concurrent results. The discrepancies are doubtless due to differences existing naturally among individuals and to variable conditions, in themselves equally incapable of exact determination: for example, the position of the diaphragm, the amount of residual air in the lungs, the quantity of gas in the stomach and intestines, and the volume of blood in the chambers of the heart at the time of examination. For clinical purposes it is possible to be overexact in variable matters of this kind.

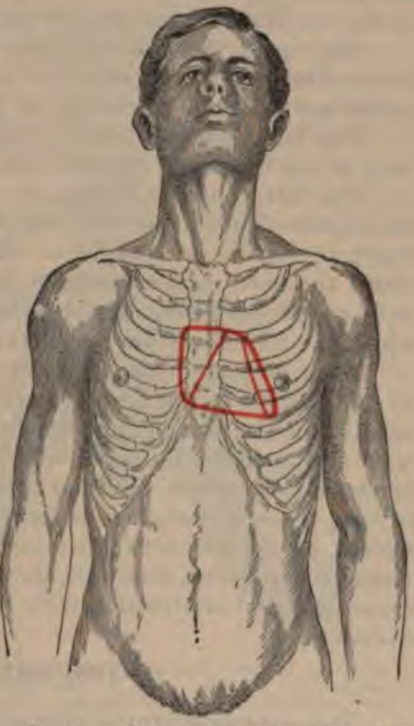


FIG. 21.—Outline of heart and lines indicating the auriculoventricular groove and the anterior interventricular groove.

The greater part of the anterior surface of the heart is not directly in relation with the inner chest wall, but separated from it by the wedge-like anterior borders of the lungs. The superior border of the heart closely corresponds to a transverse line drawn about the level of the upper edges of the third costal cartilages and extending from a point two centimetres from the right border of the sternum to the third left costochondral articulation. This line constitutes the clinical base of the heart and subdivides the precordia into the cardiac area and the area of the great vessels.

The inferior border is indicated by a line drawn from a point on the upper border of the sixth rib, directly below the outer limit of the impulse, obliquely upward and to the right, across the base of the ensiform cartilage, and terminating at the middle of the fifth right interspace near its junction with the sternum.

The right border nearly coincides with a line drawn from the point at which the superior border terminates on the right, convex to the right, to the middle of the fifth interspace as above, namely, about 2 centimetres to the right of the right sternal border.

The left border is marked by a line joining the apex and the articulation of the third left rib with its cartilage.

A line joining the third left chondrosternal articulation and the seventh right chondrosternal articulation corresponds fairly well with the line of the auriculoventricular septum.

A line joining the apex and the third left costochondral articulation corresponds closely with the interventricular septum.

The greater part of the anterior surface of the heart is formed by the right ventricle and constitutes a triangle included between the above lines and the inferior border of the heart. The apex of this triangle is occupied by the conus arteriosus and the tip of the left auricular appendix.

The upper third of the right auricle lies behind the sternum. while its two lower thirds extend to the right of the sternal edge and are bounded by the curved right border of the heart.

The left auricle is deeply seated and is completely covered by the body of the heart and the left lung.

The left ventricle is likewise deeply seated and wholly retired from the surface of the chest with the exception of a narrow longitudinal strip which forms the left border of the heart and presents anteriorly, and of which the lower end constitutes the true or anatomical apex of the heart. and is separated from the chest-wall by the lingula, the clinical apex to which the impulse is due being the apex of the right ventricle.

That portion of the anterior surface of the heart which. uncovered by the borders of the lungs, comes into relation with the wall of the chest. constitutes the area of superficial cardiac dulness and may be more or less accurately defined by percussion; that which recedes by its rounded surfaces from the chest wall and is covered by a rapidly thickening volume of lung tissue is described as forming the area of deep cardiac dulness and cannot be defined with the nicety which some assume by the ordinary methods of physical diagnosis, though the shadow of its borders may be seen expanding and contracting with the revolutions of the heart upon X-ray examination.

THE GREAT VESSELS.

The ascending arm of the arch of the aorta arises at the base of the left ventricle of the heart behind the pulmonary artery. Its course is at first upward and to the right and slightly forward as it passes behind the sternum. At the level of the second right costal or aortic cartilage, the vessel passes upward, backward, and to the left, forming the transverse portion of the arch, then backward and downward to form the descending arm of the arch which terminates in the descending portion of the thoracic aorta.

The pulmonary artery passes a little more than a centimetre beyond the left border of the sternum in a line about the level of the middle of the left third interspace upward to the second costal cartilage. behind which it divides into its right and left main branches.

The descending vena cava extends from the second interspace on the right side of the sternum to the base of the heart, which it enters at the level of the middle of the third interspace. Its course is slightly curved, the convexity being toward the right.

These vessels are situated at varying depths behind the manubrium sterni and in an area extending beyond the right and left sternal borders. This region is sometimes designated the area of the great vessels.

The Relation of the Valves of the Heart to One Another and to the Surface of the Chest.—The lines of attachment of the bases of the mitral and tricuspid valves correspond to the auriculoventricular sulcus. The semilunar cusps of the aortic and pulmonary valve systems are situated respectively at the origin of each of those vessels from the ventricles. The four sets of valves lie in close proximity to one another and to some extent overlap. The pulmonary is most superficial; the mitral most deeply situated; the aortic centrally placed and in parts of its extent covered by the pulmonary; and the tricuspid lowest in position.

Their relations to the surface of the chest are as follows:

The pulmonary valve lies horizontally immediately to the left of the sternal border at the level of the upper edges of the third left costal cartilage.

The aortic valve is at a level slightly lower than the pulmonary and situated behind the sternum at the level of the third left intercostal space and to the left of the median line. It is nearly horizontally placed.

The mitral valve—left auriculoventricular — lies on an oblique line from above downward to the right behind the sternum extending from the level of the left third intercostal space to the level of the lower border of the left fourth costal cartilage.

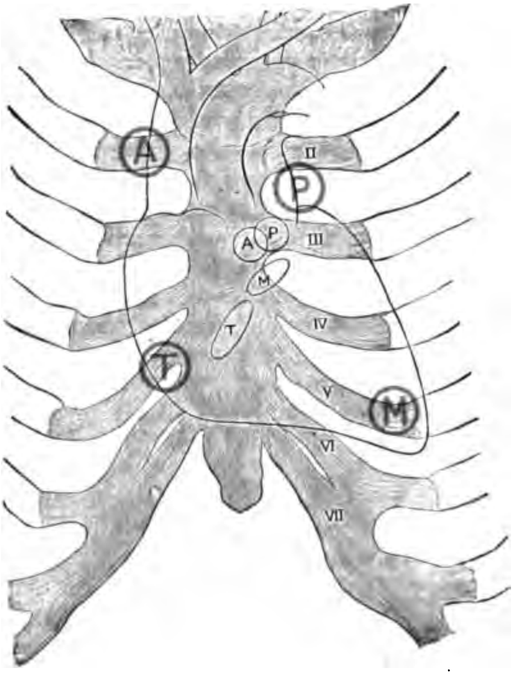


FIG. 22.—Position of heart and valves in relation to anterior thoracic wall. A, aortic valve; P, valve of pulmonary aorta; T, tricuspid valve; M, mitral valve; and puncta maxima indicated by red circles.

The tricuspid valve—right auriculoventricular—lies still more obliquely behind the sternum opposite the fourth intercostal space on a line drawn through a point in the midsternal line on the level of the third interspace to the sixth chondrosternal articulation.

These four valve systems are so close to one another that the sounds produced by each cannot be studied by auscultation directly over the seat of the valve, but at that point in the precordia at which the blood stream at the moment directly affecting the particular valve mechanism approaches the surface of the chest most closely.

Puncta Maxima.—These areas, of which there are four, corresponding to the separate valve systems, are:

1. **The pulmonary area**—at the inner end of the second left intercostal space.

2. **The aortic area**—at the second right costal cartilage.
3. **The mitral area**—at and just above the position of the apex-beat.
4. **The tricuspid area**—at the right border of the lower end of the sternum.

THE TOPOGRAPHICAL ANATOMY OF THE ABDOMINAL VISCERA.

The Stomach.

The stomach is that dilated portion of the alimentary canal which lies between the cardiac end of the œsophagus and the pyloric end of the duodenum. It is irregularly gourd-shaped, the larger left end being called the fundus or splenic extremity; the smaller right end the pyloric extremity. The orifice by which the œsophagus enters is called the cardia or cardiac orifice, that passing to the duodenum the pylorus. The former is immediately below the central part of the diaphragm and lies between the greater and lesser curvatures. The latter lies lower down, more toward the anterior abdominal wall, and to the right. The shorter inner curvature of the gourd is known as the lesser, the longer outer curvature is the greater curvature of the stomach. This hollow viscus lies chiefly in the epigastric and left hypochondriac regions, the greater part of its extent being, when distended, in about the proportion of 1 to 5, to the left of the median line. During physiological rest the healthy stomach contains only a little mucus and a small accumulation of air or gas which occupies its fundus, and forms a narrow wrinkled pouch, the long diameter of which is oblique from the cardia downward and to the right and approaches much more nearly to the vertical than to the transverse axis of the body. Its superior border is fixed at the cardia at the point at which the œsophagus pierces the diaphragm and is attached to the overlying liver and diaphragm by the gastrohepatic omentum and the gastrophrenic ligament. The gastrocolic omentum is attached to the lower, the gastrosplenic omentum to the left border. The anterior surface is in relation with the diaphragm and under surface of the liver above and the wall of the abdomen lower down; the posterior surface is in relation with the great vessels and pancreas above and the transverse mesocolon lower down. Both these surfaces are free, smooth, and invested with peritoneum. When the stomach is distended, it rotates upon its cardiopyloric axis in such a manner that the anterior surface tends to look upward and the posterior surface downward. The dimensions of the stomach vary according to the degree of distention caused by food, fluid, or gas. When moderately filled, its longest diameter is about 25 centimetres, its diameter between the greater and lesser curvature from 9.5 to 12 centimetres, and the diameter between its anterior and posterior walls about 9 centimetres. When much distended, a normal stomach may reach to the level of the umbilicus.

The cardia is situated in a direct line posterior to the left seventh chondrosternal articulation at a distance of about 10 to 12 centimetres from the anterior abdominal wall. The pylorus, which has considerable freedom of motion, lies about the level of the tip of the ensiform cartilage and near the outer border of the right rectus muscle. It is in relation with the concave surface of the liver and may extend to the neck of the gall-bladder. When the stomach is distended the pylorus assumes a position further to the right and lower in the abdomen. The fundus rises into the vault of the diaphragm to the level of the fifth interspace in the midaxillary line and is higher than the cardia, just as the lateral vault of the diaphragm is higher than its central aponeurosis. Its upper part lies behind the anterior diaphragmatic border of the left lung and the tips of the seventh, eighth, and ninth left ribs and their cartilages. The convex curve of Traube's semilunar space in this region corresponds with the curvature of the fundus of the stomach.

The Intestines.

A. **The small intestine** begins at the pylorus and terminates at the ileocæcal valve, at which point it joins the large bowel. It has an average length in the adult of about six metres. Its convolutions occupy the middle parts of the abdomen and are surrounded by the large intestine. They are attached to the back wall of the abdominal cavity by the mesentery. The small intestine is divided into (1) an upper portion, or duodenum, about 25 to 30 centimetres in length, into which in its middle third the common bile duct and pancreatic duct discharge their contents; (2) a middle portion, or jejunum; and (3) a lower portion, or ileum. In the last are situated Peyer's patches. The duodenum is the widest and least movable of the three portions of the intestines. The coils of the jejunum and ileum are freely movable within the abdomen and among themselves and bear no constant relation to the regions of the surface.

B. **The large intestine** extends from the termination of the small intestine at the ileocæcal valve to the anus. Its average length is between 1.5 and 2 metres. Its diameter varies at different parts and ranges from 3.5 to 6 centimetres. There is a pouch-like dilatation of the rectum immediately above its lower end. It is divided into three parts.

(1) **The Cæcum; Intestinum Cæcum; Caput Cæcum Coli.**—The shortest and widest part of the large intestine. It measures in length and width each about 6 centimetres. As a rule, there is no mesocæcum, and this part of the intestine is attached to the fascia covering the right iliacus muscle. The cæcum is situated in the right iliac fossa and is comparatively fixed. Its position determines that of the ileocæcal valve which lies between 6 and 7 centimetres mesial to the right anterior superior spinous process.

(2) **The appendix vermiformis** arises from the inner and posterior aspect of the cæcum near the ileocæcal valve. It lies in the right iliac region and its base is opposite McBurney's point. Its dimensions are extremely variable, its width being that of a large quill and its length from 6.5 to 9 centimetres. From its comparatively fixed base, the appendix, being free, may extend in any direction. As a rule it lies downward or inward. It may, however, extend backward, in which case the symptoms of appendicitis may suggest renal colic; or upward, and, if inflamed, suggest gall-bladder disease.

(3) **The Colon.**—This part of the large intestine constitutes its greatest length. It occupies the peripheral parts of the abdominal cavity, and, owing to the lack of a mesocolon in its ascending and a portion of its descending course, maintains a comparatively fixed position. In some

instances there is a short mesocolon in these portions. It is divided. according to its course and direction. into four parts. namely. an ascending, a transverse, a descending portion. and the rectum.

(a) The ascending colon. commencing at the cæcum. passes upward in a vertical direction to the under surface of the liver near the gall-bladder. where it turns forward and sharply to the left. forming the hepatic flexure. It is as a rule fixed in its whole course and overlaid by some coils of the ileum. It is contained in the right lumbar and hypochondriac regions.

(b) The transverse colon passes across the umbilical region from the right to the left hypochondrium. It is deeply situated at its right and left extremities. but in its intermediate course it bends forward and approaches the anterior wall of the abdomen—arch of the colon. It rises slightly at its left extremity to pass behind the costal margin in relation with the fundus of the stomach and turns abruptly downward to form the splenic flexure.

(c) The descending colon is continuous with the transverse colon at the splenic flexure. It descends nearly directly downward through the left hypochondrium and lumbar region to the left iliac region. where it curves inward and then downward to form the sigmoid flexure. The descending colon is covered only in front and at its sides by peritoneum. but the sigmoid flexure has a distinct mesocolon and is freely movable. The latter lies well toward the front of the cavity of the abdomen in the left iliac region.

(d) The rectum. notwithstanding its name. is not straight in man. but curved from its beginning at the brim of the pelvis in front of the left sacro-iliac articulation obliquely downward from left to right to the middle line of the sacrum. then forward in the hollow of the sacrum to the level of the prostate in the male or the vagina in the female. where it again turns and proceeds downward and obliquely backward to the anus. This part of the large intestine lies entirely within the pelvis. but is accessible to examination by the finger. the rectal bougie, and the proctoscope.

The Liver.

The liver is the largest gland in the body and occupies a large space in the abdominal cavity. It measures from 22 to 24 centimetres in its transverse. about 15 centimetres in its maximum anteroposterior. and 14 to 16 centimetres in its maximum vertical diameter. It is large and rounded in its right extremity; narrow and wedge-shaped toward the left; convex and smooth upon its upper surface; concave. uneven. traversed by various fissures, and showing the gall-bladder and extrahepatic bile passages upon its lower surface. The rounded. thick posterior part is the most fixed; the thin, sharp anterior margin the most movable part of the organ.

The liver occupies the right hypochondriac and extends across the epigastrium into the left hypochondriac region. It is closely adapted to the vault of the diaphragm and is in relation with the anterior wall of the abdomen on the right side as far down as the margin of the ribs. The right lobe reaches higher than the left—a fact in accord with the shorter vertical diameter of the right thorax as compared with the left. At its highest point the convex upper surface of the right lobe of the liver corresponds to the fourth intercostal space in the midclavicular line. The upper boundary gradually declines to the base of the ensiform cartilage in the direction toward the left and continues on the right and to the back almost upon the same level, crossing the midaxillary line at the level of the seventh intercostal space and the line of the angle of the scapula about the level of the ninth rib. Owing to the dome-like shape of the upper surface of the right lobe of the liver and the concavity of the base of the lung into which it is adapted, the diaphragm being interposed, there is a considerable difference in the level of the actual upper border of the organ and that of the portion which lies in contact with the wall of the thorax. The latter in the midclavicular line corresponds with the sixth rib; in the midaxillary line with the eighth rib, and posteriorly with the tenth rib. Upon percussion that portion of the liver which lies in relation with the wall of the chest yields well-marked dulness; that which is covered by the interposed border of the lung modified dulness. The former is spoken of as the area of superficial hepatic dulness, the latter as the area of deep hepatic dulness, and these two areas together constitute the area of hepatic dulness.

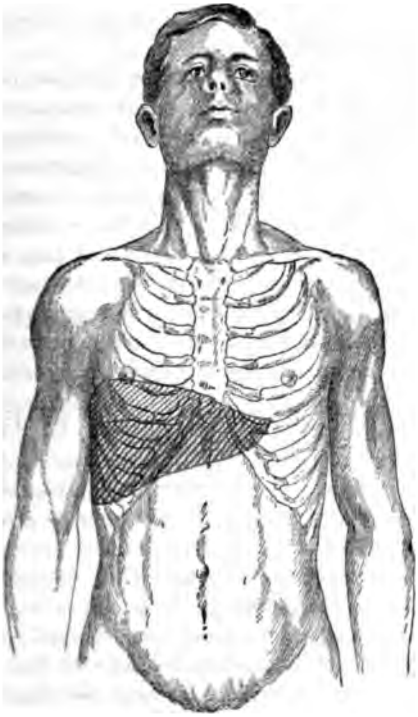


FIG. 23.—Aieas of deep and superficial hepatic dulness.

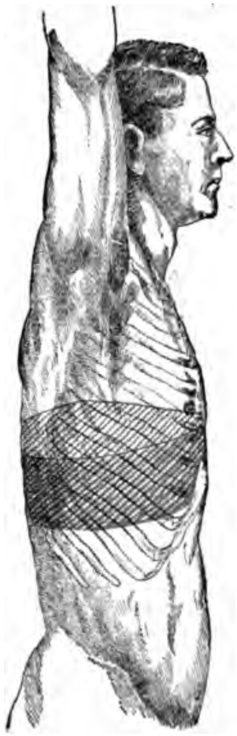


FIG. 24.—Areas of deep and superficial hepatic dulness.

The lower anterior margin corresponds in the midclavicular line with the margin of the ribs; in the median line it lies slightly above a horizontal line midway between the base of the ensiform cartilage and the umbilicus; about the left parasternal line at the lower border of the sixth rib; in the right midaxillary line at the tenth interspace; and at the spine about the level of the eleventh intercostal space.

The interlobar notch lies nearly in the median line. The thin edge of the left lobe reaches closely to the midclavicular line. To the right of the right midclavicular line the lower border corresponds approximately to the costal margin. In aged persons the liver occupies a slightly higher level; in children it is large in proportion to the size of the body and extends higher, displacing the apex beat of the heart to a point behind the fifth rib or in the fourth interspace, and causing the lower border to fall below the line above indicated by 1 or 2 centimetres.

The Gall-Bladder and Extrahepatic Bile Passages.

THE GALL-BLADDER.

This membranous sac is situated in a fossa in the base of the liver. It is pear-shaped, measuring in its long diameter from 7 to 10 centimetres and in its greatest transverse diameter about 4 centimetres. It lies obliquely, with its fundus, which projects beyond the anterior margin of the gland, looking downward, forward, and to the right. There is often a slight notch in the margin of the liver at this point, which corresponds to the outer border of the right rectus muscle at the level of the inner edge of the ninth costal cartilage.

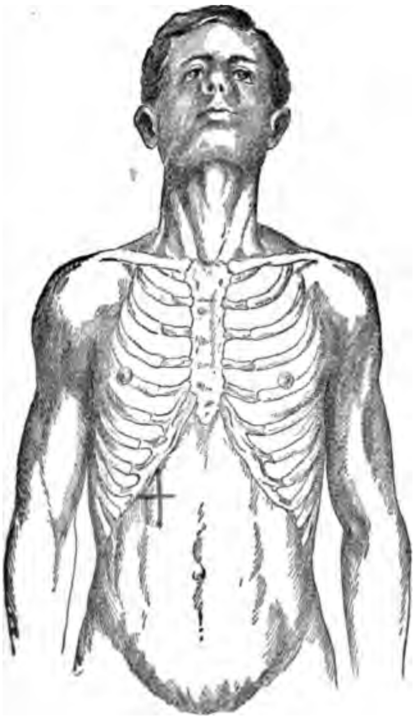


FIG. 25.—Position of fundus of gall-bladder.

THE EXTRAHEPATIC BILE PASSAGES.

THE CYSTIC DUCT.—The neck of the gall-bladder, which grows gradually narrower, forms a double curve like the letter S, and then becoming much constricted it turns abruptly downward to form the cystic duct, which runs downward and to the left and unites with the hepatic duct to form the common duct.

THE HEPATIC DUCT.—This duct is formed by the union of a right and a left branch, which issue from the transverse fissure and unite at an obtuse angle. Its diameter is 3 or 4 millimetres and its length about 4 centimetres. It unites with the cystic duct to form the common duct.

THE COMMON BILE DUCT; DUCTUS COMMUNIS CHOLEDOCHUS.—This is the largest of the bile passages, being 5 or 6 millimetres in width and 6 centimetres or more in length. It runs downward and backward to the inner and posterior wall of the duodenum, where, uniting with the pancreatic duct to form a dilatation, known as the ampulla of Vater, it penetrates the wall of the duodenum very obliquely in the course of its middle third.

Pathological conditions involving the ducts, such as cholangitis and gall-stone disease, do not directly give rise to physical signs, but they cause serious symptoms and, indirectly, marked physical signs, and a knowledge of the position and size of these ducts and their relations to each other is of prime importance in the diagnosis of the diseases to which they are liable.

The weight of the liver and its direct relationship with the diaphragm render it to a high degree subject to the influence of gravity in different postures of the body, as, for example, the erect position as compared with the dorsal decubitus, and to the influence of the respiratory movements. Due allowance for these changes in the position of the organ is to be made in its physical examination.

The Pancreas.

This elongated, flattened gland is situated deeply in the abdominal cavity directly behind the stomach and at the level of the first lumbar vertebra. The larger right extremity is called the head and is embraced by the curvature of the duodenum. Its smaller left extremity, the tail, is situated in a slightly higher level than the head and reaches to the spleen, with which it is in contact. This organ varies considerably in size, being between 15 and 20 centimetres in length, about 4 centimetres in average breadth, and about 2.5 centimetres in thickness. It extends across the epigastric region and into the right and left hypochondrium. Its principal duct traverses the entire length of the gland and in association with the common bile duct enters the duodenum by an oblique passage through its wall. Its great depth in the body renders it as a rule inaccessible to direct physical examination. The close relations of the head of the pancreas with the portal vein, the inferior vena cava, and the ductus communis choledochus are of clinical importance, since malignant or other disease attended by enlargement of that part of the gland constitutes a not infrequent cause of œdema, ascites, or persistent jaundice.

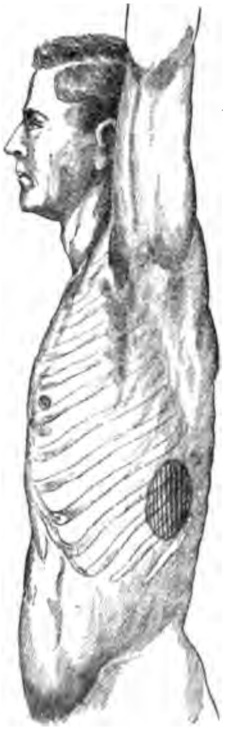


FIG. 26.—Position of spleen.

The Spleen.

This soft, vascular organ is situated in the left hypochondrium, opposite the ninth, tenth, and eleventh ribs, and in the posterolateral portion of the upper part of the abdominal cavity. It undergoes considerable variation in size in health and may be enormously enlarged in disease. It is irregularly oval in shape, its upper and posterior borders being rounded and thick, its lower and anterior borders sharp and the latter indented by two or more notches. Its convex outer surface is in relation with the inner surface of the left side of the diaphragm. Its concave inner surface presents a vertical fissure called the hilus, and is in relation at its posterior portion with the suprarenal capsule and the upper part of the left kidney, and at its anterior portion with the stomach, the splenic flexure of the colon, and coils of the small intestine. Its average long diameter under normal conditions is between 8 and 10 centimetres and it cannot be felt upon palpation. Supernumerary spleens are not uncommon.

The Kidneys.

The right and left kidneys are deeply seated in the lumbar region in the back part of the cavity of the abdomen and behind the peritoneum, opposite the last dorsal and the first, second, and sometimes the third lumbar vertebræ. The position of the right kidney is slightly lower than that of the left. Each kidney is about 9 centimetres long, 6.5 centimetres in width, and 3 centimetres in thickness, the left being usually longer and thinner than the right. Their oblong, rounded concavo-convex shape is characteristic. The convexity of each is directed outward and backward; the concavity inward and slightly forward. Near the middle of the concave surface is a longitudinal fissure or hilus at which the vessels and nerves enter or emerge and the ureter arises. This excretory duct expands within the hilus into the pelvis of the kidney, from which arise three or sometimes two funnel-shaped spaces which subdivide into a number of smaller tubes called calices or infundibula, similarly funnel-shaped but into which the papillæ of the kidney project. The kidneys are supported by the vessels and the perirenal fat. The right kidney is in relation with the duodenum and colon in front and the liver above; the left with the spleen above and colon anteriorly. Both lie against the corresponding pillar of the diaphragm, the anterior layer of the lumbar fascia, and the psoas muscle. The deep situation of the kidneys and the thick layers of muscles against which they rest, embedded in a layer of fat behind, render them under normal circumstances inaccessible to the ordinary methods of physical examination. When they are displaced or enlarged they present characteristic physical signs. The suprarenal bodies are also beyond the reach of the usual procedures of physical diagnosis. The ureters descend from the hilus of each kidney to enter the bladder at its base. When dilated—hydronephrosis—they form characteristic abdominal tumors.

The Bladder.

When empty this organ lies below the symphysis pubis; when distended it gives rise to a globular area of dulness in the hypogastrium. In some neglected cases of urethral stricture or enlarged prostate an overdistended bladder forms a large fluctuating tumor, reaching as high as the umbilicus and inclining somewhat more to one side of the median line than to the other.

III.

THE EXAMINATION OF THE PATIENT AND CASE-TAKING.

Case-Taking.

An accurate knowledge of the facts in the case constitutes the first requisite to a diagnosis. Those relating to the medical life of the patient and his illness up to the time of his coming under observation are known as the HISTORY OF THE CASE, or the ANAMNESIS; those relating to his immediate circumstances, alike subjective or objective, are described under the heading PRESENT CONDITION, or STATUS PRÆSENS.

The examination to ascertain the necessary facts should be conducted in an orderly and systematic manner. Time is thus saved, a general survey of the clinical phenomena made, and those of chief importance brought into contrast and proper relation with those of subordinate value. Data not otherwise obvious are brought to light and the chances of oversight minimized. Vague and pointless inquiries are omitted. The interrogation is precise and explicit. Above all, leading questions are to be avoided. Running comments in the presence of the patient produce an especially unfavorable effect. Tact and patience are necessary. An examination thus conducted has a favorable influence upon the patient, especially in chronic and difficult cases, and always inspires confidence. The investigation should not be unduly extended or minute. The examination of an experienced and thoroughly trained clinician stands in striking contrast to the vague and unsystematic questions of the beginner. On the other hand, the inquiry may be too concise and brief. The former method has been spoken of as the extensive, the latter as the intensive. The middle course is the best.

There are two principal modes of case-taking, the synthetic and the analytic.

THE SYNTHETIC METHOD.

In the synthetic, sometimes spoken of as the historical method, the inquiry begins with the history of the patient, rather than with his present condition. His place of birth, age, social state, occupation, previous diseases, habits, hereditary and constitutional tendencies are first ascertained, then follows an investigation into the beginning and progress of the present illness. All this constitutes the anamnesis. The status præsens is then considered. The condition of the several physiological systems, the digestive, the circulatory, the respiratory, the genito-urinary, the nervous, and so on, being carefully inquired into in regular order. Finally, the symptoms and signs referable to the organs or structures especially affected are carefully studied. The next step in the process is the diagnosis, upon which the prognosis, treatment and general management of the case depend. Case-taking by this method follows the natural order. It is scientific and useful in obscure cases. The chief objections to it are the time it consumes and the fact that in the progress of the inquiry unnecessary attention must be given to facts which are found later to have little or no bearing upon the patient's present condition.

THE ANALYTICAL METHOD.

In the analytical method the order of procedure is reversed. The principal symptoms are taken as the point of departure for the investigation. The organ or region to which these symptoms are referred is examined by the proper diagnostic measures. The general condition of the patient, his facies, the state of nutrition of his body, his posture, his movements, are carefully observed; meanwhile he is questioned as to the duration and progress of the present illness and an inquiry is made into such facts in his previous history and antecedents as may bear upon the case. The clinical study is then extended, the condition of the other organs investigated, the history of the case more systematically reviewed, an opinion formed as to whether the malady is general or local and a diagnosis reached. This is the plan commonly pursued in ordinary professional work where the data are sufficient for a diagnosis by the direct method, and is available in all cases except those where the symptoms are obscure and ill defined.

QUESTIONS.

Great care is necessary in formulating questions. It is not sufficient to ask the patient if the present illness began with a chill and be content with an affirmative answer. Many patients regard the transient shivering which so often marks the onset of an acute febrile disease as a chill, whereas it is a very different matter from the prolonged and intense rigor that attends the onset of pneumonia or the malarial paroxysm. The physician must be on his guard also in regard to statements made by patients or their friends concerning their previous illnesses. Very often such diagnoses are popular rather than professional, and questions must be so framed as to determine their accuracy. Accounts of influenza, malaria, catarrh of the stomach, rheumatism, and the like cannot be accepted without close investigation into the symptoms, course and duration of the illnesses referred to. The "stomach cough" and "malaria" of the consumptive are familiar to all practitioners. In the matter of hereditary and family tendencies to disease the examination must be conducted with great care. It is no uncommon thing for patients, even those who are well informed and intelligent, to deny the existence of malignant disease, chronic nephritis, a tendency to tuberculosis, and the like, when careful inquiry or the independent statements of their friends render the occurrence of these diseases in the family in the highest degree probable. A patient will affirm that no case of consumption has ever occurred in his family, and upon cautious questioning admit that his father or mother or other near relative suffered from chronic cough, abundant expectoration, blood-spitting, and progressive emaciation. An epileptic will deny the occurrence of nervous diseases, and subsequently admit that near relations have presented the symptoms of hysteria or neurasthenia or been insane. Patients very often withhold in the presence of a nurse or other attendant important facts that they willingly communicate to the physician alone.

RECORDS.

Records should be kept in private as well as in hospital and dispensary practice. How full these should be will depend upon the physician's estimate of the importance of the individual case. Their preparation demands close attention, concise statements, and accuracy. They constitute a permanent store of professional experience for future reference and study. They are of great value in the review of the history of patients previously seen, as an aid in comparing one's personal observations with those of the profession at large, in the preparation of articles for publication, and not infrequently as bearing upon medico-legal cases. They should be preserved in accordance with a uniform plan in books prepared for the purpose, or preferably upon cards of convenient dimensions arranged in cabinets, in the same manner as the index catalogues used in libraries. Uniformity is important. It prevents the oversight of significant facts and facilitates the comparison of cases. The following scheme is suggestive; it may be modified in accordance with individual views:

SCHEME FOR CASE RECORDS.

Case record number.........Diagnosis.........Revise........Result............
Admitted..........Discharged.........(In hospital patients).
Date of examination..........
Name........Age.........Sex........Race.........Place of birth.........Present abode........Former occupation........Present occupation.........Social state.......
Married, single, widowed.

ANAMNESIS.

1. FAMILY HISTORY: Hereditary tendencies; health of parents, brothers and sisters; deaths in family—cause, age.

2. PERSONAL HISTORY: (a) Diseases of childhood; (b) menstruation; (c) pregnancies, miscarriages, date of last confinement; (d) previous illnesses or injuries; (e) habits—regularity of meals, kind of food, method of eating; bowels; sleep; habitual or occasional physical or mental overexertion; tobacco; alcohol; narcotics.

3. PRESENT ILLNESS: (a) Date of onset; supposed exciting cause; exposure to contagion; prodromes; initial symptoms; course of the attack; previous treatment. (b) Antecedent derangements of health not amounting to positive disease, appetite, pain, cough, disturbances of sleep, headache, etc.

STATUS PRÆSENS.

A. GENERAL APPEARANCE: Expression, height and weight, musculature, bony structure, panniculus adiposus; posture in bed; movements, gait and station out of bed; temperature; pulse; respiration; color and condition of the skin; perspiration; œdema; eruptions; psychical condition; sensations and complaints; delirium; convulsions; stupor; coma.

B. PARTICULAR PHENOMENA: Symptoms and signs relating to special structures, organs and functions.

1. The Digestive Apparatus: Inspection of the mouth, tongue and gums; tonsils and pharynx; palpation of the abdomen, its form and contour, visible peristalsis, tenderness upon pressure, resistance, tumors; percussion and palpation of the stomach and intestines, liver, gall-bladder, spleen; inspection of vomited matters and fæces.

2. The Circulatory Apparatus: Inspection and palpation of the cardiac area; visible and palpable pulsation; thrill; precordial prominence; position of the apex; percussion and auscultation of the heart; the pulse-frequency, rhythm, fulness, tension; condition

of walls of arteries; venous pulsation; capillary pulse; liver pulsation; auscultation of the arteries and veins; arterial pressure, positive and negative; examination of the blood, etc.

3. The Respiratory Apparatus: Nose, mouth, and larynx; cough and expectoration; chest and lungs—character of the respiration, dyspnœa, stridor, Cheyne-Stokes respiration; contour of the thorax; local, lateral or bilateral retraction or expansion; respiratory excursus; fremitus; local and general physical signs obtained by percussion, auscultation, and mensuration; the cyrtometer.

4. The Genito-Urinary Apparatus: Palpation of the kidneys and bladder; percussion of the bladder; retention of urine; suppression; frequency of micturition; pain; quantity of urine; total amount for twenty-four hours; disturbance at night; chemical and microscopic examination of the urine; sexual organs.

5. The Nervous System: Intelligence; mental state; subjective sensations; sleep, gait, station, reflexes, tremor, convulsions, spastic conditions, paralysis; aphasia and other disorders of speech; derangements of sensation; the organs of special sense.

6. The Osseous System—Bones and Joints: General and local changes in the skeleton; cranium, spine, thorax, pelvis, long bones, extremities; striking deformities; the joints; size and shape, color, pain, degree of impairment of function, fixation, disintegration.

7. The Tegumentary System: Itching, burning, tension, pain, inflammatory phenomena; presence and character of eruptions, macular, papular, vesicular, pustular; uniformity; polymorphism; hypertrophy and atrophy; cicatrices; pigmentary changes; animal and vegetable parasites; subcutaneous structures; enlargement or atrophy of thyroid body; lymph nodes; constitutional disturbances.

Diagnosis; Prognosis; Treatment; Subsequent observations.

The results of special clinical and laboratory examinations are to be incorporated under the appropriate headings. Among these are rhinoscopic and laryngoscopic, ophthalmoscopic and otoscopic examinations; hæmatologic investigations; the chemical and microscopic examination of the gastric contents, vomited material, and the stools; of expectorated matters; bacteriologic examinations of the blood, sputum, secretions, exudates, etc., by the methods of staining, culture, and inoculation; examination of the rectum by the finger, the speculum, and by inflation; cystoscopy; special examination of the genital organs in both sexes, examination of the fluids obtained by exploratory puncture, and examination by the X-rays.

In febrile cases temperature charts should be preserved with the records, and superficial deformities, as swelling or retraction, as well as changes in the viscera revealed by the various methods of diagnosis, may be indicated upon outline clinical diagrams and incorporated in the notes. Changes of contour, glandular enlargements and topographical lesions, such as local consolidations and cavity formation in the lungs, cardiac dilatation or hypertrophy, pleural and pericardial effusions and the resulting displacement of adjacent viscera, enlargement of the liver and spleen, dilatation of the stomach and displacement of the abdominal organs may in this manner be more or less accurately delineated. The location of tumors and circumscribed exudates may also be indicated, and in the case of the nervous system the extent and distribution of areas of disturbance of sensation and other phenomena.

Some further explanation of the bearing of the facts noted in the anamnesis upon the mental processes by which a diagnosis is reached may be of service to the student.

Age. The age is important. Each period of life has its peculiar susceptibility to morbid influences. In the new-born, congenital defects, the results of the accidents of parturition, diseases arising from faulty management of the cord, those directly transmitted from the mother, and those produced by improper diet and unhygienic surroundings are common. In childhood, anatomical peculiarities of the growing organism and the sensitiveness of physiological processes to external impressions give rise to special predispositions to disease. Thus, the ready proliferation of the lymph tissues explains the frequent occurrence of respiratory obstruction in the nasopharynx from adenoid hypertrophy, while the narrowness of the larynx accounts for the gravity of catarrhal and infective processes involving that organ, and the great vascularity and rapid overgrowth of the epithelium of the bronchi when irritated explain the peculiar liability of children to bronchitis and bronchopneumonia. In the instability of the nervous system in children we find a ready explanation of their liability to fever, its high range and rapid fluctuations, and to various reflex disturbances, and in the absence of acquired immunity, an explanation of the wide prevalence among them of the transmissible infections, as the exanthemata, which are spoken of as the diseases of childhood. In adolescence, hereditary predispositions begin to show themselves, as in the occurrence of tuberculosis and of epilepsy or other nervous affections. The late sequels of infantile diseases, as chronic valvular trouble following rheumatic endocarditis, or chronic nephritis subsequent to scarlatina, often now appear. Changes in the environment of the individual subject him to special pathogenic influences, and pleurisy with or without effusion,

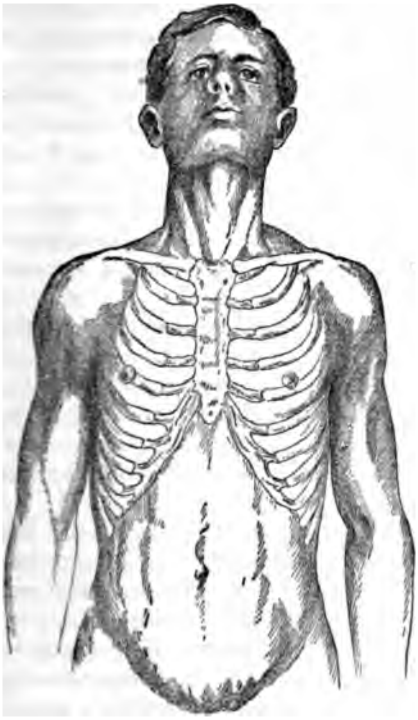


FIG. 27.—Clinical diagram.

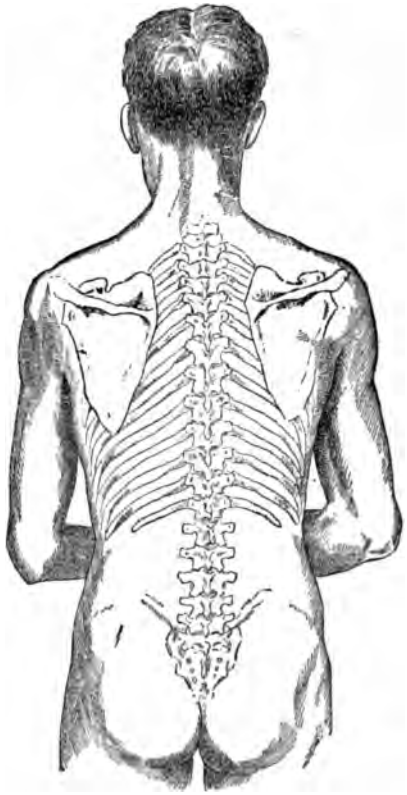


FIG. 28.—Clinical diagram.

pneumonia, and enteric fever are common. The middle period of life is especially prone to diseases that result from occupation, examples of which are lead intoxication, caisson disease, and scrivener's palsy, to those which result from the habitual use of narcotics, as gastric catarrh, hepatic cirrhosis, and alcoholic neuritis, to those resulting from the stress of life and anxiety, among which may be named cardiac hypertrophy, the neurasthenias and other nervous diseases and insanity. It is in this period that hereditary and acquired tendencies to sclerotic changes in the vessels and in the nervous system begin to develop and that diabetes and the paroxysms of gout commonly first show themselves. Later in life the indications of progressive degenerations become more marked. The wrinkled skin, the failing sight and hearing, the feeble heart, winter cough, and renal inadequacy are the indications of sclerotic and nutritive changes which are more apparent in the rigid, tortuous, or atheromatous superficial arteries. This is especially the period of apoplexy, chronic bronchitis, diabetes, cystitis from hypertrophied prostate, Parkinson's disease and the special infections erysipelas and pneumonia, which are frequently terminal events. In general terms the evolution of life is the period of infections, the involution the period of degenerations; but in pathology age cannot be measured by years, and the significant saying that "a man is as old as his arteries" has become a modern medical aphorism.

Physiological Epochs.—The epochs of life are also marked by special liability to disease. Thus at the first dentition nutritional diseases and gastro-intestinal troubles are common; at puberty, chlorosis and hysteria; at the menopause, hysteria, obesity, and arthritis deformans. It is to be noted, however, that the maladies of these physiological epochs are not the direct result of functional changes, but are the outcome of previous morbid conditions or tendencies.

Sex. — Sex is likewise important. In early and advanced life the sexes are equally liable to disease. Women between the age of puberty and the menopause are exposed to the danger of many accidents and diseases peculiar to the anatomical and physiological development connected with the sexual life and child-bearing. Consideration of these matters properly belongs to gynæcology and midwifery. Sedentary living, the monotony of the household, and depressing moral influences also act as causes of disease in women. Hysteria, neurasthenia, and special forms of insanity occur. These peculiarities do not, however, carry with them an exemption from other pathogenic influences, and among the peasantry of those countries where the women largely engage in the same occupations as the men they suffer, in addition to their own peculiar disorders, from the maladies of the other sex and practically to the same extent. In more enlightened districts and among the upper classes of society women escape many risks of disease to which men are exposed. In the male sex occupation, exposure, the strenuous life, and self-indulgence are common causes of disease, hence the more frequent occurrence of plumbism, farcy, pneumonia, chronic arthritis, gout, tabes, and alcoholism. As a consequence, arteriosclerosis and atheroma are more marked in men than in women at advanced age.

Race and Nationality.—These points demand consideration in the anamnesis. The peculiar liability of the Hebrew to diabetes and neurasthenia; of the negro and mulatto to tuberculosis, and the relative immunity of the former to malaria and yellow fever; the prevalence of beriberi among the oriental races, of leprosy in Scandinavia, the Sandwich Islands, and the West Indies; and the frightful ravages of tuberculosis, syphilis, and alcoholism among the Indians of North America are well-known facts.

Nativity.—The place of birth and residence frequently shed light upon an obscure case, as in ill-defined malaria, the malarial cachexia, goitre, cretinism, and leprosy. A knowledge of the district or locality of the patient's present residence, the situation of his home, its sanitary conditions and surroundings, the source of the water supply, and the disposition of the sewage may shed light upon the diagnosis.

Occupation.—The occupation of the patient demands careful investigation. The habitual over-use of certain muscles, and exposure to particular irritants or poisons or an atmosphere laden with minute mineral or metallic particles or chemicals, or to infections peculiar to certain crafts, cause definite diseases. Examples of such affections are writer's cramp, anthracosis or miner's consumption, chronic phosphorus poisoning among workmen engaged in the manufacture of matches, malignant pustule or woolsorter's disease, and glanders. It is necessary to inquire carefully into former occupations as well as the present; thus chronic bronchitis with bronchiectasis may have had its origin in the inhalation of the dust caused by stone-cutting—an occupation long abandoned by the patient. In those occupied in professional or literary work functional derangements of the stomach, constipation, and insomnia are common. Even amusements may be the cause of disease, as in the golfer's back and the heart-strain of the athlete.

Heredity.—The family history has a very important bearing upon the diagnosis, especially in chronic diseases. It is difficult to frame a satisfactory definition for heredity, but we know that traits and lineaments are transmitted from parents to children through the generations, and we occasionally observe in a son who has his mother's features some trick of expression that makes his resemblance to his father for the moment almost startling. So too are transmitted from one generation to another tissue peculiarities and constitutional tendencies to disease. The inquiry into the family history must be, as has been pointed out in a previous paragraph, conducted with tact and caution. Blunt inquiries in regard to "consumption," "cancer," "Bright's disease," or "insanity" irritate the patient and usually elicit vague replies or absolute denials. A patient should be asked if his parents are living and in good health; if not in good health, the symptoms and duration of the illness; if dead, the cause of death and the age at which it occurred. He should be questioned as to the number of his brothers and sisters, their health, and the cause of any deaths that may have occurred among them. It is very important to learn whether or not deaths in the family have been the result of acute or chronic disease. The inquiry may be extended to the preceding generation and collateral branches of the family. Diseases, it is true, are conveyed by hereditary transmission, but their number is comparatively few. Hæmophilia is a striking example. Syphilis is very commonly thus transmitted. When the mother has contracted an acute infection, as measles or enteric fever, the child may be born during the period of incubation or with the symptoms of the disease already manifest. A number of nervous diseases are clearly hereditary. As examples may be mentioned progressive muscular atrophy, hereditary chorea, Friedreich's ataxia, and migraine. The definite symptoms may not show themselves for some years after birth, in some cases not until adult life. Much more commonly it is the predisposition that is transmitted. This is especially the case in tuberculosis. The peculiar exposure of the young infant to infection from a tuberculous mother and the length of time that the tuberculous lesions in many instances remain localized render it in the highest degree probable that the predisposition to tuberculosis rather than the disease itself is hereditary. This view is confirmed by the results of pathological and bacteriological investigations. The direct transmission of tuberculosis from the mother to the fœtus in the human being is of uncommon occurrence. The doctrine of the direct hereditary transmission of tuberculosis, so long entertained but now fortunately abandoned, was a stumbling block in the way of the recognition of the infectious character of this disease. That the predisposition rather than the disease is hereditary is also true of cancer. The occasional occurrence of chronic Bright's disease in nearly every member of a family in two or three generations, usually first showing itself in adolescence or early adult life, must be attributed to hereditary defects of the renal and vascular tissues, while faults of metabolism, the constitutional tendency to which is transmitted from father to son, bear a direct etiological relation to gout and its associated cardiovascular and renal changes. The development of forms of insanity in successive generations of a family, usually at the physiological epochs of life, often not until late middle age, must likewise be attributed to hereditary defects of nervous and mental organization.

A further peculiarity in regard to the hereditary transmission of disease is to be found in its diverse manifestations among various members of a family. The radical defect or susceptibility may find expression in pathological conditions which are allied but which have wholly different symptoms. Thus the tendency to deranged metabolism and arteriosclerosis may in one show itself in contracted kidney and hypertrophied heart; in another in disease of the aorta or angina pectoris; in a third in gout, renal calculus and gravel, or yet again in early cerebral hemorrhage or thrombosis. The neuropathic constitution may manifest itself in one member of a family in forms of neuralgia, neurasthenia, or hysteria; in another in the development of epilepsy, and in a third in the guise of hypochondriasis or insanity. The family susceptibility to certain infections may reveal itself in different individuals in recurrent attacks of tonsillitis or rheumatism, chorea or chronic valvular disease; or the susceptibility to tuberculosis, on the one hand in pulmonary consumption, on the other in tuberculosis of the bones and joints or glandular disease, or finally in the implication of the meninges, pleura, or peritoneum.

Immunity may be transmitted by inheritance as well as the predisposition to disease. There are families and individuals who possess a remarkable natural immunity against the exanthemata. This is especially true in regard to scarlet fever. When we consider the wide prevalence of pulmonary tuberculosis and the diffusion of its cause in the centres of population and certain districts and houses, and the fact that so large a proportion of individuals and families constantly exposed to the inhalation of an atmosphere containing the tubercle bacilli escape the disease, the common existence of a natural immunity which is frequently transmitted by inheritance becomes evident. The predisposition to tuberculosis is far less general than that to scarlatina and measles. The occurrence of personal peculiarities and morbid tendencies in an individual which were not manifested in his parents but existed in their ancestors is known as atavism. In rare instances, and especially in cases of nervous disease and insanity, this condition is important in the anamnesis. Curious facts in regard to the duration of life are occasionally observed. There are families in which in successive generations few members survive the early middle period of life. In such instances death is very often due to an acute disease not always the same. On the other hand, all the members of certain families reach an advanced age, the exceptions being where death is due to accident or violence.

Medical History.—The personal history is essential to a diagnosis in the broad sense. A knowledge of the significant facts in the past life of the patient may clear up a doubtful case. The present disease may be a late sequel of some previous illness, as bronchitis or emphysema after whooping-cough, or an obscure manifestation of one of the exanthemata which the patient escaped in childhood, as scarlatina in the adult with fever of moderate intensity and an irregular patchy eruption, or it may be the expression of a peculiar constitutional susceptibility, as tonsillitis, rheumatic fever, or chorea, from which the patient has suffered on previous occasions. In this connection it is to be borne in mind that many of the acute infectious diseases, and especially the exanthemata, result in an acquired immunity which usually lasts throughout life, hence second attacks are exceedingly infrequent, while the immunity conferred by other infections, for example rheumatic fever, erysipelas, croupous pneumonia, and diphtheria, is incomplete and of limited duration, so that many individuals suffer from repeated attacks of these diseases. In acute febrile attacks and in the presence of epidemics careful inquiry as to exposure to the contagion must be made. The period of incubation and the occurrence of prodromal symptoms are to be taken into consideration. In women abnormal menstruation, the accidents and diseases of pregnancy, the occurrence of miscarriages, too frequent child-bearing and prolonged lactation may be the cause of serious impairment of health or of actual disease. These matters must be carefully inquired into. In exceptional cases, especially in aggravated and intractable functional nervous diseases, it becomes necessary to inquire more closely into the sexual life of the patient. The investigation must be conducted with great delicacy and discretion. The part played by vicious practices and excesses in the production of such diseases must be ascertained. It is necessary also to learn whether or not the patient has suffered from venereal infection, the date of its occurrence, the nature, character, and duration of the primary symptoms, the presence or absence of secondary lesions, and the treatment. Gonorrhœa is not always merely a local affection. The frequency with which it is followed by stricture is well known, but the symptoms of the latter condition may first show themselves after the lapse of years. Local abscess formation, acute and chronic cystitis and pyelitis also occur. The immediate recognition of the specific nature of gonorrhœal ophthalmia, whether in the new-born or in the adult, is a matter of overwhelming importance. The diagnosis of many a case of disabling and stubborn arthritis is made clear by a knowledge of gonorrhœal infection. Nor is the fact to be overlooked that endocarditis, both in its benign and malignant forms, may be a secondary process. In women the history of primary gonorrhœa is very often obscure. Tubal disease and other pelvic inflammations, only to be relieved by the knife of the gynæcologist, are common results of the extension of the infection. A dissolute life on the part of the patient is presumptive evidence of the nature of the process. There is also gonorrhœa insontium; a virtuous wife may suffer. The protean manifestations of syphilis are to be borne in mind. The symmetrical arrangement and sequence of the early cutaneous lesions, their later polymorphism and irregular distribution, the buccal and anal mucous patches, the adenopathy, the obscurity of the visceral and nervous phenomena, their irregularity and chronicity, are all to be considered in the diagnosis of an obscure case. The presence of the specific organism—spirochæta pallida—is conclusive. Where syphilis is suspected in a family, we must inform ourselves as to whether or not a mother has aborted, especially in her early pregnancies, or has had later a series of abortions or still-born children, and as to snuffles and cutaneous eruptions, especially on the buttocks, in her new-born children, and corneal opacities, interstitial keratitis, Hutchinson's teeth, and arrested development or nervous diseases in those who have survived. Nor must the physician overlook the fact that many innocent persons contract syphilis. Not only the blameless wife but also the unsuspecting girl, from the kiss of her betrothed, may become the victim to this disease, while the methods of accidental inoculation are innumerable. Familiar examples are to be found in the chancre upon the hand of the surgeon or accoucheur, or upon the lip or tongue of the incautious borrower of a pipe from an infected friend. When matters of this kind concern members of a family, the physician cannot be too guarded in respect to the way in which his questions are framed or in his statements to a husband or wife. Suggestive questioning or injudicious statements may seriously aggravate existing troubles. If definite communications become necessary, his knowledge of the circumstances will enable him to decide whether it is best personally to assume the whole responsibility or to invite a colleague of high reputation to share it with him.

The history of a surgical operation and the conditions which led up to it, as well as its results, are important. The patient's present condition may be due to a recurrence of the original trouble, or, as in the case of an abdominal operation, to the development of adhesions or constricting bands.

Personal Habits.—The habits must be closely studied. Important information bearing upon the diagnosis may often be obtained by directing the patient while continuing his ordinary method of living to keep a record of the hours at which his meals are taken, the kind and quantity of food and drink, the action of his bowels, the hours and character of sleep, and his various occupations and amusements, which may be submitted at a subsequent consultation. The causal relation of improper clothing to bronchopulmonary affections, of badly regulated work and sleep to neurasthenic conditions, of injudicious or irregular eating to gastro-intestinal troubles, of the abuse of alcohol to nervous diseases and cirrhosis of the liver, of excess in tobacco to irritable heart and amblyopia, will guide us in the inquiry. Late hours and dissipation, in fact all matters which enter into consideration from the stand-point of the moral hazard of the insurance companies, have a most important bearing upon diagnosis.

Present Illness.—The history of the present illness must be systematically investigated and its symptoms recorded in chronological order from the onset to the time of the patient's coming under observation. It is important to learn if possible the effect of treatment. The disappearance of a rash after mercurials or the subsidence of headache after continued large doses of the iodides constitutes presumptive evidence in favor of syphilis. The failure of quinine to prevent the recurrence of chills renders the diagnosis of malaria improbable, or of the proper administration of suitable preparations of iron in full doses to correct the pallor, breathlessness upon exertion, and headache of a highly anæmic young woman militates against the diagnosis of chlorosis. Much allowance must be made for the statements of patients both as regards the symptoms of the illness and their reports of previous treatment and the opinions of physicians whom they may have consulted. In many cases the unravelling of a diffuse and inconsequent story can only be accomplished by the exercise of skill and patience. On the other hand, the history communicated by intelligent persons is often curiously succinct and clear. Frequently by reason of the patient's mental condition no account of the illness can be obtained. In some cases it often happens that very little information can be gleaned from the bystanders. In hospital practice the admission of ambulance cases gravely ill, of whose previous condition nothing whatever can be learned, is a matter of daily occurrence.

Duration.—Of first importance is a knowledge of the duration of the illness, since it enables us at once to form an opinion as to whether the disease should be referred to one or the other of the two general groups of acute or chronic maladies. The fact is, however, not to be overlooked that acute symptoms may be the manifestation of an unsuspected chronic affection, as sudden loss of vision or convulsions in nephritis, angina pectoris in disease of the heart and aorta, or perforation phenomena and peritonitis in peptic ulcer of the stomach and duodenum. The mode of onset next demands our attention. In chronic cases we seek information as to whether the present illness developed insidiously or abruptly upon a condition of previous good health, or followed an acute illness, and whether its course has been gradual and progressive or interrupted by periods of improvement; in acute cases whether the attack developed insidiously, as in the case of enteric fever, or abruptly, as in influenza or typical croupous pneumonia, and whether or not prodromes occurred. It is next in order to ascertain the prominent symptoms of the disease, the region or organ to which they have been referred, whether they have been continuous, intermittent, or paroxysmal, and any changes in the patient's appearance or condition, of which he may or may not be aware, that have attracted the attention of his friends. Finally, important information is often reached by due consideration of the views of the patient or others relating to the cause of his illness.

Status Præsens.—The investigation of the present condition of the patient must also be conducted in an orderly and systematic manner. The subjective sensations are carefully considered. No complaint of the patient, however trifling, is to be wholly disregarded. The objective symptoms must be studied with equal care. Every fact is to receive proper consideration. Due regard must be paid to the feelings of the patient. Abruptness and all appearance of haste or harshness are to be avoided. The interview must not seem too business-like. The clothing, whether in the consulting room or at the bedside, must be so arranged as to facilitate the examination. No physical exploration of the thoracic or abdominal organs can be made without proper access to the regions to be studied; mistakes from a disregard of this rule are of daily occurrence. In diseases of the heart, lungs, or great vessels it is necessary to inspect the uncovered chest; palpation must also be performed upon the bare surface; percussion and auscultation upon the bare skin or more conveniently in most cases through a towel or the single layer of a smooth under-vest. In order that the influence of gravity upon the abdominal viscera may be learned or to study the station and gait, the patient must rise from bed. If there are symptoms referable to the spine, the clothing must be removed and the patient examined in the erect, sitting, or recumbent posture, in the last instance not in bed but upon the firm, smooth surface of a suitable table; the effect of various movements is studied and the condition of the muscles and joints. Accurate measurements of parts, preferably in centimetres, are essential where there is a departure from normal standards or asymmetry. We measure and note the circumference of the head in hydrocephalus, the chest on quiet breathing, on full held inspiration and on forced expiration, its lateral circumferences for comparison, its contour by means of the cyrtometer, and we may measure diameters of the head and chest by means of calipers. It frequently, especially in the case of ascites and tumors, is desirable to take the circumference of the abdomen The muscles in relaxation and contraction are studied by the hand and one side is compared with the other. Where necessary the circumference of the limbs is measured at the same point upon the two sides. Where symptoms relating to the brain or spinal cord dominate the clinical picture, the examination must be made with especial attention to the details bearing upon the localization of the lesions. Specimens of the urine must be obtained for examination as a matter of routine in all cases. The diagnosis of obscure conditions, the symptoms of which are referred to the nervous system, digestive organs, or general condition of the patient, frequently depends upon the result. The discovery of chronic disease of the kidneys or the presence of sugar in the urine as the outcome of investigations made upon application for life insurance is a matter of very common occurrence in middle-aged men who regard themselves as in excellent health.

In general the examination should be methodically conducted in accordance with the foregoing scheme, prominence being given in the record of the case to the symptom-complex which bears directly upon the diagnosis.

Abbreviations.—Time and space may be saved in case-taking by the use of abbreviations. Thus:

f & m l & w—father and mother living and well.

b 3; *2 d in infancy;* 1 *l & w*—three brothers; two dead in infancy; one living and well.

s 2; 1 *d at* 7 *sc fever;* 1 *at* 10 *acute nephritis.*

w & s till 18 *then ent fever; l crural phlebitis; elas stk still*—well and strong till 18, then enteric fever; followed by left crural phlebitis; still wears elastic stocking.

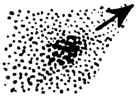
Epigast pain p c; occas v; blood 12 *mos &* 1 *mo ago*—Epigastric pain after food; occasional vomiting; hæmatamesis one year and again one month ago.

D r u a; def expn; br-vesic resp; crep râles—Dulness right side upper lobe, anteriorly; deficient expansion; broncho-vesicular respiration; crepitant râles.

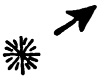
Tend r l q; circ D; 3d d of attack; n & v; T. 101°—Tenderness in right lower quadrant; circumscribed dulness; third day of attack; nausea and vomiting.

Many similar abbreviations, at once familiar to the writer and intelligible to any trained clinician, will suggest themselves.

Murmurs may be shown on the clinical diagrams (Figs. 27 and 28) by stippling or washes, the point of maximum intensity being most deeply colored and the direction of propagation shown by an arrow:

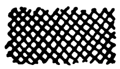


or more simply by a many-pointed star to indicate the point of maximum intensity and an arrow the direction; thus

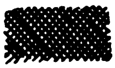


These signs should be drawn in a different color from that of the ground plan, red if the latter is black, or *vice versa.*

Dulness may be indicated by cross hatching; its degree by closeness of the mesh; thus



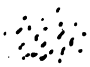
Relative dulness.



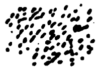
Marked dulness

Flatness by solid color; thus

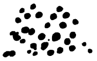
Râles by dots, their size and abundance corresponding to the physical signs; thus



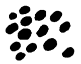
Crepitant.



Subcrepitant.

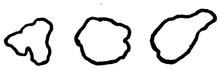


Small mucous.

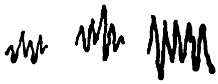


Large mucous.

Cavities by irregularly outlined spaces; thus



Friction sounds by zigzags, the extent and coarseness of which indicate the distribution and intensity of the rub; thus



PART II.

OF THE METHODS AND THEIR IMMEDIATE RESULTS.

I.

MEDICAL THERMOMETRY.

THE art of taking and recording the temperature of the body is called medical thermometry. The instruments used are known as clinical thermometers. They are marked off in degrees upon the glass, and each degree is subdivided into fifths, so that the readings may conveniently be recorded in fractions of the decimal system. The thermometers commonly used in the United States and Great Britain are marked in degrees of *Fahrenheit's* scale; those used in Europe are graduated according to the *Centigrade* scale. The scale of *Réaumur* is rapidly going out of use, but is still employed in some parts of Europe. On the scale of Fahrenheit the distance through which the mercury rises from zero to the boiling-point of water is divided into two hundred and twelve degrees, of which the thirty-second marks the melting-point of ice. Between the melting-point of ice and the boiling-point of water there are one hundred and eighty degrees (32° + 180° = 212° F.). The melting-point of ice is taken as zero in the Centigrade scale and in that of Réaumur, but in the Centigrade the boiling-point of water is at one hundred (100° C.), while in Réaumur's it is at eighty (80° R.). The relation of the three scales to each other is, therefore,—

F.	C.	R.
9	5	4

To convert recordings of the Fahrenheit scale into Centigrade degrees,—

Subtract 32, multiply by 5, and divide by 9; thus: 98.6 — 32 = 66.6 × 5 = 333.0 ÷ 9 = 37. That is, 98.6° F. = 37° C.

To convert Centigrade degrees into Fahrenheit degrees,—

Multiply by 9, divide by 5, and add 32; thus 37 × 9 = 333 ÷ 5 = 66.6 + 32 = 98.6. That is, 37° C. = 98.6° F.

The Centigrade scale is more convenient than that of Fahrenheit, and many physicians in this country prefer to use it. The following table of approximate equivalents may prove of use:

96.0° F. = 35.5° C.
96.8° F. = 36.0° C.
97.8° F. = 36.0° C.
98.0° F. = 36.6° C.
98.6° F. = 37.0° C.
99.0° F. = 37.2° C.
99.5° F. = 37.5° C.
100.0° F. = 37.8° C.
100.4° F. = 38.0° C.
101.0° F. = 38.3° C.
101.3° F. = 38.5° C.
102.0° F. = 38.9° C.
102.2° F. = 39.0° C.
103.0° F. = 39.4° C.
103.1° F. = 39.5° C.
104.0° F. = 40.0° C.
104.9° F. = 40.5° C.
105.0° F. = 40.5° C.
105.8° F. = 41.0° C.
106.0° F. = 41.1° C.
106.7° F. = 41.5° C.
107.0° F. = 41.6° C.
107.6° F. = 42.0° C.
108.0° F. = 42.2° C.
108.5° F. = 42.5° C.
109.0° F. = 42.8° C.
109.4° F. = 43.0° C.
110.0° F. = 43.3° C.
111.2° F. = 44.0° C.

Seasoning.—As thermometers after a time give readings that are slightly too high, in consequence of the gradual contraction of the glass, it is necessary at long intervals carefully to compare them with a standard instrument. This is done at the public observatories, to which any will send them. This contraction of the glass is called "seasoning," and goes on very slowly. After two or three years it practically comes to an end, and the thermometer is then seasoned.

Description of Thermometers.—Clinical thermometers as at present made are of the kind known as maximum, or self-registering; that is, a small portion of the mercury is separated from the main bulk of it, or separates itself from it as it contracts, by reason of a device in the twist of the tube, in such a way that it remains in position in the tube when the temperature falls, until shaken down, and thus indicates the highest temperature reached during the observation. The separated portion of the mercury is known as the "index." The reading is taken from the upper end of the index, which is then shaken down by a quick motion of the wrist, such as is made in cracking a whip, the thermometer being held by its upper end. Before taking the temperature the index should be below 95°. The best clinical thermometers are now made with a curved surface, which, acting as a lens, magnifies the width of the mercury; and with a flattened back, which lessens the danger of breakage from rolling. Aseptic thermometers have an outer glass tube encasing the engraved scale so that the external surface is perfectly smooth.

Technic.—The object being to measure the internal temperature, the thermometer must be placed in such a position that the tissues of the body completely surround its bulb. The positions available are the armpit, or axilla, the mouth, the vagina, and the rectum. The fold of the groin, when the thigh is bent up or flexed over the abdomen, is in infants also occasionally used.

The axilla is usually selected. If very moist, it should be dried with a towel before the instrument is introduced; or, if dry and harsh, it must be bathed with warm water and then dried. There is no difference in the temperature of the two armpits under ordinary circumstances. The bulb of the instrument must be placed deeply in the hollow and the arm brought well across the chest. Care must be taken that no fold of clothing interfere with the contact of the instrument with the skin. Some thermometers are more sensitive than others; that is, they act more quickly. The mercury rises rapidly at first, then more slowly. Thick thermometers require five minutes to record the maximum temperature, but the best instruments now made reach the highest point in about two minutes. In the rectum or vagina less time is required.

When the temperature is taken in the mouth the bulb must be placed under the tongue and the lips closed about the stem, the patient breathing through his nose. It is an excellent plan to dip the instrument in water and wipe it with a clean napkin in the presence of the patient both before and after using it in the mouth. It is not safe to take the temperature in the mouth either in young children or in conditions of delirium. When the patient is in an insensible state, or when doubts arise as to the correctness of an axillary observation, the rectum or the vagina may be used for applying the thermometer, and with self-registering instruments this plan involves no exposure of the person. In European countries the common custom is to take the temperature in the rectum. In restless children care must be taken to prevent the instruments being broken, and in all cases to prevent a short thermometer from slipping entirely into the bowel, from which it might be difficult to extract it. The temperature may be rapidly taken in unmanageable children by means of an old-fashioned thermometer which is not self-registering, by cautiously warming it until the mercury reaches a very high point, say 108°, and then quickly placing it in the armpit. The mercury falls rapidly to the temperature of the patient's body and then stops.

Frequency. — It is desirable to take the temperature at least twice daily, the best times being between seven and eight in the morning and about eight in the evening. The observations must be repeated at the same hours each day. In cases characterized by great or sudden variations of temperature, by very high temperature, or when the influence of treatment upon the fever is being closely watched, observations must be made at shorter intervals of time, and it may become necessary to take the temperature as often as every hour.

Abnormal Temperatures.

The temperature in disease may range below or above the normal. Sudden falls of temperature in fever are very significant; just as are abrupt rises from the temperature of health. The following terms are used to indicate the general condition of the patient in abnormal ranges of temperature:

Below the Normal.	F.		C.
a. Temperature of collapse	Below 96.5°		35.8°
b. Subnormal temperature	96.5°— 98°		35.8°—36.7°
c. Normal temperature	98° — 99.5°		36.7°—37.5°
Above the Normal.			
d. Subfebrile temperature	99.5°—100.5°		37.5°—38.1°
e. Moderate febrile temperature	100.5°—102°	A.M.	38.1°—38.9°
(Mild pyrexia)	102.2°—103°	P.M.	39° —39.5°
f. High febrile temperature	102° —104°	A.M.	38.9°—40°
(Severe pyrexia)	104° —105.8°	P.M.	40° —41°
g. Intense febrile temperature (Hyperpyrexia)	105.8°—110°		41° —43.3°

The range of deviation from the normal within the limits of which life can be maintained for brief periods is comprised between 92° F. and 110° F. A temperature of 95° F. on the one hand or of 106° F. on the other, already indicates great danger, especially if it be prolonged, and beyond these limits in both directions the danger to life speedily becomes extreme.

(a) Temperature of Collapse or Shock. — A considerable and rapid fall of temperature attends the collapse which sometimes occurs during or towards the close of some of the essential fevers. In enteric fever this condition may be produced by hemorrhage, or by sudden peritonitis due to perforation, or in consequence of sudden failure of the heart. The last of these accidents is liable to occur in any very grave case of fever, and occasionally follows the critical fall of temperature which occurs in pneumonia, relapsing fever, and more rarely in other febrile diseases.

Very low axillary temperatures are met with in the stage of collapse in the algid or cold stage of cholera, the internal temperature as indicated by the vagina or rectum remaining high. Great depression of the general temperature occurs in the collapse produced by various poisons, and especially by large quantities of alcohol. The temperature is apt to fall considerably below the normal in ordinary deep alcoholic intoxication, especially if the patients have been exposed to cold and wet.

(b) **Subnormal Temperature.** — This condition attends considerable losses of blood; starvation from any cause; the wasting of certain of the chronic diseases, such as cancer of various organs; some diseases of the brain and spinal cord and the later stages of chronic diseases of the lungs and heart, especially when accompanied by dropsy.

The temperature is very apt to reach subnormal ranges in the morning for a few days at the termination of febrile disorders.

(c) **Normal Temperature.**—If in the course of a continued fever, as enteric, the temperature, which has been elevated two or three degrees or more, *suddenly* falls to normal or near it, though not below, this in itself is significant of something wrong, and may even acquire the importance of the "temperature of collapse," as indicating internal hemorrhage, perforation, or failure of the heart.

(d) **Subfebrile Temperature.**—Slight elevations of temperature often accompany trifling and transient disturbances of the general health, especially in children. They are also observed at the beginning of gradually developing fevers, as enteric, and at the close of slowly subsiding febrile conditions. In obscure chronic cases they are of importance as indicating the existence of actual disease which may not manifest its ordinary symptoms.

(e) **Moderate Febrile Temperature.**—When the morning temperature reaches 101°–102° F. and the evening shows a further increase of one or two degrees, we have to do with actual fever. So long, however, as the temperature does not exceed these limits, there is no serious danger from the fever process itself.

(f) **High Febrile Temperature.**—When the temperature in the morning is above 102°–104° F. and in the evening reaches or ranges higher than 104.5°, the case becomes serious from the intensity of the fever alone, and active treatment becomes imperative. High fever is unattended by immediate danger to life if it be transient, but when prolonged it is ominous. A temperature of 105° or even 107° in the hot stage of an ague, when the whole attack lasts but a few hours, is much less dangerous than the same temperature occurring, even for a short time, in the course of one of the continued fevers, when the patient's powers of resistance are called upon to withstand some degree of fever for several days or weeks.

(g) **Hyperpyrexia, or Intense Febrile Temperature.**—The temperature reaches 105.8° and continues to rise, or at all events does not fall. The condition is one of extreme and imminent danger to life. The resources of the art of medicine are put to their severest test. Hyperpyrexia often supervenes with great suddenness. Not a moment is to be lost. The most prompt and radical measures to reduce the temperature of the body too often fail to avert the fatal result. This condition has been encountered after injuries to the brain and to the upper part of the spinal cord; in lockjaw; in sunstroke, and very often in the infectious diseases, especially scarlet fever and pneumonia. It sometimes occurs in rheumatic fever, especially after the intensity of the symptoms has begun to subside, or even when the patient is apparently almost well. Hyperpyrexia is often one of the indications of approaching death. Hence, in certain cases the futility of treatment. In such cases a temperature of 110° to 112° is sometimes seen. The temperature sometimes continues to rise slowly for an hour or two after death.

The thermometer may be made to indicate a temperature much higher than that of the patient's body, by friction, or by being slipped against a poultice or hot-water bag, or into a cup of tea, when the attention of the nurse is given to other duties. These tricks are sometimes played by hysterical girls. They are readily detected by repeated observations under the eye of the attendant. A number of cases have been recorded in the medical journals in which excessively high temperatures—120°, 150°, even 170° F.—have been noted and apparently verified by repeated and most careful observations. Many of the patients have subsequently been found to be very clever pretenders and tricksters, but the method by which the high temperatures have been recorded has not been explained. In such cases the temperature should be taken in several different regions, axilla, mouth, rectum, etc., at the same time, and the temperature of the urine when voided.

Transitory Variations.—The temperature of a fever patient may be somewhat affected by excitement, fatigue, or exposure. Hence hospital patients often show for a few hours after admission a temperature higher than subsequently, or, if they have been exposed to cold, lower than really corresponds to their condition.

It is a peculiarity of the state of convalescence from the acute fevers that the temperature, though normal, is disturbed by trifling causes, and may be made to rise two or three degrees by the first visit of a friend, the first solid food, or even by sitting up. Such rises are usually very brief, the temperature quickly falling again to normal. They occasion uneasiness lest they be the beginning of a relapse. On the other hand it occasionally happens that, though all the other symptoms have disappeared and the patient is almost well, the temperature remains subfebrile, and the patient is for that reason alone kept in bed. In such cases all traces of fever vanish upon cautiously allowing the patient to sit up an hour or so each day.

Surface Thermometry.

This method is of inferior value for diagnostic purposes. The bulb consists of a fine coil at right angles to the tube and forming an expanded base for it. Observations may be taken at the same time in corresponding positions on both sides of the body. The general temperature must be noted.

Normal surface temperature (Kunkel). Temperature of room 68° F.—20° C.

	F.	C.
Forehead	93.38–93.92°	34.1–34.4°
Cheek under the zygoma	93.92°	34.4°
Tip of ear	83.84°	28.8°
Back of hand	90.5 –91.76°	32.5–33.2°
Hollow of the hand (closed)	94.64–95.18°	34.8–35.1°
Hollow of the hand (open)	93.92–94.64°	34.4–34.8°
Forearm	92.66°	33.7°
Forearm (higher)	93.74°	34.3°
Sternum	93.92°	34.4°
Pectorales	94.46°	34.7°
Right iliac fossa	93.92°	34.4°
Left iliac fossa	94.28°	34.6°
Os sacrum	93.56°	34.2°
Eleventh rib (back)	94.1°	34.5°
Tuberosity of ischium	89.6°	32.0°
Upper part of thigh	93.56°	34.2°
Calf	92.48°	33.6°

The temperature of the skin is slightly higher over an artery than at some distance from it, over muscle than over sinew, over an organ in activity than when at rest, in the frontal than in the parietal region of the head, and on the left side of the head than on the right.

Local elevation above the general temperature has been noted on the surface of the head in cases of mania and meningitis. Local elevation of the temperature has also been observed in cerebral tumor and abscess. A local rise of temperature also occurs over the painful points in some cases of neuralgia and in areas of superficial inflammation. The surface temperature is increased in the region corresponding to the exudate in croupous pneumonia. Irregularly distributed areas of elevated surface temperature sometimes occur in hysterical persons.

Subnormal temperature may be observed in a limb from which the blood supply is cut off by the tourniquet or obstruction of the main artery, in an œdematous or cyanosed part, and in gangrenous areas. Weir Mitchell called attention to the effect of posture upon local temperature. He found the surface of the dorsum and sole of the foot 0.4° C. to 1° C. cooler in the erect than in the recumbent posture.

Charts.—The temperature must be recorded at once. At the same time a record of the pulse-beats and movements of respiration per minute is to be made. They are to be carefully counted while the thermometer is in position.

Ruled sheets, called "temperature charts," or "clinical charts," are sold in the shops for this purpose. The form here shown will be found very convenient. It may be so kept with little trouble as to preserve in a compact form all the important facts of an acute case, and is equally useful in hospital and in private practice. The ruled space is arranged for twenty-one days by vertical lines, the weeks being divided by heavy lines. The space for each day is again subdivided for the morning and evening record, as indicated by the M and E. At the left margin the purposes of the spaces formed by the transverse rulings are indicated. At the top the number of movements of the bowels; immediately below the quantity of urine passed, which may be recorded in fluidounces or cubic centimetres; then the scale of *Fahrenheit*, with the equivalent *Centigrade* opposite on the right margin. The coarse horizontal line at 98.4° F. indicates approximately the normal. At the bottom are, first, spaces for each day of the disease, then similar spaces divided by a diagonal line for morning (upper, left triangle) and for the evening (lower, right) pulse-rate; below these again corresponding spaces for the respiration-rate, and at the bottom of the chart spaces for the date or day of the month.

Important clinical facts, as "hemorrhage," "convulsions," "suppression of urine," etc., may be noted at the time of their occurrence between the vertical lines on the right or upper side of the chart in the position indicated by the arrows, under the words "clinical memoranda."

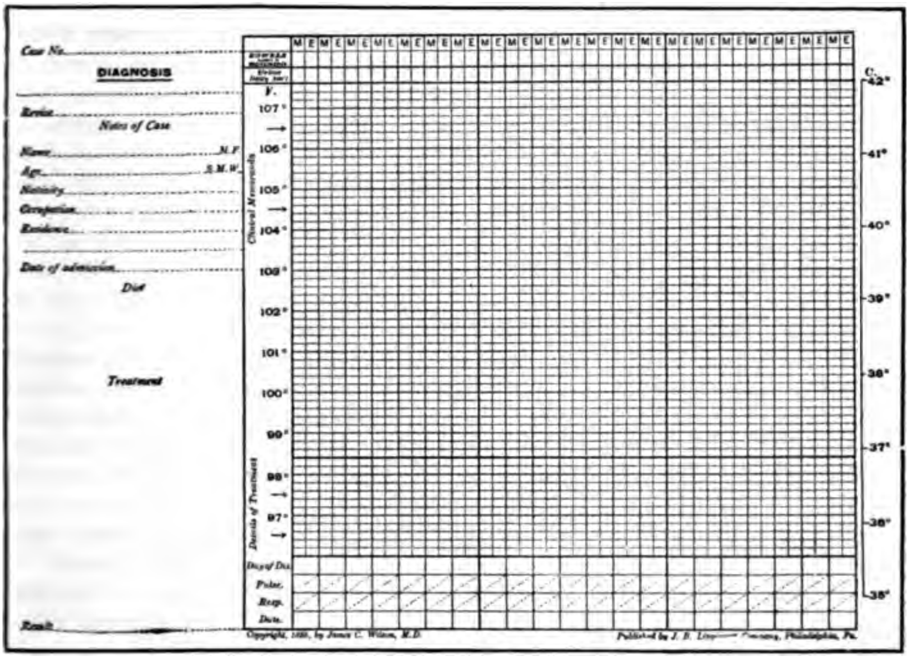


FIG. 29.—Clinical chart. Actual size 28 x 21 cm.

While changes in treatment, and in particular such temporary changes as are made necessary by accidents, like hemorrhage, convulsions, or suppression of urine, may be noted at the left or lower side under the words "details of treatment," as shown by the arrows.

The previous history and the condition of the patient when first seen may be written on the back of the chart.

The spaces corresponding to a degree of the *Fahrenheit* scale are divided into fifths. The temperature, as observed, is designated by a dot in the appropriate position. These dots joined by ruled lines form a zigzag line, called the temperature curve. It is usual to form the general curve of the case by means of the regular morning and evening temperatures, and to indicate the result of observations made at other hours by dots in the appropriate positions, with figures and letters showing the hour at which they were made; thus, 12 noon, 3 P.M., or 6 A.M.

It is customary to join the general curve or range by lines drawn with black ink; the hourly or three-hour observations by lines drawn with red ink. If the fever be prolonged beyond three weeks two or more charts may be pasted together. These charts thus kept are not only of value for preservation: they are also of immediate use as showing at a glance and with precision the facts of the case at every period from its coming under observation, the course it is running by a comparison of the symptoms day by day, and in a general way the effects of treatment, the changes of which are fully presented. Especially are they valuable in fevers in enabling us to watch the course of the temperature, which is a conspicuous part of the natural history of the disease and conforms in most of the acute infections to a type not only in its daily fluctuations but also in its duration.

II.

PHYSICAL DIAGNOSIS.

General Considerations.—Physical diagnosis is the method of discriminating diseases by the direct aid of the special senses, namely the eye, the ear, the touch. The diagnostic criteria thus obtained are known as physical signs. They depend upon the physical nature and structure of the organs or parts examined and vary with the changes caused by disease. Hence they are divided into two groups—normal or healthy, and abnormal or morbid physical signs. As they bear a direct relation to the anatomical condition of structures, their form, contour, density, elasticity, and so forth, and similar physical conditions may be present in different diseases, and as morbid processes may arise in the absence of perceptible alterations in parts, it is evident that physical signs taken singly are not diagnostic of particular diseases. They reveal the anatomical condition but not the morbid process causing that condition, and attain their full value in diagnosis only when considered in relation to other signs and symptoms and the clinical history of the case.

Pathognomonic signs are those supposed to be diagnostic of particular diseases. In view of the facts just mentioned, the use of the term pathognomonic in this sense is erroneous in theory and misleading in practice. A physical sign is the manifestation of a normal or morbid physical condition, not of health or disease. It is most important for the student to bear this fact clearly in mind. Signs at one time regarded as pathognomonic, as for example the crepitant râle in pneumonia, are now known to occur in other conditions, as œdema of the lungs and partial atelectasis.

Physical diagnosis is constantly employed in the study of general maladies and in local diseases of all parts of the body, but it is of special service in the investigation of diseases of the respiratory and circulatory organs.

Methods.—The methods of physical diagnosis are INSPECTION, PALPATION, MENSURATION, PERCUSSION,—including RESPIRATORY PERCUSSION, PALPATORY PERCUSSION, and AUSCULTATORY PERCUSSION,—and AUSCULTATION. In the examination of patients these methods are used systematically and in succession, the signs elicited by one serving to confirm, extend, or control the knowledge obtained by the others.

Technic.—The physical examination must under all circumstances be conducted in a routine manner. More errors in physical diagnosis arise from want of system than from want of knowledge. When the patient is in bed, the bared chest, abdomen, and back must be in turn examined by the several methods. Under some circumstances a towel or a single thickness of raiment may be used as a covering, especially in percussion and immediate auscultation. For inspection and palpation the surface should be bared.

The same rules apply to the examination of ambulatory patients. In all cases the outer clothing should be removed. Physical signs cannot be elicited through heavy clothing, starched linen, or the corset; while silk, stiff shirt fronts and the braces cause upon deep respiratory movements crackling and friction sounds that have nothing to do with the organs within the chest. In all cases the examination must be conducted with tact, judgment, and due regard for the sensibilities of the patient.

The recognition of abnormal physical signs involves a familiar knowledge of those which are normal and their variations within the bounds of health and of the anatomy and physiology of the organs or parts examined. Equally necessary is a ready knowledge of the pathological changes upon which abnormal signs depend. The skilled diagnostician cultivates the habit of seeing with his mind's eye the changes in structure caused by disease. A long apprenticeship in the post-mortem room is an essential preparatory course for good work at the side of the bed.

INSPECTION.

This method of physical diagnosis is of the widest application in the study of disease. In many cases a provisional, in some a positive diagnosis may be made upon a careful study of the external clinical phenomena by inspection alone. The facies hepatica, emaciated neck and limbs, and enormously distended abdomen in cirrhosis of the liver, the enlarged parotids, disfigured countenance, and projecting lobule of the ear in mumps, the unilateral flushing and jerky dyspnœa in croupous pneumonia, and the rash in the eruptive diseases, tell their own tale. In a narrower and more technical sense inspection is especially of value in the diagnosis of diseases of the thoracic and abdominal organs.

The clothing must be removed. The light must be good. The skilled diagnostician makes use now of direct light, by which extensive surfaces are fully illumined; now of oblique light, by which local elevations and depressions are accentuated and pulsations are marked by moving shadows.

By inspection we obtain information in regard to the size, form, or contour, the appearance of the surface, and the movements of the thorax and abdomen.

Inspection of the Thorax.

The Size.—The size of the chest is determined by the volume of its contents. Within the limits of health there are wide variations. A sedentary life tends to shallow breathing and small lungs. The chest then conforms to the inspiratory type. The anteroposterior diameter is short, the upper intercostal spaces wide, the lower narrow, the costal angle acute. We speak of such a chest as shallow. Active, out-door occupations favor habitual deep breathing and increase in the size of the lungs. The chest now conforms to the expiratory type. The anteroposterior diameter is relatively long, the upper intercostal spaces narrow, the lower wide; the costal angle is obtuse. Such persons are deep chested. Diseases which diminish the size of the lungs, as chronic tuberculosis and fibroid phthisis, correspondingly reduce the size of the thorax, while so-called pseudo-hypertrophic emphysema greatly increases its size. But these changes are accompanied by definite changes in form. Excessive subcutaneous fat sometimes gives rise to an apparent increase in the size of the chest.

The Form.—The form of the chest varies with its size. In infancy and early childhood it is somewhat cylindrical,—that is, its anteroposterior diameter and its transverse diameter are nearly the same,—and the respiration is chiefly diaphragmatic. In adults the cross section of the trunk is oval and symmetrical. Upon deep inspiration the anteroposterior diameter of the chest is increased; on forced expiration it is diminished.

Deformities.—The general deformities in childhood are commonly due to respiratory obstruction in the upper air-passages, as from adenoid growths in the nasopharynx, enlargement of the tonsils; or in the lungs, as in bronchopneumonia or phthisis. Rickets plays an important part. In adult life they are commonly caused by fibroid changes in the lungs, pulmonary tuberculosis and emphysema. Unilateral and local deformities are caused by pleural effusions, the retraction which follows the resorption or removal of such effusions, hypertrophy of the heart, and aneurismal or other intrathoracic tumors. These abnormal modifications in form are more marked when they occur early in life. The following deviations in form are to be considered:

(a) **The Alar or Pterygoid Chest.**—The chest is unnaturally small and narrow. The inner borders of the scapulæ project like budding wings, the ribs are extremely oblique, the shoulders droop, the neck and chest appear preternaturally elongated, the head is carried unduly forward, and the costal angle is acute. This form of chest is sometimes described as the "paralytic chest." Persons suffering from pulmonary tuberculosis frequently present this form of chest, but it may also occur in poorly nourished individuals who are not phthisical.

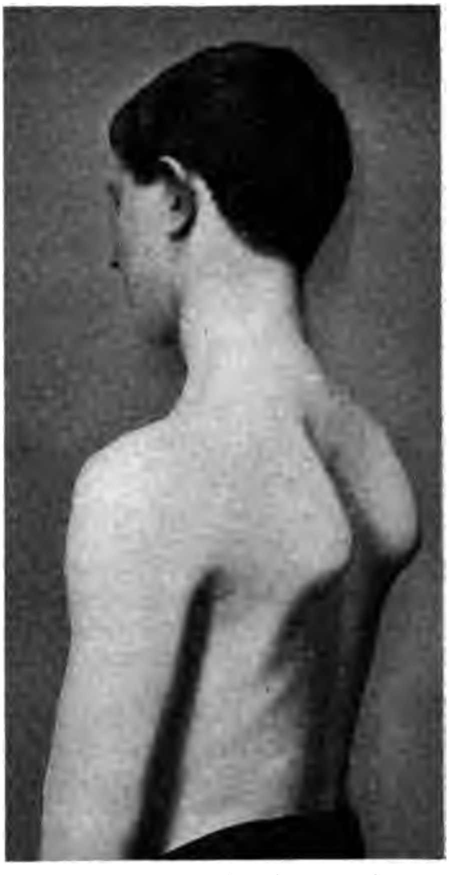


FIG. 30.—Alar deformity of chest.—German Hospital.

(b) **The Rhachitic Chest.**—The sternum may project, giving rise to the deformity known as *pigeon breast*. The sides of the chest are flattened and curve forward to the prominent sternum, as the sides of a boat to the keel—*pectus carinatum*. From the base of the ensiform cartilage a broad shallow depression or groove passes downward and outward to the infra-axillary region—HARRISON'S FURROW. In some instances the cartilages of the ribs lose their curve and become straight, causing the chest to be quite flat in front instead of being rounded. In others there is a shallow longitudinal groove on each side of the front of the chest, a little external to the sternum and nearly parallel to it. The remarkable deformity known as FUNNEL BREAST sometimes but by no means always is due to rickets. It consists in a deep and rather abrupt crater-like depression in the region of the base of the ensiform cartilage. Not rarely there may be felt and sometimes seen a line of nodular thickenings along the chondrocostal articulations on each side, known by the fanciful name of *the rhachitic rosary*.

(c) **The Barrel Chest.**—The deformity characteristic of emphysema is very striking. The antero-posterior diameter is greatly increased. The thorax is in a state of distention greater than that produced in health by the deepest inspiration. It is arched before and behind. The manubrium and body of the sternum are sometimes bent at an angle—ANGULUS LUDOVICI. The shoulders are high, the neck short, and the costal angle very obtuse. Dorsal kyphosis due to the carrying of burdens upon the shoulders, to advancing years, or to vertebral caries may simulate the barrel-shaped chest of emphysema.

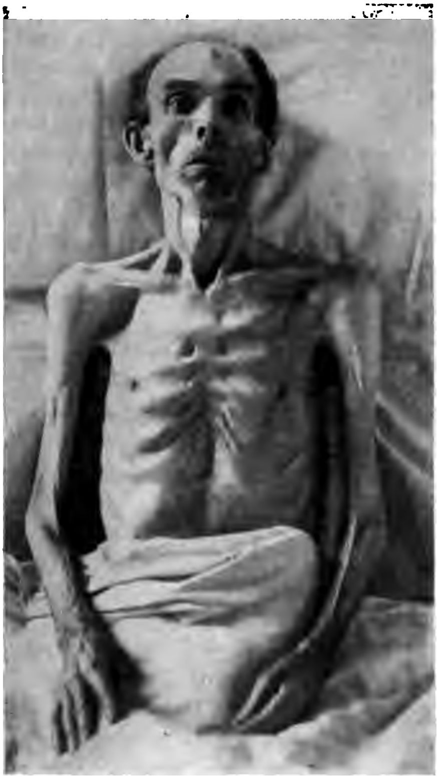


FIG. 31.—Paralytic chest.

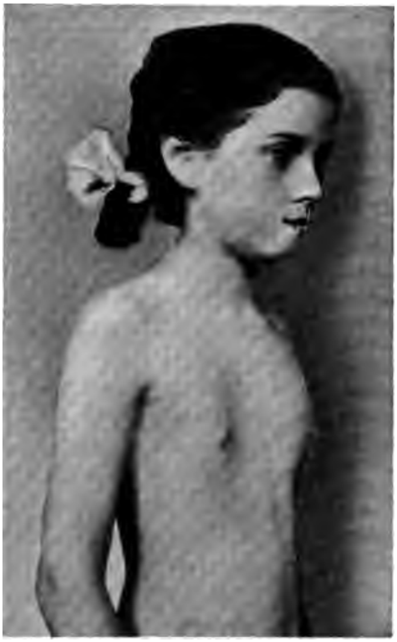


FIG. 32.—Funnel-shaped deformity of chest. —Jefferson Hospital.

(d) **Deformities of the Spine.**—Curvatures and twisting are very common. The slighter forms are often overlooked. They may be recognized upon careful inspection of the bare back, the spinous processes being marked by a dermatographic pencil. Marked curvatures in which rotary displacements are prominent derange the relations of the thoracic viscera to the bony landmarks and render the physical examination of the chest difficult and the signs uncertain. The cardiac impulse may be displaced upward or to the left; abnormal bulging may simulate aneurismal or other intrathoracic tumor and areas of atelectasis with compensatory emphysema occur. Abnormal rigidity of the spine may be due to spastic contraction of the muscles in Pott's disease or to spondylitis deformans. When ankylosis has developed the spine is persistently rigid. These signs may be recognized upon attempts to bend forward or backward or to rotate the shoulders while the pelvis is held fixed. An examination of the vertebral column forms part of every routine examination of the chest.

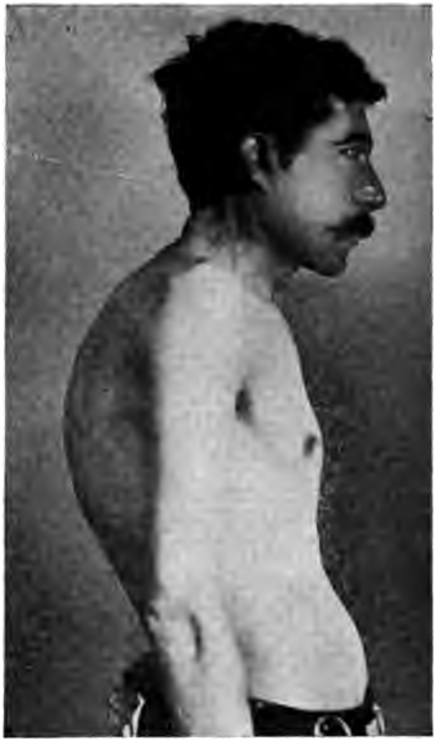


FIG. 33.—Emphysematous type of chest.—German Hospital.

Unilateral changes in the shape of the chest consist in diminution and enlargement.

Unilateral Diminution.—Flattening of one side of the chest is a sign of chronic pulmonary tuberculosis of the corresponding lung, fibrosis of one lung, or a pleural effusion which has undergone resorption or been cured by operation. The circumference and anteroposterior diameter are diminished; the bilateral diameter is increased; the side is angular and flattened before and behind; the upper intercostal spaces are widened, the lower narrowed; the shoulder is lowered and there is lateral curvature of the spine, the convexity being towards the opposite side. The vicarious enlargement of the sound lung gives rise to marked differences in the circumference of the two sides. When the deformity is due to tuberculous disease of the upper lobe, the flattening is more marked in the upper region of the chest; when to old pleurisy it is more marked at the base. Unilateral flattening of the chest is attended by pleural adhesions. If obstruction of the main bronchus occurs in childhood. the resulting collapse of the lung may cause an acute unilateral flattening of the chest. Lateral spinal curvature may simulate diminution of the chest from pulmonary disease.

Unilateral Enlargement.—This deformity of the chest is a sign of vicarious enlargement of one lung as a result of chronic disease of its fellow, pleural effusion, large hæmothorax, pneumothorax, and rarely of rapidly growing malignant disease. Pseudohypertrophic emphysema may in rare instances involve one lung when the other has undergone fibroid changes in consequence of previous disease. The enlarged side is rounder than the other; its anteroposterior diameter longer; the intercostal spaces wide;

the shoulder raised and the spine curved laterally, the dorsal convexity being towards the enlarged side.

The foregoing alterations in the form of the chest are very obvious when the physician stands behind the seated patient and looks obliquely over his shoulders and the front of his chest.

Intercostal Spaces.—In large pleural effusions and in pneumothorax the normal depression of the intercostal spaces is obliterated and the surface smooth as contrasted with the opposite side. Bulging of the intercostal spaces is rare. It may be seen at the base of the chest in large empyema of long standing.

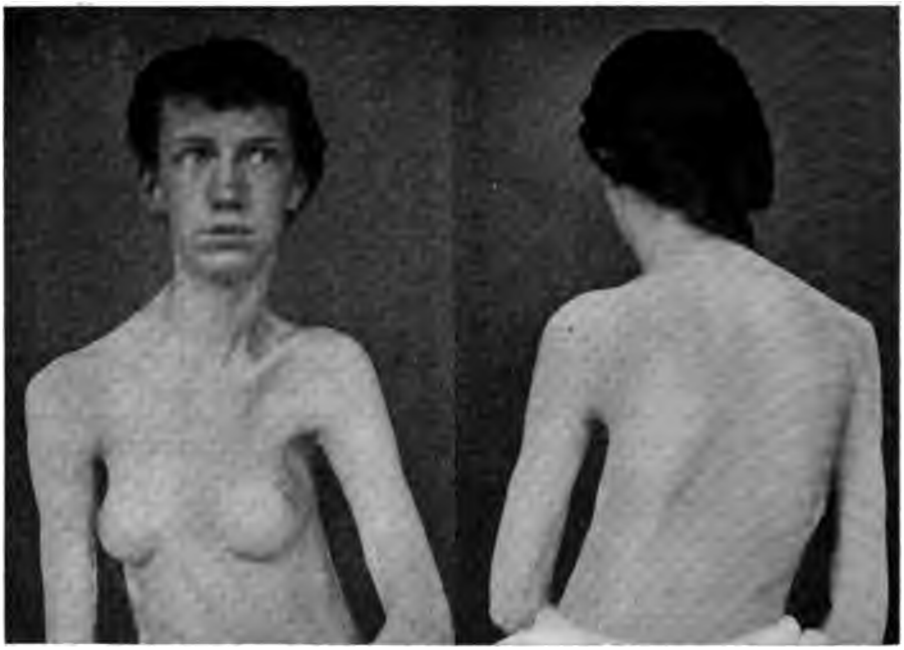


FIG. 34.—Deformity following the resorption of a pleural effusion.—German Hospital.

Local Changes.—Local changes in shape consist in (a) circumscribed retraction or (b) prominence.

LOCAL RETRACTION is a sign of the following conditions:

Tuberculous Consolidation of a Portion of the Lung.—This is usual at the apex and most obvious in the supra- and infraclavicular regions. It is attended by pleural adhesions.

A Superficial Cavity.—Circumscribed depressions due to this cause are often seen on the anterior surface of the chest near the sternal border and extending over one or two intercostal spaces. Flattening in the posterolateral aspect of the chest opposite the spine of the scapula and below its level is sometimes seen in pulmonary abscess.

Old Pleurisy.—A broad, shallow depression in the anterolateral region at the base of the chest is common after pleural effusions. The funnel breast sometimes follows unilateral pleurisy. This deformity in shoemakers has been attributed to the pressure of the last against the breastbone.

Local retraction in children may follow croup, bronchopneumonia, and ts. The deformities caused by these agencies are symmetrical and already been considered.

LOCAL PROMINENCE is a sign of circumscribed pleural effusion, large cæ when distended with fluid, diaphragmatic hernia when congenital, r of the lung or of the chest wall, mediastinal tumor, abscess of the wall, and empyema necessitatis. In a considerable proportion of hy persons that region of the chest wall which overlies the heart—*recordial space*—is slightly prominent. In children and occasionally ults prominence of the precordial space results from cardiac hyper- ιy or pericardial effusion. The bulging occupies the space between hird and seventh costal cartilages on the left side and the left mid- ular line and the sternum. It may extend to the right nipple. Aneu- of the arch of the aorta causes local bulging of the chest wall and in instances aneurism of the de- ling aorta may erode the ribs give rise to a circumscribed r of the dorsal region to the f the spine. Inspection of the frequently reveals local prom- es of importance in diagnosis. arp projection of the spinal sses occurs in vertebral caries. inner borders of the scapulæ l out prominently in the ptery- chest. A congenital rounded r in the middle line, translu- and partly reducible, is the of spina bifida. This defect of opment is frequently associated with other deformities, as hydro- ilus or club-foot. A dusky-red, brawny swelling, commonly in the cal region, discharging pus from several sinuses, is a carbuncle. It s frequently in diabetes mellitus, and in all cases the urine should be ined for sugar. Rounded or lobulated elastic tumors, painless and ly movable, are fatty. They sometimes so closely resemble abscesses require aspiration for the differential diagnosis. Abscesses appear ctuating swellings as the result of caries of the vertebræ, usually culous, and may burrow in various directions. I have seen a large, g tumor to the left of the dorsal spine formed by an aneurism of the nding aorta, and a similar tumor in the left lumbar region which pul- and was connected with a left-sided empyema. In disseminated ma of the skin the lesions are common on the back, appearing as mscribed nodular masses varying in size from a small shot to a walnut, in color and mostly movable.

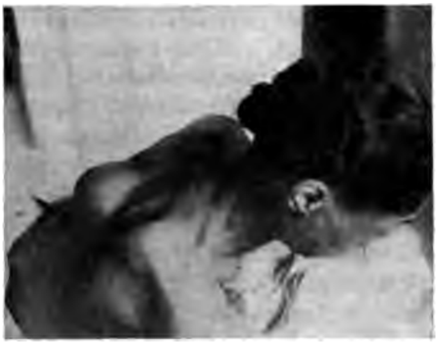


FIG. 35.—Aneurism of the descending thoracic aorta; perforation of chest wall.—Penna. Hospital.

Surface.—The appearance of the surface of the chest only excep- ly yields, upon inspection, physical signs of importance. We note ation or an abundant panniculus adiposus, jaundice, cyanosis and ntation. the eruptions and scars of the exanthemata or of syphilis her chronic disease, enlargement of the superficial lymph-nodes at the root of the neck and in the armpits, patches and lines of dilated venules and dilated and tortuous venous trunks. The appearance of linear patches of herpes in shingles—zona, herpes zoster—in the course of the intercostal and lumbar nerves, often clears up the diagnosis where there has been severe burning pain upon one side of the chest or abdomen.

The Movements of the Chest. — Normal and abnormal types of respiration will be considered in a subsequent section. Anomalous movements that affect both sides of the chest occur in dyspnœa, inspiratory dyspnœa, expiratory dyspnœa, Cheyne-Stokes respiration, exaggerated thoracic, exaggerated abdominal breathing, and so on.

Abnormally deep respiration in the absence of any apparent difficulty either in inspiration or expiration is seen in diabetic coma.

In the emphysema of the aged and in earlier life in some cases of hereditary syphilis and pulmonary tuberculosis, calcification of the costal cartilages and associated changes in the ribs cause the walls of the chest to move through a limited space as a whole—*en cuirasse*.

Unilateral modifications of the respiratory movements may consist of (a) diminished expansion of one side or (b) increased expansion of one side.

(a) DIMINISHED EXPANSION of one side may involve the entire side, as in large pleural effusion, pneumothorax, pneumonia involving the whole of one lung, tuberculous consolidation of a lung, or tumor of the lung or pleura. The affected side is not only immobile but it is also distended and altered in contour. In tuberculosis it is, however, usually contracted in consequence of pleural adhesions and sclerotic changes in the lung. In massive pneumonia it is almost immobile but not enlarged. Contraction also occurs in the occlusion of a large bronchus from the presence of an aneurism or other tumor.

In tuberculosis confined to the apex of one or both lungs there is failure of expansion in the corresponding region of the chest.

Diminished unilateral expansion may be a sign of infradiaphragmatic disease—on the right side, of an enlarged liver or hepatic tumor; on the left, of an enlarged spleen or tumor in the splenic region.

In rare instances a hemiplegia or paralysis of one side of the diaphragm or a diaphragmatic hernia may be the cause of diminished expansion of one side of the chest.

Non-expansive inspiration is attended with retraction of the interspaces. This sign is especially noticeable in the inframammary, the infra-axillary, and the infraclavicular regions in partial atelectasis or collapse of the lungs, in obstruction of the glottis as in pseudomembranous laryngitis, œdema of the glottis, or pseudomembranous bronchitis such as occurs in infralaryngeal diphtheria or in the diffuse atelectasis of bronchopneumonia. Under those conditions both sides are involved. When a main bronchus is occluded the sucking in of the intercostal spaces upon inspiration is limited to the affected side. This phenomenon is caused by intrathoracic negative pressure during inspiration, in consequence of which the soft parts of the thoracic wall yield to the external pressure of the atmosphere.

(b) INCREASED EXPANSION of one side of the chest is usually compensatory. It occurs when the respiratory movement of the opposite side is interfered with by pathological conditions of the lung, as tuberculosis, pneumonia, fibrosis and atelectasis from other causes, or by pleural effusion, pneumothorax, or tumor, and thus becomes a sign of those conditions.

The Diaphragm Phenomenon — Litten's Sign. — The diaphragm approaches the wall of the thorax in expiration and comes into contact with it at the end of the act. It is separated or *peeled off* from it in inspiration. These movements are rendered visible by the procedure suggested by Litten in 1892. The patient is placed upon his back with his chest bared and his feet toward a window. Cross lights are excluded. If the examination is made at night, a strong light held at the foot of the bed serves the purpose. The observer stands at a little distance and views the surface of the lower part of the chest obliquely. Upon deep inspiration a short, narrow, horizontal shadow is seen to move from the sixth intercostal space downward over two or more interspaces upon both sides. During expiration this shadow moves up again to the line from which it started but is less distinct. It may in some cases be seen in the epigastrium. This phenomenon is practically present in all healthy persons, the only exceptions being due to abnormal thickness of the chest walls and inability on the part of the patient to make full, deep respiratory movements. It is best observed in young, lean, muscular persons. The extent of the movement of the shadow in normal chests is about two and a half inches; upon forced breathing slightly more than this.

The descending shadow is due to the undulation of the chest wall caused by the separation of the diaphragm from its contact with the lower part of the thorax and the descent of the border of the lung into the wedge-shaped space between them during inspiration, and the reverse shadow by the retraction of the lung and the coming together of the diaphragm and chest wall during expiration.

The shadow is absent upon the affected side in pneumonia of the lower lobe, pleural effusion, extensive pleural adhesions, intrathoracic tumors, and marked emphysema. In these conditions the diaphragm does not approach and recede from the chest wall and the undulations which cause the shadow do not occur. The extent of the movement is lessened in conditions of debility, slight emphysema, and upon the affected side in phthisis. In the latter condition there are probably two factors in restricting the movement, diminished pulmonary expansion and limited pleural adhesions.

Litten's sign is present in hepatic and splenic enlargements and in subphrenic abscess and may be of service in the differential diagnosis between those conditions and pleural effusion. In very large ascites it may be absent.

The Movements of the Heart. — Inspection yields important physical signs in regard to the heart and great vessels in health and disease. These signs relate to (a) the cardiac impulse; (b) other movements of the surface having the cardiac rhythm: (1) pulsations at the root of the neck, (2) aneurism, (3) tumors in contact with large arterial trunks, (4) pulsating empyema.

(a) **The Cardiac Impulse.** — With the systole of the heart there is seen in most normal chests an outward movement or pulsation in a limited area in the fifth left intercostal space just beyond the parasternal line—the visible impulse or so-called apex-beat of the heart. In infants and young children, owing to the proportionately greater size of the liver, the impulse is often visible as high as the fourth interspace, while in aged persons it may normally be as low as the sixth interspace. It is occasionally absent in healthy persons, especially those having deep chests and capacious lungs. It invariably takes place at the time of the contraction of the ventricles. The most important factor in the production of the impulse is the change in the direction of the long axis of the ventricles against the resistance of the chest wall. It is a mistake to speak of it as a "blow" or "impact" against the wall of the chest, since that part of the heart which causes it, namely, the apex of the right ventricle, is already in contact with the wall in diastole and simply becomes more tense and prominent during systole. Around the point where the soft parts are protruded by the impulse they are very slightly retracted at the time of its occurrence—the "*negative impulse.*" This is due to the lessening size of the contracting ventricles, which, being air-tight within the cavity of the chest, must be followed down under the pressure of the atmosphere by the elastic and yielding lungs and the somewhat yielding intercostal tissues. A clear conception of this fact renders intelligible the systolic recession of the chest wall occasionally seen in emaciated persons in the third, fourth, or even the fifth intercostal space, close to the left border of the sternum.

Since the normal impulse is caused by the apex of the right ventricle and not by that of the left, which extends further downward and is separated from the wall of the chest by a tongue-like projection of the lower lobe of the left lung, the apex of the right ventricle is sometimes spoken of as the "clinical apex" and that of the left ventricle as the "anatomical apex" of the heart.

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The impulse becomes forcible and extended when the normal heart is acting rapidly and with force under physical or mental stress and in thin, nervous persons, and it is often extended in young children even at rest.

The character of the impulse and its extent are best studied by palpation, but inspection alone enables us in many cases to determine that the impulse is extended, heaving, tapping, or undulatory.

DISPLACEMENTS OF THE IMPULSE OF THE HEART.—*Displacements due to Changes in the Heart Itself.*—The impulse is displaced downward and toward the left in hypertrophy and dilatation of the heart, and the combination of these conditions is the most common cause. Enlargement of the left ventricle tends to displace the visible impulse downward, enlargement of the right ventricle tends to displace the impulse to the left, and both of these conditions tend to increase its extent.

Pressure Displacements are next in Order of Frequency.—The heart is dislocated upward in pressure from below the diaphragm, as in excessive tympany, ascites, massive tumors, large cysts, and pregnancy. In any of these conditions the impulse may be seen in the fourth interspace and to the left of the midclavicular line. The heart in pleural effusion, pneumothorax, or rapidly growing malignant tumors of the pleura, is displaced towards the opposite side. When these conditions are left-sided the impulse may disappear behind the sternum or become visible at its right border, or in extreme cases in the right nipple line. In large right-sided effusions, on the contrary, the impulse may be displaced as far left as the line of the anterior axillary fold. Cysts and abscess in the right lobe of the liver may displace the heart somewhat to the left and shift the impulse to a corresponding extent.

The heart may be displaced downward by an aneurism of the arch of the aorta or a mediastinal tumor. Under those circumstances the impulse is not only lower than normal but it is also somewhat further to the left.

Traction displacements of the heart occur in pulmonary cirrhosis and long-standing disease of the pleura. The displacement is toward the affected side. Pleuropericardial adhesions and negative pressure constitute the mechanical factors by which this group of displacements is brought about. A cardiac impulse may be seen to the right of the sternum, or to the left of the left midclavicular line, or if there be great retraction of the upper lobe on either side there may be visible cardiac pulsation at the corresponding border of the manubrium. Spinal curvatures and rotations may produce such displacement of the heart as to cause a wholly abnormal position of the visible cardiac impulse or its absence altogether. Dextrocardia may be the cause of a right-sided impulse, an anomaly also present in complete transposition of the viscera.

SYSTOLIC RETRACTION.—I have already spoken of the negative impulse present under normal conditions in the immediate proximity of the apex-beat and the more extended systolic recession of the interspaces occasionally seen along the lower sternal border. These are distinctly accentuated when a hypertrophied and somewhat dilated heart is acting forcibly. As these forms of systolic recession are due to atmospheric pressure, they may be spoken of as *pulsion recessions* in contradistinction to those due to the drawing in of the surface in consequence of adhesions, which may be called *traction recessions.*

The latter are seen in adherent pericardium with chronic mediastinitis. The impulse is undulatory and in the region of the apex there is marked systolic retraction. Owing to the enlargement of the heart the precordial region is prominent and the chest asymmetrical. The impulse is greatly extended.

BROADBENT'S SIGN.—When the heart is extensively adherent to the diaphragm, there occurs with each pulsation a systolic tug. This may be communicated through the diaphragm to the points of its insertion in the wall of the chest and well seen in the eighth and ninth intercostal spaces in the root of the neck and in the armpits, patches and lines of dilated venules and dilated and tortuous venous trunks. The appearance of linear patches of herpes in shingles—zona, herpes zoster—in the course of the intercostal and lumbar nerves, often clears up the diagnosis where there has been severe burning pain upon one side of the chest or abdomen.

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Systolic Retraction. — I have already spoken of the negative impulse present under normal conditions in the immediate proximity of the apex-beat and the more extended systolic recession of the interspaces occasionally seen along the lower sternal border. These are distinctly accentuated when a hypertrophied and somewhat dilated heart is acting forcibly. As these forms of systolic recession are due to atmospheric pressure, they may be spoken of as *pulsion recessions* in contradistinction to those due to the drawing in of the surface in consequence of adhesions, which may be called *traction recessions.*

The latter are seen in adherent pericardium with chronic mediastinitis. The impulse is undulatory and in the region of the apex there is marked systolic retraction. Owing to the enlargement of the heart the precordial region is prominent and the chest asymmetrical. The impulse is greatly extended.

Broadbent's Sign.—When the heart is extensively adherent to the diaphragm, there occurs with each pulsation a systolic tug. This may be communicated through the diaphragm to the points of its insertion in the wall of the chest and well seen in the eighth and ninth intercostal spaces in the parasternal line; but Broadbent has pointed out the fact that it is often also seen on the left side behind, between the eleventh and twelfth ribs. Careful inspection in this region will frequently reveal a systolic retraction of the chest wall, which becomes more evident upon deep inspiration.

VISIBLE PULSATIONS OF THE HEART IN REGIONS OTHER THAN THE APEX.—These are mostly due to retraction of the lungs. In debilitated and bed-ridden persons and especially in the graver forms of anæmia, the breathing is shallow and the lungs are not fully expanded. Their borders are therefore more or less withdrawn from the space which they normally occupy between the heart and the chest wall. The pulsations of the conus arteriosus and right ventricle thus frequently become visible in the second, third, and fourth left interspaces near the sternal border. In some instances these pulsations may also be observed to the right of the sternum. Such pulsations are also seen when the borders of the lungs are retracted as the result of fibroid phthisis.

(b) **Other Movements of the Surface of the Chest having the Cardiac Rhythm.**

1. PULSATIONS AT THE ROOT OF THE NECK will be described and their significance as physical signs pointed out in a subsequent section. They are venous and arterial.

Prominence of the veins of the neck is observed in emaciated and elderly persons otherwise in health. These veins are more or less distended upon expiration, particularly when cough occurs or dyspnœa is present. Transient engorgement results from efforts at lifting or from straining. Pathological conditions that give rise to engorgement of the jugulars are aneurism, mediastinal tumor, adhesive mediastinitis, and obstruction to the pulmonary circulation from any cause. Respiratory engorgement and collapse of the jugulars are especially marked in the dyspnœa of asthma and emphysema.

Collapse of the jugular upon one side, not disappearing when pressure is made upon it immediately above the clavicle, is a sign of thrombosis of the lateral sinus.

Pulsating Jugulars. — The pulsations are best studied on the right side of the neck and during quiet breathing. Pulsation communicated from the underlying carotid may be recognized by emptying the vein by stripping it upward gently with the finger-nail or the blunt edge of the tongue spatula. It does not refill from below.

The visible pulsations in the carotids often seen in thin, nervous persons without disease of the heart are without clinical importance. Violent throbbing of the carotids is common in aortic regurgitation and frequently occurs in simple hypertrophy of the heart without valvular lesions.

2. ANEURISM.—Careful inspection of the anterior surface of the chest must be made in all cases of suspected aneurism. Direct and oblique illumination must be in turn employed, and the examination must be so conducted that profile views are made from above, the patient being in the sitting posture, and from the side, the patient being recumbent. In this way slight pulsations and pulsating prominences may be discovered. The pulsation of aortic aneurism is commonly present in the first and second

interspaces near the sternal border and is sometimes accompanied ght systolic elevation of the inner end of the clavicle. When the iinate is involved the pulsation may be seen at the root of the neck the right side or at the notch of the sternum. Aneurismal pulsations imes occur to the left of the manubrium sterni and elsewhere in the and are to be sought for in every doubtful case. Aneurisms that perforated the chest wall appear as circumscribed globular or irregular :ing tumors, the overlying skin being thinned and adherent and ately ulcerated, so that there is ficial clot formation and more s continuous oozing of blood. umor may be soft and fluctu- g; more commonly in conse- 'e of the deposition of stratified layers within the sac it is dense 'esistant. In the former case ulsation is expansile; while in itter case it is apt to be non- isile, but forcible and heaving.

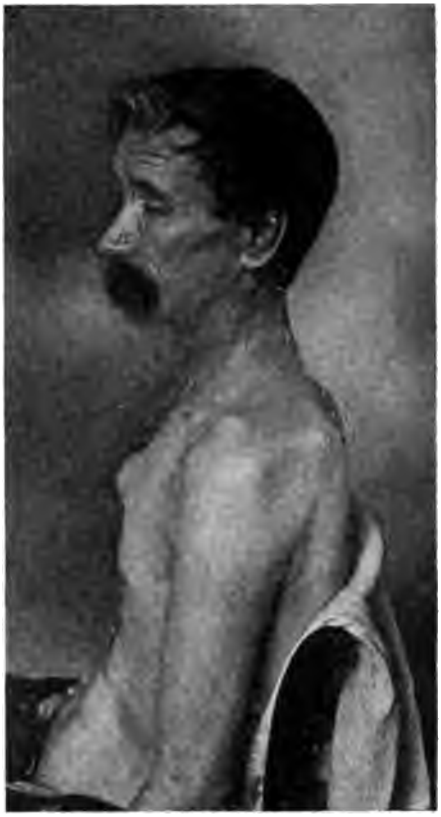

FIG. 36.—Aneurism of the thoracic aorta.—German Hospital.

:. **Tumors in Contact with** E **Arterial Trunks.**—En- l lymph-nodes, especially single, and neoplasms in the overlying the carotid artery times move synchronously the pulsations of the vessel and nt superficial resemblances to risms. The tumor is dense,)ulsation not expansile, and signs of aneurism are lacking.

l. **Pulsating Pleurisy.**—In cted purulent effusions a pul- g movement synchronous with :ardiac rhythm is sometimes ved. The cases are not numer- The phenomenon is almost 's associated with left-sided ons and occupies an extensive of the lower anterolateral sur- of the left chest. In cases where it is circumscribed and confined ie precordial region the differential diagnosis relates to aneurism is attended with difficulty. A limited number of cases have been sided and in one or two of the reported instances the effusion has serofibrinous. Pulsating empyemata may be intrapleural or the tions may occur in the extrapleural *empyema necessitatis.* None of xplanations of the mechanism by which the cardiac impulse in these is transmitted through the pus collection to the surface of the chest isfactory. An important factor is superficial ulceration of the costal a with loss of tone in the intercostal muscles.

Inspection of the Abdomen.

In the examination of this portion of the body the patient should be in the recumbent position and preferably in bed. The abdomen in exposed from the arch of the ribs to the suprapubic region. The patient should lie straight and flat. The head should be at first low and the lower limbs extended; later the head should be raised upon pillows and the thighs and knees strongly flexed, the heels being drawn up towards the buttocks, in order to relax the abdominal wall; finally it is often necessary to have the patient assume the standing posture, in which case the clothing or a sheet is supported about the hips by the patient or an assistant. The light must be good and the examination made from above, from the sides and obliquely. Physical signs of importance, such as asymmetry in contour or movement or slight local elevation or depression of the surface, may often be detected when otherwise not very obvious, if the observer stands at the patient's head and views the abdomen obliquely from above downwards. Combined inspection, palpation, and percussion are necessary. Auscultation is of inferior value in the examination of this region.

(a) **The Normal Abdomen.** — In infants and young children the abdomen is relatively larger as compared with the size of the chest than in adults. It is also more protuberant than in well-formed adults. It is larger in women than in men and is enlarged and protuberant in obese and elderly persons. In thin women who have borne many children it is relaxed, coarsely wrinkled, and pendulous. Tight corsets cause bulging of its lower segment. Transient prominence of the upper segment may sometimes be observed after a hearty meal.

The size of the abdomen in health varies greatly in different individuals according to the amount of subcutaneous and omental fat and the size of the intestines, which are apt to be distended in persons who habitually eat large quantities of coarse food. The physiological enlargement of the abdomen in pregnancy is frequently enormous.

The normal abdomen is symmetrical in contour, slightly arched from above downward and from side to side, the curves being more prominent, especially in the lower part, in the erect than in the recumbent posture. The navel is shallow and marked by irregularly spiral folds of skin in thin persons and deep and funnel-shaped in those who are fat.

The skin of the abdomen in healthy persons is opaque and the superficial veins are not conspicuous. In brunettes regularly distributed areas of increased normal pigmentation are present in the median line and above the flexures of the thighs. This coloration is deepened and conspicuous in pregnancy—*chloasma uterinum.* The respiratory movements of the diaphragm are communicated to the upper portion of the abdomen, the ensiform cartilage and the arch of the ribs being elevated and becoming more prominent with inspiration. In persons with very thin and relaxed abdominal walls the peristaltic movements of the stomach and intestines may be occasionally seen.

(b) **Inspection of the Abdomen in Disease.** — We study the size of the belly as manifest in general or local retraction or distention, alteration in form and contour, the appearance of the surface and abnormal movements. In this connection the general rule that the size of a hollow anatomical structure or viscus varies with the contents must be borne in mind.

General Retraction of the Abdomen. — When the longitudinal and transverse curves of the surface are reversed and become concave instead of convex, the abdomen is described as scaphoid or boat-shaped. Two factors may cause this condition and they are frequently combined, namely, extreme wasting and irritative tonic spasm of the abdominal walls. The former occurs in actual starvation; inanition from any cause, especially malignant disease of the larynx or œsophagus, stricture of the latter from other causes, stricture of the pylorus without marked gastric dilatation, diabetes, phthisis, cerebrospinal fever, cholera, chronic diarrhœa, anorexia nervosa, and the pernicious vomiting of pregnancy; the latter, in meningitis, cerebral tumor, and lead colic, and, especially when combined with muscular rigidity and marked tenderness, is a most important sign of early peritonitis.

Extreme retraction of the abdomen occurs in wasting of the subcutaneous and omental fat and atrophy of the abdominal organs.

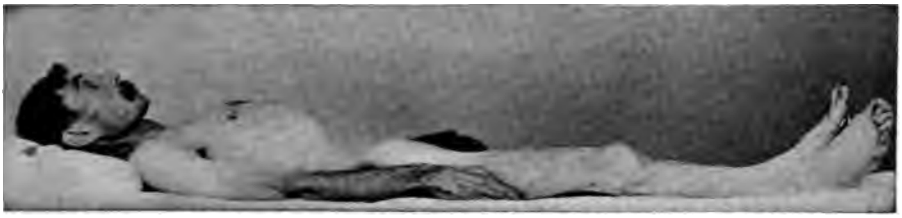

FIG. 37.—Scaphoid abdomen caused by starvation in a case of œsophageal carcinoma.—Jefferson Hospital.

Local retractions of the abdomen are not of importance as physical signs. They are seen around the base of large hernias, especially in the lateral regions of the abdomen, in large ventral hernias, and in the upper regions in diaphragmatic hernias. These areas of depression disappear when the hernias which cause them are reduced.

In moderately large peritoneal effusions of some standing, when the patient assumes the lateral decubitus the side of the abdomen which is uppermost shows a concave retraction while the anterior and dependent portions bulge more prominently.

General Distention of the Abdomen.—This condition may be caused by subcutaneous and intra-abdominal fat, the excessive accumulation of gas in the stomach or intestines, fluid in the abdominal wall or peritoneal cavity or both combined, or a large intra-abdominal tumor.

Subcutaneous and intra-abdominal fat accumulations in the obese frequently cause enormous distention of the belly. In such cases there is excessive and often irregular development of the panniculus adiposus elsewhere; while in ascites and tumor the general nutrition is usually impaired. In cases where there is reason to suspect pregnancy or the presence of an abdominal tumor a large deposit of fat may render the diagnosis difficult. Large accumulations of fat in the omentum, such as sometimes occur in persons of middle age, may simulate pregnancy or a tumor. Fat in the belly walls interferes greatly with the examination by means of the X-rays.

The Excessive Accumulation of Gas — Meteorism, Tympanites. — The distention is symmetrical and may be extreme. There is tympanitic percussion resonance and absence of fluctuation. The association of these physical signs renders the diagnosis easy. When extreme the condition causes restriction of respiratory movement, the disappearance of the respiratory excursus in the epigastric zone, and displacement of the cardiac impulse upward as high as the fourth interspace and to the left of its normal position.

Moderate distention may result from injudicious eating, acute and chronic gastro-intestinal disorders. especially in neurotic persons, and the slight paresis of the intestines which occurs in acute febrile diseases, as enteric fever or pneumonia. Nervous women are apt to "bloat," as it is popularly called. after eating. Excessive tympany occurs in grave cases of the infectious diseases, as enteric fever with deep ulceration, septic conditions, acute general peritonitis, intestinal obstruction, after the release of a constricted loop of intestine after operation, as in strangulated hernia, and in some cases of hysteria.

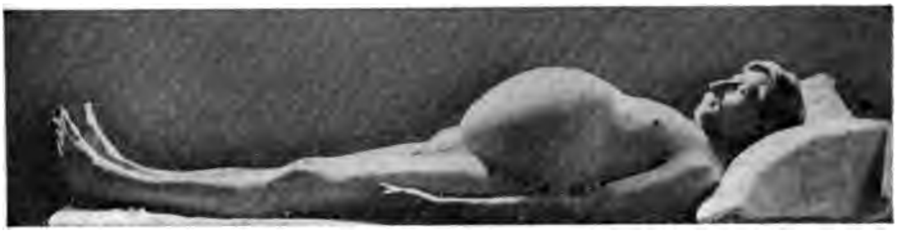

FIG. 38.—Ascites, caused by cirrhosis of the liver.—**Jefferson Hospital.**

Free gas in the peritoneal cavity occurs as the result of the perforation of an air-containing viscus into that space. The abdomen is greatly and uniformly distended, its surface tense and smooth, the outlines of intestinal convolutions and vermicular movements are not visible, and the respiratory movement of the upper part of the abdomen ceases. The most common causal conditions are peptic ulcer of the stomach or duodenum, a perforating typhoid ulcer, and ulcerative or necrotic appendicitis. As the air occupies the highest region of the cavity, it causes a disappearance of the normal percussion dulness of the liver and spleen, which is replaced by a tympanitic note in these areas, of the same character as that over the abdomen elsewhere. The mere disappearance of the hepatic dulness does not, however, justify the diagnosis of pneumoperitoneum, since the intestines and especially the transverse colon may occupy the space between the liver and the wall of the thorax and separate them completely. Moreover, the liver dulness may be greatly diminished in pulmonary emphysema of high grade, atrophic cirrhosis of the liver and acute yellow atrophy. The diagnosis of free air in the peritoneum may, however, be determined by careful percussion in the axillary line according to the following procedure: In the dorsal posture there is dulness alike in the condition under consideration and when the liver is separated from the wall of the thorax anteriorly by the distended intestine. When, however, the patient is turned upon his left side there always remains a limited area of dulness in the axillary line high up in the case of meteorism, while the dulness wholly disappears in the case of pneumoperitoneum. The same method of examination is applicable to the spleen, although the small size of this organ renders its recognition alike in large meteorism and in pneumoperitoneum a matter of much greater difficulty than in the case of the liver.

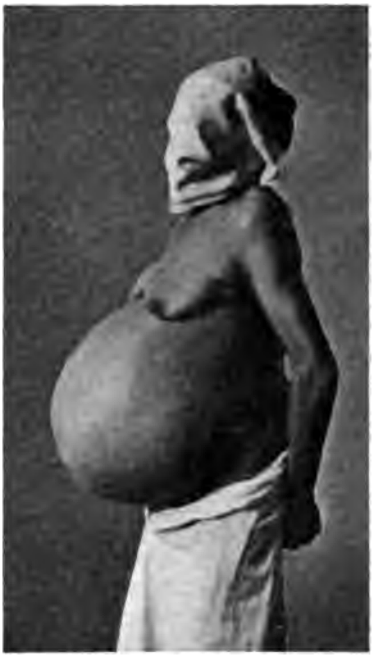

FIG. 39.—Ascites.—German Hospital.

Fluid in the Abdominal Wall or Peritoneal Cavity.—An excessive dropsy of the wall in some cases of anasarca may cause distention of the abdomen. This condition is encountered in acute nephritis and in the later stages of cardiovascular disease. The abdomen is tense, doughy, and pits upon pressure; the more dependent parts of the body, feet, ankles, legs, thighs, and pudenda, are highly œdematous, and the condition is usually associated with effusion into the peritoneum and sometimes also into the other serous cavities. The pallid and puffy facies in acute nephritis is characteristic.

Ascites or free fluid in the cavity of the peritoneum yields characteristic physical signs. The enlargement of the abdomen is general and symmetrical. Its degree and outline depend upon the amount of the fluid and the fact that under the influence of gravity it changes its position with changes in the posture of the patient. In moderate effusions, in the dorsal decubitus the middle of the abdomen is more or less flattened while the lateral regions bulge outward, in the lateral decubitus the lower lateral and anterior walls of the belly protrude while that which is uppermost is slightly incurved, in the knee-elbow posture the weight of the fluid causes the abdomen to sag down in an unusual manner, and in the erect posture the lower segment of the abdomen is especially prominent. In all these positions there is dulness upon percussion over the dependent areas and tympanitic resonance

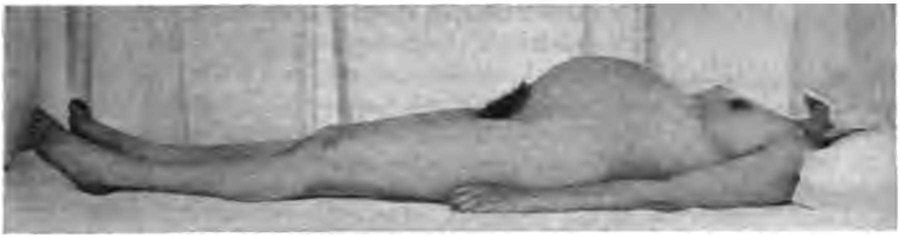

FIG. 40.—Pregnancy—ninth month.

over the upper, since the fluid gravitates toward the dependent regions of the cavity and the air-containing intestines float upon it in the upper spaces. Ascites in a belly previously relaxed or pendulous causes in the erect posture a prominent and somewhat conical symmetrical protrusion of the lower parts. Massive ascites gives rise to uniform symmetrical enlargement of the abdomen, but little influenced by change of posture.

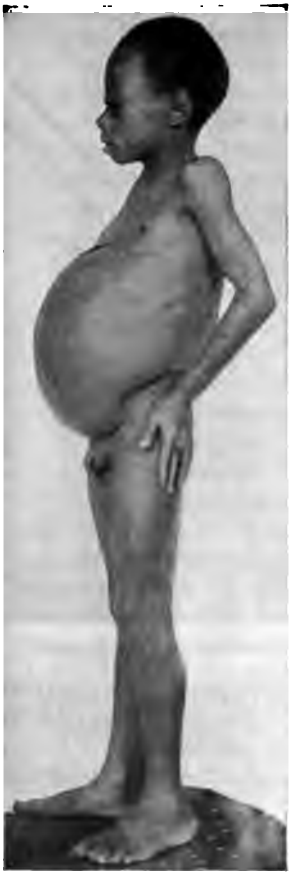

FIG. 41.—Dilatation of colon. Male, 12 years old.—Rotch.

Ascites results from pathological processes directly implicating the peritoneum, as ordinary infections or tuberculous inflammation or cancer, or the portal vessels, as the pressure of new growths, gall-stones, cancerous invasion, extreme sclerosis or pylephlebitis due to other causes, or disease of the liver. Cirrhosis of the liver is a common cause of ascites. Tumors of the abdomen and especially large solid tumors of the ovary are frequently attended by ascites. The foregoing have been spoken of as local causes. The general causes of ascites are those which give rise to anasarca and effusion into the other serous sacs. Peritoneal effusion resulting from local causes is not usually at first associated with œdema of the lower extremities. As the fluid accumulates it exerts pressure upon the large abdominal veins, especially the iliacs and ascending vena cava, giving rise to dropsy.

Tumor as a Cause of General Abdominal Enlargement.—The pregnant uterus, ovarian, pancreatic, and hydatid cysts, and large new growths cause distention which may simulate that due to the causes just considered. The enlargement caused by these conditions differs from that caused by fat, tympany, or fluid in being usually more prominent in the anteroposterior than in the bilateral diameter, not so symmetrical, and not yielding uniform signs upon palpation and percussion. Other causes of general enlargement of the abdomen are fecal accumulation, cancer of the bowel, disseminated cancer of the peritoneum, and large peritoneal or retroperitoneal sarcomata and lipomata. To this list must be added hydronephrosis and enormous dilatation of the stomach or colon.

Local Prominence of the Abdomen.—Circumscribed swellings or tumefaction may be caused by abnormal conditions of the belly wall or of the contents of the cavity. These changes in contour should be carefully sought for in all cases presenting symptoms referable to the abdominal viscera. The methods of especial value are inspection, palpation, and percussion. In thin persons radioscopy yields important results.

The recognition of the nature of local bulgings in the abdominal wall is as a rule not attended by great difficulty, but the diagnosis of visceral mors is frequently obscure and in many cases can only be positively etermined by an exploratory operation.

Local Prominences due to Changes in the Wall of the Abomen.—These comprise abnormal conditions of the muscles, irregular llections of subcutaneous fat, hernia, abscess, enlarged lymph-glands, nd neoplasms, particularly sarcomata.

A spasmodically contracted rectus muscle may simulate a tumor. The iagnostician must be on his guard against the appearance and sensation nparted to the touch by a contracted right rectus in the pyloric region.

Phantom Tumor.—The condition known as phantom tumor, due to ersistent gaseous distention of a knuckle of gut with spasmodic contracion of the overlying muscle, causes a tumor-like swelling. Such swellings ppear and disappear, with alterations in contour and position; sometimes ubside under gentle friction with the warmed hand and always under næsthesia. They occur in hysterical persons. Fitz has suggested that n some of the cases phantom tumors are symptomatic of congenital or cquired dilatation of the colon.

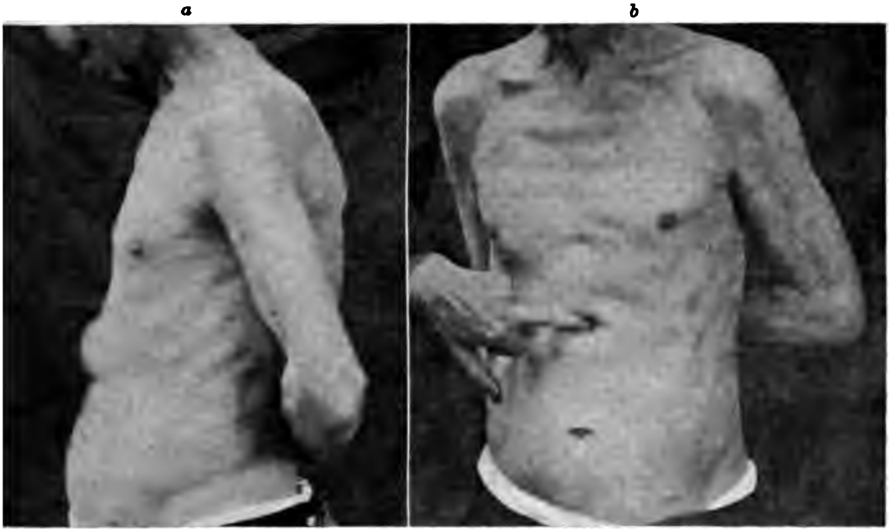

Fig. 42.—*a*, paraumbilical hernia; *b*, hernia reduced.

Fat. — In very obese persons remarkable rolls and masses of subutaneous fat collect in the abdominal wall. These are usually but not lways symmetrical in arrangement, and may simulate tumors, from which hey may be differentiated by their continuity with the panniculus adiosus, their consistency and want of tenderness, and the general condition f the patient. Circumscribed fatty tumors—lipomata—are common. They are hemispherical or egg-shaped, elastic, painless, somewhat movble, and more common in the lateral and posterior aspects of the trunk han in the abdominal wall. They frequently occur in spare persons.

Hernia.—No examination of the abdomen is complete that does not [illegible] sites of hernia. This is especially important in cases attended by intestinal obstruction and vomiting, or persistent pain in the inguinal region. The inguinal and femoral regions should be examined by palpation under the cover of the sheet or clothing and if necessary by inspection as well. Ventral and umbilical hernias and scar-hernias after operation may be readily recognized. The tumor varies in consistency according as it consists wholly of gut or partly of omentum. It is usually soft, without pain upon manipulation, and reducible. It varies in size from a mere nodule to a sac containing a large portion of the abdominal contents. It very often disappears spontaneously when the patient assumes the recumbent posture, or is then readily reduced. Strangulated hernia does not, as a rule, yield to taxis.

Abscess.—Purulent collections in the abdominal wall may be recognized by the signs of inflammation, swelling, redness, heat, and pain, by their contour, and especially by fluctuation. Pus may form in any part of the wall or find its way to any point upon the surface. Appendiceal abscess usually forms a circumscribed, fluctuating tumor in the right lower quadrant of the abdomen.

Lymph-nodes.—The superficial lymphatic glands of the groin do not form visible tumors unless distinctly enlarged. They are frequently palpable as small nodular bodies in adults who are in good health. They may become enlarged and tender in injuries of the leg or foot, in venereal disease, and in common with the superficial lymph nodules in other parts of the body in some of the acute infectious diseases and in particular in the bubonic plague. Slight enlargement of the inguinal lymphatics is common in generalized malignant diseases—carcinomatosis, sarcomatosis. Massive enlargement of these structures takes place in Hodgkin's disease. The enlarged inguinal glands in venereal disease and the plague frequently form suppurating buboes.

Neoplasms of various kinds may develop in the abdominal wall. The most common variety is sarcoma. In sarcomatosis cutis many small subcutaneous nodules appear scattered over the abdomen. In a recent case a sarcoma developed at the umbilicus and was followed, after operation, in about a year by a small nodule in the immediate neighborhood and many others in different parts of the body.

LOCAL PROMINENCES DUE TO ABNORMAL CONDITIONS WITHIN THE ABDOMINAL CAVITY.—These conditions comprise:

Temporary Dilatation of the Stomach from Excesses at Table,
Gastrectasis,
Local Gaseous Distention of the Bowel,
Fecal Accumulations,
Ectopic or Floating Viscera,
Visceral Hypertrophies and Enlargements,
Intra- and Perivisceral Abscess,
Abscess from Caries of the Spine,
Cysts,
Extra-uterine Pregnancy,
Abdominal Aneurism,
Glandular Enlargements, and
Malignant and other New Growths.

Any of these can exist without being the occasion of prominence cognizable upon inspection; but under favorable conditions this method physical examination yields suggestive—even positive—physical signs. is usually a question of the degree of their development respectively.

Excessive fat and muscular rigidity mask the signs of these conditions, d in the case of great abdominal tenderness a satisfactory examination impossible. The plain recognition of an abdominal tumor does not in l instances justify a further diagnosis of its cause or nature.

Gastrectasis.—Temporary dilatation of the stomach from excesses in ting causes in persons who are not obese a visible prominence in the igastric region. Substantive gastrectasis from any cause shows a bulg- g of the abdominal wall in the region of the umbilicus r above it. This bulging has downwards and to the ft the outline of the greater curvature; if the stomach, s is very commonly the case, is displaced downward nd its longitudinal axis more vertical than normal, e outline of the lesser curvature may also be visible elow the ensiform cartilage.

Intestinal Obstruction.—The entire abdomen may be istended or only parts of it. If the colon be distended y hard fecal masses the course of the bowel is marked y an elongated eminence, the contours of which cor- spond to those of the gut. If the intestinal stenosis e acute a local area of gaseous distention without peri- alsis occurs above the obstruction. The obstruction ay be caused by fecal accumulations, large gall-stones, nteroliths, or the pressure of a tumor. Any of these ay give rise to a distinct, localized, asymmetrical rominence of the abdominal wall.

Intussusception is most common in childhood and hows itself as an elongated sausage-shaped tumor usu- lly in the region of the cæcum or at the sigmoid flexure.

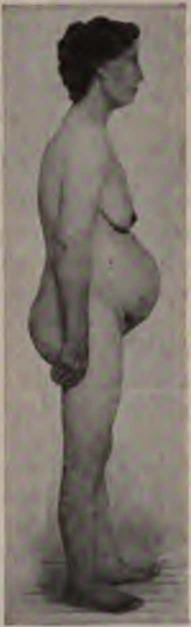

FIG. 43.—Visceroptosis.—Pennsylvania Hospital.

Ectopic or Floating Viscera.—General splanchnop- sis—Glénard's disease—causes a prominence or pro- rusion of the lower segment of the abdomen and is ommon in women, being favored by tight corsets, the ethod of supporting the skirts, and the relaxation due to childbearing. nteroptosis causes a similar deformity more or less marked; gastroptosis usually associated with dilatation of the stomach. A vertical position f the stomach may be congenital or acquired as the result of tight lacing. he pylorus then occupies a position in the median line or to the left of , and the greater curvature lies below the level of the umbilicus. In thin ersons these displacements of the organ may sometimes be demonstrated y the methods of physical diagnosis, especially if it be inflated with gas llowed by the introduction of water through the tube so that the greater urvature may be determined by dulness on percussion in sharp contrast ith the tympanitic resonance of the colon.

Floating kidney sometimes gives rise to an oval prominence plainly isible upon inspection, which may be made to shift its position or disappear upon manipulation or upon changes in the posture of the patient. The swelling caused by a displaced kidney is usually upon its own side, but when very movable it may sometimes be forced beyond the median line to the opposite side. It may occupy a position anywhere between the ribs and the pelvis and is freely movable with deep respiration. *Ren mobilis* is much more common in women and upon the right side.

Floating Spleen.—The normal spleen which, in consequence of elongation of the gastrosplenic ligament and the splenic artery and veins, has become dislocated—*lien mobilis*—does not cause a visible abdominal swelling. When, however, the displaced organ is also enlarged, as is frequently the case, there may sometimes be seen a rounded swelling upon the left side in any position from the hypochondrium to the pelvis. This swelling, like that caused by the dislocated left kidney, with which floating spleen is often associated, is freely movable upon manipulation and change of posture.

Floating liver is among the rarest of clinical or anatomical findings. The dislocation of the organ is usually slight. There is general enlargement of the right lateral region and a large mass of characteristic outline which descends when the patient assumes the erect posture. Tympany in the upper part of the right hypochondrium—normal area of liver dulness—disappearing when the organ is replaced, and the well-defined lower border of the liver upon palpation, render the diagnosis a matter of comparative ease.

Enlargement of the Gall-bladder. — This condition may properly be considered at this point, since the position of the enlarged bladder is very different from that of the normal gall-bladder. The enlargement is the result of cholecystitis, frequently associated with cholelithiasis, or carcinoma.

The gall-bladder is distended by a serous fluid which gradually accumulates in consequence of the inflammatory changes in its walls,—dropsy of the gall-bladder,—the bile no longer entering, because of obstruction of the cystic duct by a calculus, a plug of tenacious mucus, adhesive cholangitis, or a carcinomatous nodule. In comparatively rare instances infection by pyogenic organisms causes suppurative cholecystitis—empyema of the gall-bladder. If the gall-bladder be sufficiently distended and the abdominal wall thin, there may be seen an elongated, smooth prominence in the region of the notch of the liver, projecting below the liver margin and rising and falling with the respiratory movements. The gall-bladder may be greatly distended, reaching in some instances the size of the fist or more. It is then sometimes pear-shaped, the fundus being freely movable from side to side upon manipulation and change of posture.

In some instances when the cholecystitis is associated with cholelithiasis the gall-bladder is distended by an enormous accumulation of calculi; in others the tumor may be due to primary or secondary carcinoma of the gall-bladder.

Visceral Enlargements.—The so-called corset liver may give rise to a visible prominence in the right lateral region, reaching as low as the crest of the ilium and moving with respiration. In cases in which the pressure constriction is marked the portion of the liver below it is movable and may simulate, especially when a loop of intestine occupies the groove, a displaced kidney or new growth in the ascending colon.

Enlargement of the liver, causing marked prominence in the right hypochondrium, in some cases of the entire abdomen, may be due to hypertrophic cirrhosis, carcinoma, amyloid disease, conditions causing obstructive jaundice, leukæmia, syphilis, hyperæmia due to cardiac disease, and fatty liver.

Enlargement of the spleen may attain a considerable degree before it gives rise to signs upon inspection. Massive enlargement may occur in chronic malaria—ague cake, leukæmia and pseudoleukæmia. The organ may reach to the pelvis and even to the right of the median line.

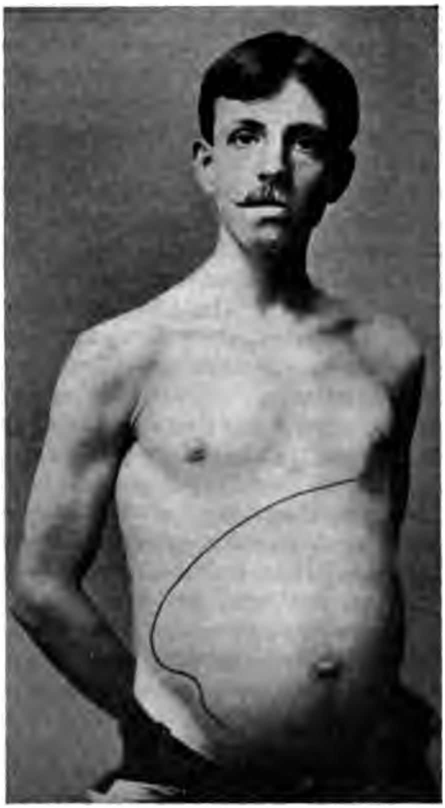

FIG. 44.—Massive enlargement of spleen in a case of splenomedullary leukæmia.—Jefferson Hospital.

Enlargement of the Kidneys.—Renal tumors develop from behind forward, tending to displace the movable organs of the abdomen, especially the intestines, aside.

The anatomical relations of the ascending and descending colon are such that these portions of the intestines, being attached to the kidneys by connective tissue, are retained in front of the growing renal tumor and tend to obscure its dulness upon percussion. The development of the tumor from the upper portion of the kidney causes a prominence of the hypochondrium on the corresponding side, which extends as the growth develops to the iliac region; the development of the tumor from the lower portion of the kidney causes prominence first in the iliac region. Two solid new growths of the kidney only are of clinical importance from the stand-point of diagnosis, namely, carcinoma and sarcoma. The former is more common in advanced life, the latter in childhood; the former is apt to cause early cachexia, while in the latter the general nutrition may be maintained; finally, sarcomatosis of the skin in connection with tumor of the kidney is highly suggestive as to the nature of the renal affection. Renal adenoma cannot be differentiated from carcinoma during the life of the patient. Much more rare is hypernephroma.

In the rare cases in which both kidneys are involved the abdominal enlargement is of course bilateral.

The very rare primary malignant disease of the suprarenal capsules may give rise to a tumor in the hypochondrium of the corresponding side, which differs in no respect from the similar manifestation caused by a tumor of the upper half of the kidney. Renal tumors move only slightly or not at all with respiration.

Enlargement of the pancreas is caused by chronic pancreatitis and carcinoma. It very rarely reaches such a size as to occasion visible prominence in the epigastrium.

Abscess.—Local bulgings of the surface may be caused by suppurative inflammation in and around the abdominal viscera.

Abscess of the Liver.—Multiple abscess does not usually reveal itself by changes in the contour of the surface. Tropical abscess commonly causes the liver to enlarge upward, especially upon the right side. The respiratory excursus is diminished or absent and the lower intercostal spaces obliterated. There is often local œdema.

Subphrenic abscess occasions marked downward displacement of the liver and a smooth, soft tumor in the epigastrium. If, as is commonly the case, there is air as well as pus in the subphrenic space, the diagnosis is not attended with difficulty.

Abscess of the spleen, when of sufficient size, sometimes reveals itself by a splenic tumor, upon the surface of which a fluctuating area or areas may be obscurely felt through a thin-walled abdomen.

Renal abscess may cause a circumscribed tumor in the hypochondrium or iliac region of the affected side, with obscure fluctuation, but without œdematous swelling of the neighboring tissues.

Perinephritic Abscess.—The swelling occupies the lumbar region and there is œdema of overlying and adjacent parts. There is frequently burrowing of the pus in a downward direction, so that a second fluctuating tumor may be present at a more dependent point.

Abscess in Appendicitis.—The common situation of the large, circumscribed intraperitoneal abscess is in the iliac region between the navel and the anterior superior spine. The abscess may form in the retroperitoneal space and burrow beneath the iliac fascia, showing itself at Poupart's ligament, or it may accumulate in the retroperitoneal tissue in the flank, forming a large paranephritic abscess, with the usual œdematous condition of the surrounding parts.

Abscess from caries may follow enteric fever and show itself as a small fluctuating tumor overlying a rib or costal cartilage. Vertebral caries may cause an abscess in the lumbar region, or the pus may follow the sheath of the psoas muscle and point below Poupart's ligament—psoas abscess.

Ovarian or tubal abscess may give rise to distention in the iliac region of either side. When upon the right side these conditions may simulate appendiceal abscess, with which they are also occasionally associated.

Cysts.—Local as well as general prominence may be caused by cysts of various kinds. If large the distention is general, if small it is local and circumscribed. From a pathologico-anatomical beginning wholly without symptoms and unrecognizable, certain cysts frequently attain enormous dimensions. Among these are especially to be mentioned cysts of the pancreas, hydronephrosis, and ovarian cysts, which are often of such size as to simulate ascites. The smaller cysts do not present physical signs which differentiate them from abscesses in the same localities. It is only by a general knowledge of the pathological processes which give rise to cyst- and abscess-formation respectively and a careful consideration of the anamnesis and associated symptoms that the differential diagnosis can in some instances be made out, as in dropsy and empyema of the gall-bladder, echinococcus and abscess of the spleen, or hydro- and pyonephrosis. Cysts springing from the liver, dropsy of the gall-bladder, echinococcus and pancreatic cysts have their early manifestations in the upper regions of the abdomen—epigastric zone—to the right and left of the median line respectively; those springing from the kidney—hydronephrosis, echinococcus—first appear in the lateral regions, while those from the pelvic organs, ovarian cysts, hydramnion, arise from the pelvis—hypogastrium. Mesenteric cysts are usually situated to the right of the umbilicus and below its level. Cysts connected with the liver and spleen are influenced by the respiratory movements; those connected with the pancreas only slightly or not at all, and those developing from the kidneys, ureters, and pelvic organs remain wholly unaffected by the respiratory movements of the diaphragm.

Aneurism.—Aneurism of the abdominal aorta may cause a distinct, pulsating tumor commonly in the epigastrium but occasionally to the left of the median line in front or in the lumbar region. This tumor is almost always immovable, but in rare instances has been influenced by manipulation and change of posture, but not by respiration. It presents the signs of aneurism, and is to be differentiated from tumors overlying the aorta and from the so-called "dynamic pulsation" of the aorta which occurs in neurotic individuals. The distended urinary bladder in urethral stricture, impacted calculus, etc., gives rise to a distinct rounded oval tumor of the hypogastrium, which reaches in extreme cases well up towards the umbilicus. To a less extent the retention of the low fevers and comatose conditions gives rise to a similar prominence. In the latter case, the incontinence of retention—*stillicidium urinæ*—prevents extreme distention. The anamnesis, the oval outline of the tumor, its central and symmetrical situation, fluctuation, and its immediate disappearance upon catheterization render the diagnosis clear.

Extra-uterine Pregnancy. — There is a history of morning nausea, paroxysmal colicky pain with faintness, enlargement and hardness of the breasts, and chloasma uterinum, together with the presence of a prominence to the right or left of the median line above the brim of the pelvis. Very often rupture of the sac takes place before it has attained sufficient enlargement to be recognized by the methods of physical diagnosis. This accident is attended by collapse symptoms, and upon vaginal examination the uterus is found to be somewhat enlarged and displaced downward and to the opposite side.

Glandular Enlargements.—Enlargement of the retroperitoneal glands, usually sarcomatous—Lobstein's cancer—may cause a visible tumor in the epigastric or umbilical region, usually tense, immovable, and nodular; sometimes slightly movable and obscurely fluctuating and crossed by the colon, which may be recognized upon palpation or by its tympanitic resonance, to secure which artificial inflation may be necessary. Tuberculous mesenteric glands—tabes mesenterica—cause, especially in children, marked protrusion of the abdomen with tympany. The enlarged lymphatic glands may cause irregular local prominence in the region of the navel or in the right iliac fossa and may be recognized upon palpation.

Malignant and Other New Growths.—Malignant diseases of abdominal organs—carcinoma, sarcoma—are of chief, while benign affections, fibroma, lipoma, myxoma, adenoma, and gumma, are of subordinate interest from the stand-point of diagnosis. This difference is to be ascribed not only to the greater frequency of the former and their disastrous effects upon the health and ultimately upon the life of the patient, but also to the fact that at some time in their course the diagnosis becomes both practicable and obvious, while in the latter with less significant symptoms the diagnosis cannot be made out and the condition often remains unsuspected during the whole course of the patient's life and only assumes pathologico-anatomical interest when the case, death having resulted from an entirely different disease, at length comes to autopsy. It is of diagnostic importance that in visceral as well as in external cancer, secondary implication of adjacent and distant organs takes place with characteristic signs and that ultimately in many cases the superficial lymphatic glands become enlarged, nodules appear in the skin and elsewhere—general carcinomatosis, general sarcomatosis.

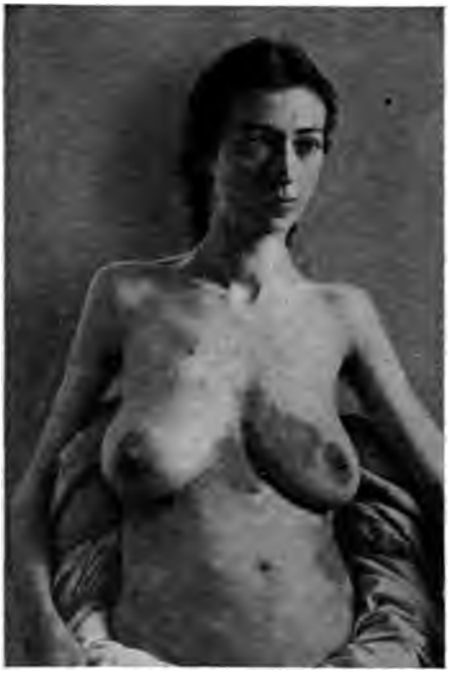

FIG. 45.—Sarcomatosis cutis,—primary tumor springing from a pigmented mole on the forehead; metastatic growths appeared in a few months after primary growth.—Jefferson Hospital.

Cancer of the Stomach.—The tumor can be seen in some cases, but is usually only to be recognized upon palpation. It most commonly occupies the region of the pylorus and may be slightly movable with respiration and freely so upon manipulation. A visible tumor occupying the greater part of the epigastrium and even extending beyond its borders, irregular, nodular, well defined at its margin, immovable and very distinct through the emaciated wall of the belly, is sometimes present in advanced cases of carcinoma extensively involving the anterior wall of the stomach.

Cancer of the Liver.—The volume of the organ is usually greatly increased. The increase is rapid and may assume enormous dimensions. It may affect the entire liver or the right or left lobe to a preponderating extent. When the right lobe is chiefly involved, there is a flaring out of the lower ribs and costal cartilages; when the left, the appearance of the tumor may suggest a new growth involving the greater curvature of the stomach, a cyst of the pancreas, or an enlarged spleen, but these doubts are immediately set at rest by palpation and percussion. The surface is usually uneven and the border irregular, and these signs may in some cases be clearly made out upon inspection. In the absence of adhesions the respiratory excursus of the liver may be seen, and in one remarkable case in my service at the Philadelphia Hospital, when upon autopsy the entire right lung was found to be solidified by secondary carcinomatous infiltration, the respiratory movement of the liver, plainly seen through the abdominal wall, was from left to right upon inspiration. When, as is often the case, extensive adhesions are present, respiratory movement of the enlarged liver does not take place Multilocular echinococcus and gumma of the liver present great difficulties in diagnosis. Splenic enlargement in the former and a history of lues in the latter are significant. An individual who has syphilis may also be the subject of echinococcus disease.

Cancer of the Gall-bladder.—The position of the tumor and its respiratory movement are important. It is apt to be mistaken for cancer of the pylorus or duodenum. In the latter affections, when the cancer is primary, free hydrochloric acid may be wanting in the gastric contents, secondary dilatation of the stomach shortly appears, and the tumor may be made out to be connected with the stomach or bowel by dilating the stomach, with simultaneous percussion and palpation, while the seat of the tumor in the gall-bladder becomes at the same time more obvious by its shape and relatively superficial situation.

A tumor formed by cancer of the head of the pancreas cannot often be positively differentiated from cancer of the pylorus, duodenum, transverse colon, or porta hepatis. At best in a majority of cases the diagnosis must be made by exclusion.

Inspection of the Surface of the Abdomen. Abnormal Signs.—Moderate ascites and large tumors may be present without changes in the integument; but excessive distention causes nutritive changes and the skin loses its natural appearance, becoming tense, glistening, and thinned. White lines or striæ—*lineæ albicantes*—irregularly parallel and slightly depressed below the adjacent surface are produced by extreme or prolonged distention, as in pregnancy, obesity, and ascites. They are seen upon the abdomen, flanks, and thighs, and persist after the condition which caused them has passed away. Jaundice is often more conspicuous here than on surfaces exposed to the air. Striking deposits of pigment occur in the linea alba in pregnancy, especially in brunettes, and pigmentation due to abdominal growths and diseases of the peritoneum, Addison's disease, melanotic cancer, exophthalmic goitre, scleroderma, arteriosclerosis, and chronic heart disease is often conspicuous upon the abdomen, especially in the lower quadrants and about the flexures of the thighs. The pigmentation of vagabondage due to lice and filth is usually characterized by the parallel linear superficial lesions of scratching. The hæmochromatosis of hypertrophic cirrhosis and diabetes and in rare instances scleroderma are attended by conspicuous pigmentation. The prolonged administration of arsenic frequently causes marked discoloration of the skin. The general discoloration of argyria is less pronounced upon the surface of the trunk than upon the face and extremities. The specific eruptions of the exanthemata, especially the initial rashes of variola, and the rose spots of enteric fever are to be sought for upon the abdomen. Tache bleuâtres, tinea versicolor, and the symmetrical diffuse macular eruption of secondary syphilis are to be seen. The scars of surgical operations, especially those performed for the relief of appendicitis, gastric and gall-bladder disease, and various diseases of the pelvic organs are common nowadays and may shed light upon many abdominal disorders—adhesions and the like—post-operative neurasthenia and other obscure maladies. Enlarged inguinal glands and retracted cicatrices in the groins may be significant of venereal infection.

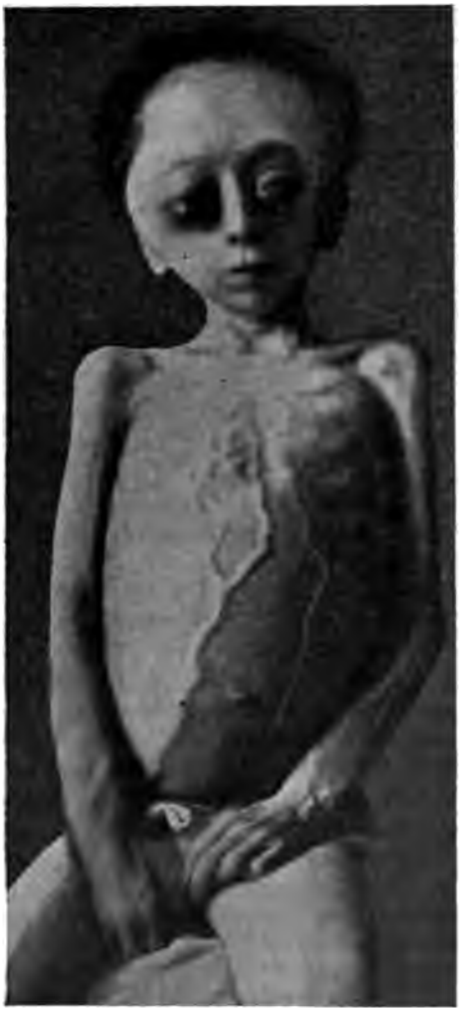

Fig. 46.—Sarcoma of spine, showing venous stasis and metastatic growth in orbit.—Young.

Vascular Changes.—Signs relating to circulatory derangements are enlarged superficial epigastric arteries and enlarged superficial veins. The former are exceedingly rare and indicate obstruction of the aorta or iliac arteries; the latter very common and constitute the evidence of collateral venous circulation in obstruction of the portal system or the inferior or superior vena cava. Among the common causes of such obstruction are, in the portal circulation, cirrhosis of the liver and tumor; in the general circulation, abdominal and mediastinal tumor, dilatation of the stomach of high grade, and ascites of long standing.

Caput medusæ is a varicose arrangement of the dermal veins around the umbilicus with radiating branches. It is made up by the dilated branches of the epigastric veins at their juncture with a large single vein which passes from the hilum of the liver and follows the course of the round ligament—para-umbilical vein of Sappey. Much more commonly the enlarged collateral veins are distributed irregularly over the surface of the abdomen and indicate one of the courses towards the right heart taken by the blood in pressure upon the inferior vena cava. There is engorgement of the blood from the lower extremities in the inferior epigastric and internal mammary veins, with dilatation of the superficial abdominal veins. In obstruction of the portal system and inferior vena cava the course of the blood in the dilated superficial veins is upward; when the superior cava is obstructed the course of the blood in the superficial veins of the chest and abdomen is downward, the blood seeking its way to the right heart by means of the right azygos which communicates with various tributaries of the inferior vena cava. Pressure upon the innominate vein of the right or left side may give rise among other signs to great dilatation of the superficial veins of the thorax and abdominal wall:

The Umbilicus. — The navel normally shows transverse or slightly spiral folds of the skin and is moderately retracted. It is deeply so and funnel-shaped in obese persons and level with the surrounding surface or protruding in large ascites and pregnancy. It may be the seat of caput medusæ or hernia, inflammation or eczema, carcinoma secondary to gastric carcinoma or tuberculous infiltration secondary to tuberculous peritonitis A mole in the region of the umbilicus may undergo sarcomatous changes.

Movements of the Abdomen in Disease. — Inspiratory retraction of the epigastrium is present in stenosis of the upper air-passages and imperfect action of the diaphragm. Diminished respiratory movement of the abdomen may be caused by upward pressure upon the diaphragm, as in tympany, ascites, and abdominal tumors on the one hand, or by massive pleural or pericardial effusions on the other. In the early stages of peritonitis abdominal respiratory movement is greatly impaired or wholly absent, on account of the pain and tonic contraction of the muscles of the wall; in the later stages on account of the tympany and upward pressure upon the diaphragm.

Visible Peristalsis.—In thin persons the normal peristaltic movements may sometimes be seen. They appear as wave-like, rounded elevations of the surface which may be attended by borborygmi and may be intensified by gentle irritation of the skin by the application of cold, brisk tapping or faradism. In some instances the peristaltic movements of the stomach from left to right are in sharp contrast to those of the transverse colon from right to left. In the wide separation of the recti occasionally seen in women who have borne many children these vermicular movements are very conspicuous.

The most important diagnostic significance of visible peristalsis relates to intestinal obstruction. The presence of peristalsis must be determined and whether or not it is always in the same direction and ceases at a certain spot. If the obstruction is at or above the ileocæcal valve the distended and mobile coils of small intestine occupy a position in the central portion of the abdomen, but if the obstruction involves the lower part of the large intestine—sigmoid flexure—the distention and movements of the bowel may be manifest in the region occupied by the ascending and transverse colon. The inflated fixed intestinal coil of acute stenosis of the gut, ileus—strangulated hernia—shows no peristaltic movement. Prior to immobility there is peristalsis. In chronic obstruction, after the muscularis of the gut has become hypertrophied, there is active peristalsis, with marked recurrent tumor subsiding with coarse borborygmi, just in advance of the stenosis. As the gas in the tumor is under tension, it does not yield tympanitic resonance but dulness upon percussion. Visible peristalsis in the left hypochondrium, with the vermicular contractions from left to right, has been observed in extreme gastrectasis.

Pulsation Synchronous with the Cardiac Systole.—Dynamic pulsation occurs in neurotic persons. It is seen in the median line and is often violent but neither diffuse nor expansile. The pulsation of abdominal aneurism usually has both these characters and very often in addition systolic thrill and bruit. It is mostly situated in the median line, but may be seen in the left lateral region of the abdomen.

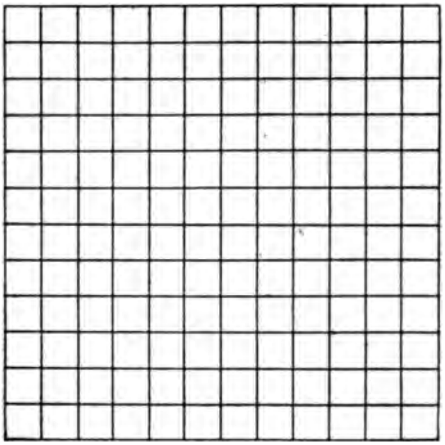

Fig. 47.—Beall's aid to inspection.

An Aid to Inspection in Circumscribed Movements not Well Defined.—I have found the following suggestion of K. H. Beall of much service:

"Over the area under inspection there is drawn with a skin pencil a square plaid figure, the squares of which are from 1.5 to 2.5 cm. in diameter and from 12 to 50 in number, according to the size of the area being studied. Any slight movement of the skin at any point in such a marked area causes a change in the direction of some of the lines and a distortion of the figure, and so renders visible movements of the internal organs which are not to be detected otherwise."

PALPATION.

The method of physical diagnosis in which the sense of touch is employed is known as palpation. It consists in the systematic examination of the surface of the chest and abdomen by the laying on of the hand. The physical signs elicited depend upon the condition and movements of the parts and the underlying structures. As in inspection, we study the form, size, condition of the surface, and movements. The method is applicable and essential to the examination of the thorax and abdomen.

Palpation in the Examination of the Thorax.

The chest should be bared, the attitude easy, the arms symmetrically disposed, the muscles relaxed. The examining hand should be warm and laid gently upon the surface. The amount of pressure employed must be determined in individual cases. Ticklishness, tenderness, and excessive fat constitute obstacles. The first may be overcome by care and diverting the attention of the patient; the others often amount to insuperable difficulties in the application of this method of diagnosis. The palmar surface of the whole hand is employed for a general survey, as in locating the position of the cardiac impulse or a thrill; the more sensitive finger tips for the study of the particular characters of such phenomena, for example the force and extent of the impulse or the coarseness or fineness and extent of a thrill.

By palpation we confirm and amplify the signs obtained by inspection, especially those dependent upon the form and contour of the chest, the width of the interspaces, the presence of local swellings and deformities, and the respiratory and cardiac movements. These it is not necessary at this point to repeat. But there are other physical signs, not always recognizable upon inspection, which we investigate by palpation. These comprise the condition of the wall of the chest as regards

Muscular Tension,
Œdema,
Width of the Interspaces,
Fluctuation,
Nodes, Gummata, and Periosteal Thickening,
Location and Character of the Heart's Impulse.
Extracardial Pulsation and Diastolic Shock, and in particular the following physical signs which are exclusively within the scope of this method:
The Crepitation of Subcutaneous Emphysema,
Thrills, Cardiac and Vascular,
Fremitus, Vocal, Friction, and Rhonchal,
Tracheal Tugging.

Tension.—The tension of the muscular wall of the chest in the intercostal spaces and about the ensiform cartilage is not a sign of great value, yet it is to be studied in doubtful cases. The inspiratory retraction of the base of the chest is a sign of obstruction to the entrance of air, which may be at the larynx, as in œdema of the glottis, or in the smallest bronchial tubes, as in bronchopneumonia. The slight normal furrow of the lower intercostal spaces may be obliterated by pleural or pericardial effusion or a rapidly growing new growth. In old empyemata there is great relaxation and bulging and the cardiac pulsations may even be transmitted to the surface—pulsating empyema. Epigastric rigidity and tenderness are conspicuous in tetanus, and these phenomena are early symptoms in peritonitis beginning in the upper part of the abdomen.

Œdema.—Local œdema may indicate intrathoracic suppuration as in empyema or hepatic abscess, inflammation of the wall of the chest as in carbuncle, or obstruction to the venous circulation as in mediastinal tumor or aneurism. The puffiness involves the head and neck on both sides when the pressure involves the precava and is unilateral when it affects the right or left innominate only.

Spaces.—The width of the intercostal spaces may be felt when not seen upon inspection, and should be carefully investigated in cases of pleural effusion, since they are wide when the chest is distended and become narrow as the fluid undergoes resorption.

Fluctuation.—Elasticity or fluctuation in any prominence or tumor upon the surface of the chest is an important sign. It may be due to abscess of the wall itself, empyema necessitatis, cyst formation, or sarcoma. The differential diagnosis rests upon the associated clinical phenomena. In abscess of the wall the volume of the tumor is not affected by the respiratory movements; in empyema necessitatis the tumor diminishes upon inspiration and increases with expiration and the physical signs of intrapleural effusion are present; a cyst is usually sharply circumscribed, distinctly globular, tense, sometimes translucent, and commonly movable within a limited range.

Nodes.—Nodes upon the ribs, cartilages, or sternum or thickening at the chondrocostal or sternoclavicular articulations and periosteal thickening are important signs of disease. They may sometimes be felt when not obvious upon inspection, and their size and consistence can be recognized upon palpation. Among the earliest of the skeletal lesions of rickets is a nodular enlargement of the ribs at the juncture of the bone with the cartilage. These nodules are present upon the ribs of both sides and are symmetrical in their arrangement—the so-called rosary of rickets. Gummata are common upon the sternum and roughening and enlargement of the clavicles may be a manifestation of late syphilis. The clavicles are enlarged and the sternum deformed in acromegaly. Acute painful enlargement of the sternoclavicular articulation is not rare in gonorrhœal arthritis. A soft, elastic, slightly fluctuating tumor upon the upper part of the sternum may be a tuberculous abscess. Tender points are found upon palpation. They are not physical signs, but may be mentioned in this connection as symptoms of great value. They are found in intercostal neuralgia and correspond to the points of emergence of the intercostal nerves; in neurasthenia tender points are also found along the dorsal spine and the tenderness is very often present upon light and absent upon firm pressure; in necrosis of a rib; in fibrinous pleurisy and especially in that form of pleurisy which occurs in pulmonary tuberculosis, where the tenderness is most common and most marked in the infraclavicular region.

Apex-beat.—The precise location and character of the impulse of the heart. The palm of the hand should be first laid over the precordia below the left nipple. The signs elicited by inspection are thus confirmed and amplified. We determine whether the rhythm of the heart is regular or irregular and, if irregular, whether the arrhythmia is in time or in force or both, that is, whether there are differences in the intervals between the ventricular contractions, or in the power with which the heart contracts or these are combined. We observe also in this way the general character of the heart's action, that is, feeble or strong; heaving powerfully so as to move the whole chest, as in great hypertrophy or the overaction of mental or physical excitement—palpitation; that it has the diffuse slap often encountered in dilatation of the right ventricle, the sharp tap of mitral stenosis or the slow, heaving, forcible impulse sometimes met with in aortic stenosis.

The more sensitive tips of the fingers are next brought into service. They are placed over the point of maximum impulse and moved in various directions. The apex of the heart as determined by finger-tip palpation and by percussion is usually two or three centimetres below and to the left of the point of maximum or visible impulse. It frequently happens that the impulse not recognized upon inspection may be felt and rarely that a visible impulse cannot be appreciated by the trained touch. These two methods must be used in all cases.

Inspection and palpation yield the most satisfactory results in the study of the size of the heart. The base of the organ is fixed and is as a rule not greatly displaced even by the pressure of an aneurism or new growth. To fix the position of the apex is to determine the long axis of the heart and gain a fairly correct idea of its size. The data obtained by percussion are much less definite, partly because of inherent difficulties in recognizing the limits of dulness in the rounded body of the heart surrounded by resonant lung and partly because of the modifying effects of pleural adhesions or effusion, gastric dilatation or abdominal tympany. When the impulse cannot be located by inspection or palpation, we employ auscultation and consider the clinical impulse to be near the point at which the first sound is most distinctly heard.

The changes in the relation of the apex to the wall of the chest caused by changes in the posture of the patient have already been considered.

Extracardiac Pulsation. — Pulsation beyond the limits of the heart is frequently seen, but its precise location, extent, and character are best studied by the sense of touch. A heaving impulse at the root of the neck occurs in hypertrophy, especially that form associated with aortic insufficiency and in overaction from nervous causes. It occurs also in anæmia and large hemorrhages, in apoplexy, and rarely in the stage of onset of intense infections, as variola. It is a conspicuous phenomenon in exophthalmic goitre. In neurotic persons the pulsating dilated transverse aorta may in rare instances be felt in the sternal notch—dynamic pulsation. Aneurism of the innominate artery or of the transverse portion of the aortic arch may give rise to similar pulsation. Anomalies in the distribution of the subclavian or thyroid arteries may also give rise to pulsation in this region. In old pleural adhesions at the apex and in pulmonary tuberculosis subclavian pulsation is often marked and extended. Pulsation commonly to the right of the manubrium, sometimes to the left of it, occurs in aneurism of the thoracic aorta and may often be felt when it is not seen. The force and extent of the impulse in pulsating empyema are best estimated by palpation.

Epigastric Pulsation. — This phenomenon is generally regarded as the sign of hypertrophy of the right ventricle and this view is unquestionably in some cases correct. The hypertrophied and overacting right heart communicates its movements to the tissues at the tip of and below the ensiform cartilage. The retraction corresponds to time with the ventricular systole and is due to the negative pressure caused by the alteration in size and diminution in the volume of the ventricles at this moment of the heart's revolution. Epigastric pulsation has been observed in cases in which no hypertrophy of the right ventricle has been found post mortem. Liver pulsation is much more frequently palpable than visible, and the distinction between this condition and a liver jogged by an overacting heart may often be made by bimanual palpation, since a pulsating liver expands and contracts, a jogged liver merely moves. Bimanual palpation, one hand upon the upper dorsal spine and the other upon the manubrium, may detect the expansile pulsation of a deep-seated aortic aneurism which presents no external signs. Diastolic shock is an important physical sign of aneurism. The tips of the fingers upon the sac in case erosion of the chest wall has taken place, or upon the surface directly overlying the sac, may often detect a diastolic shock, sometimes of considerable force.

Crepitation.—In wounds and operations upon the neck and chest air may find its way into the subcutaneous tissues and give rise to crepitation upon palpation. In rare cases this condition may result from the rupture of dilated peripheral pulmonary vesicles in emphysema.

Succussion.—When both fluid and air are present in a large space with rigid walls, as in pneumohydrothorax or pneumopyothorax, a distinct vibration or impulse may be felt upon shaking the patient or causing him to suddenly twist his body. This phenomenon, which is accompanied by a splashing sound, constitutes the sign known as Hippocratic succussion.

The arterial pulse is studied by palpation. This subject will be fully considered in a later section.

Thrills.—The palpable vibrations of the surface transmitted from the interior of the heart or arteries are known as thrills. They are usually confined to limited areas and may be easily overlooked unless the surface is first searched with the palmar surface of the open hand. They may then be studied with the finger-tips. They can frequently be felt only upon the lightest pressure, wholly disappearing if the pressure be increased. The sensation has been compared to that communicated to the hand by the purring cat—*frémissement cataire.* Thrills are usually felt during a portion of the cardiac revolution only—presystolic, systolic, post-systolic. They may disappear when the heart is acting feebly and become manifest again when, with general improvement in the condition of the patient, the heart contracts with greater power. They usually correspond in the time of the cardiac cycle with audible murmurs or bruits and are significant of the same lesions and produced by the same mechanism, namely, fluid veins, the vibrations of which transmitted through tissues to the surface are realized by the ear as murmurs, by the touch as thrills. In other words the thrill is the sensory equivalent of the murmur. The fact that very coarse thrills, especially the presystolic thrill, sometimes occur when no murmur can be heard, does not militate against the foregoing statement, since regular vibrations may be palpable though not frequent enough to produce sound. It is in accordance with these statements that thrills vary in the rapidity of their vibrations—fineness, coarseness—and that the finer thrills correspond to the higher pitched murmurs and the reverse.

A thrill at the base of the heart of maximum intensity in the aortic area is common in aortic stenosis.

A thrill of coarse quality, limited in extent, presystolic in time, more marked during expiration, and most distinctly felt in the fourth or fifth intercostal space inside the midclavicular line—mitral area—is the sign of mitral stenosis.

A systolic thrill in the same area is sometimes present in mitral incompetence and in rare instances in aortic stenosis.

A thrill often accompanies the presystolic murmur of aortic incompetency—Flint's murmur.

A thrill diastolic in time is occasionally felt in aortic incompetency, but is not very common.

Thrills are common in congenital defects of the heart.

A thrill systolic in time at the second left costal cartilage and interspace—pulmonary area—is occasionally observed in exophthalmic goitre;

very rarely it is a sign of pulmonary stenosis. A diastolic thrill in this area may be the sign of a rare condition, pulmonary incompetency.

A thrill over the lower portion of the sternum and at its right border—tricuspid area—sometimes occurs in dilatation of the right ventricle and tricuspid incompetence.

Systolic thrills when beyond the limits of the precordial space are more likely to be indicative of thoracic aneurism than of valvular disease. They are oftenest felt to the right of the sternal border and above the fourth rib, but may be present in the left side in a corresponding region. They are more common in aortic dilatation than in sacculated aneurism.

It is important to recognize the difference between a thrill and the slight shuddering tremor which may be felt in the merely overacting heart, as in palpitation from any cause.

Fremitus.—*Fremere,* to roar or murmur as a crowd or mob; technically, palpable vibration. The difference between a thrill and fremitus is much more readily recognized than described. It is, however, an essential difference and depends upon the difference in the mechanism by which they are respectively produced. Fremitus is usually much coarser than thrills, the vibrations are irregular and variable, the extent is far wider, and fremitus, even when produced by the movement of the heart as in the friction fremitus of pericarditis, does not constantly conform to definite movements in the cardiac cycle. Fremitus is a tactile phenomenon communicated to the surface of the chest by the act of phonation—vocal fremitus; by the friction of roughened surfaces against each other—friction fremitus; or by the respiratory movement of exudates of varying consistency within the bronchial tubes—rhonchal fremitus.

Vocal Fremitus.—This physical sign is of great value in the diagnosis of diseases of the respiratory organs. It is frequently spoken of as tactile fremitus, but erroneously so, since all fremitus is tactile. The hand is laid upon the bared chest while the patient counts "one, two, three," or repeats some words, as "twenty-one" or "ninety-nine." Under normal circumstances the fremitus is more intense in men than in women, in adults than in children, and in persons whose voices are powerful and low-pitched than in those whose voices are feeble and shrill. The patient should be asked to repeat the same phrase as the examining hand passes from one part of his chest to another, and to let his speaking be loud, low, and slow, always as nearly as possible in the same tone. This method of physical diagnosis is without value in persons suffering from aphonia or in those so feeble that they can only use the whispering voice. It is practicable in young infants who cry during the examination.

VOCAL FREMITUS IN HEALTH. — The vibrations of the vocal cords in phonation are transmitted along the walls of the trachea and bronchi and the column of air which they contain to the surface of the chest, which is thus set into vibration from within. These vibrations vary in different regions normally, and are most distinct where the large bronchial tubes approach the chest wall, less distinct where the mass of intervening vesicular tissue is greatest, and feeble or absent where the lung tissue does not come into contact with the wall, as in the precordial space. Pathological conditions which increase the capacity of the lung to conduct vibrations, as consolidation, intensify the vocal fremitus; those which separate t lung from the wall, as pleural effusions, diminish or abolish it, as the ca may be. There is normally considerable difference in the intensity of t vocal fremitus in the two sides, especially in the upper regions. Th inequality is to be constantly borne in mind. The vibrations are mo intense on the right than on the left side, in the upper (subclavicula region than in the lower (inframammary), and in front than behind. It feeble over the scapulæ, and usually absent or very feeble in that porti of the precordial space which corresponds to the area of superficial cardi dulness. A thick layer of subcutaneous fat impairs the value of this phys cal sign, while a thin, elastic chest wall and deep voice render it very usef

VOCAL FREMITUS IN DISEASE OF THE RESPIRATORY ORGANS.—T vibrations are intensified by conditions which cause consolidation of t lung, as tuberculous infiltration, croupous and bronchopneumonia, hyp static congestion and atelectasis; they are enfeebled or absent altogeth in pathological conditions which separate the periphery of the lung fro contact with the chest wall, such as pleural effusion, pneumothorax, a cysts or tumors in the pleural cavity. Pleural thickening is usually attend with enfeeblement of the vocal fremitus proportionate to its degree, an as a much thickened pleura gives rise to impairment of resonance, t differential diagnosis between a moderate effusion and pleural thicken may be attended with difficulty.

Temporary disappearance of vocal fremitus in pneumonia in an ar corresponding to a lobe or part of a lobe may be caused by the plugg of a large bronchus with a mass of tenacious mucus. In the same mann a foreign body may cause localized absence of this sign. In infants a less frequently in adults distinct vocal fremitus is occasionally encounter upon the affected side in large effusions—a very puzzling phenomeno The most probable explanation of this anomaly in children is that t intense fremitus caused by violent crying is transmitted along the elast chest walls from the sound to the affected side of the chest; in adults, th tensely stretched strands or bands, the result of partial adhesions caus by a former attack of fibrinous pleurisy, conduct the vibrations from t compressed lung to the wall of the chest. In a moderate effusion und favorable circumstances the following variations may be recognize normal vocal fremitus over the apex, enfeebled fremitus in the mamma region, and the complete absence of this sign at the base.

If the limitations of its usefulness be borne in mind, vocal fremit is a sign of very great value, but it may mislead the unwary. In massi pericardial effusion it is of great service in the differential diagno between that condition and large left-sided pleural effusion.

Friction Fremitus.—In health the smooth and moist pleural and pe cardial surfaces move upon each other without appreciable sound. Wh these surfaces are the seat of a fibrinous exudate they cause friction soun which vary with the arrangement and density of the exudate and t energy of the respiratory or cardiac movements as the case may be. T vibrations which cause the sounds are transmitted to the surface and co stitute the tactile sign known as friction fremitus. The sensation co municated to the examining finger is that of grating or rubbing and vari he finest grazing to a coarse attrition. It corresponds in location xtent with the friction sound which is its auditory equivalent.

LEURAL FREMITUS is common in the infra-axillary region or below ipple and is not transmitted beyond a limited area.

ERICARDIAL FREMITUS, which is the sign of fibrinous or dry peri-is, is felt in the precordial space over the right ventricle. It does sually correspond in time accurately with the systole or diastole, the impression of being very superficial and is limited to a circum-d area. It differs from the thrills felt over the heart in the tactile ies referred to in a preceding paragraph.

LEURAL FREMITUS AND PERICARDIAL FRICTION FREMITUS disappear as on takes place, separating the roughened surfaces, and as adhesions op, by which the surfaces are united.

honchal Fremitus. — Coarse bronchial râles, both dry and moist, imes communicate irregular vibrations to the surface of the chest y recognized upon palpation. This form of fremitus is common in children and may occur in thin-chested adults. It differs from fric- remitus in being coarse and more irregular and varying in intensity uality with the râles that cause it. The sign is of little diagnostic value.

racheal tugging, a sign first described by Oliver, is of great value diagnosis of deep-seated thoracic aneurism. "Place the patient in ect position and direct him to close his mouth and elevate his chin most the full extent; then grasp the cricoid cartilage between the and thumb and use steady and gentle upward pressure on it, when, tation or aneurism exists, the pulsation of the aorta will be distinctly ansmitted through the trachea to the hand." A better method con-in the application of the index and middle fingers of the same hand e sides of the cricoid cartilage, or the physician may stand behind atient, who is seated, and place the forefingers upon the sides of the d, with gentle upward pressure. The downward tug may be readily nized. The tug is due to the fact that the arch of the aorta passes he left primary bronchus in such a manner that when the aorta is d it impinges upon the bronchus with each pulsation. The tension bronchus is communicated through the trachea to the larynx. A ward tug felt only upon inspiration is frequently present in health as no diagnostic value. Pulsation transmitted from the vessels of eck to the cricoid must not be confounded with tracheal tugging. movement of the former is forward and backward; of the latter a ct downward pull with release.

Palpation in the Examination of the Abdomen.

his is the most valuable of the methods of physical diagnosis in es of the organs below the midriff. The patient should be in bed he belly should be bared as for inspection. The hand of the physi-hould be warmed and applied to the surface with gentle pressure. r both hands may be necessary. Bimanual palpation may be from o side, the wall of the abdomen being deeply folded between the , or any accessible organ or tumor being thus investigated, or the bimanual method may be used in the study of the lateral regions of the abdomen, one hand being placed in the lumbar region, the other in front. In this manner the border of the liver may be raised up against the anterior wall or a floating kidney thrust forward for examination, or deep fluctuation elicited in paranephritic or appendiceal abscess, or a hydronephrosis studied, or the contour of an enlarged spleen or carcinoma of the sigmoid flexure made out. When the object of the examination is to localize and determine the degree of tenderness it is better to study the face of the patient than to depend upon his statements or exclamations. It is also important to distinguish between superficial tenderness, as in cutaneous hyperæsthesia and the deep tenderness of an inflamed or tumid organ. It will frequently be found that here as elsewhere, in neurotic persons, more vivid expressions of pain are called forth by a light touch than by firmer pressure—a fact in itself of great diagnostic importance.

Excessive abdominal fat, muscular tension, and ticklishness are obstacles. The first often nullifies the results of palpation; the others may be overcome. Muscular tension due to apprehension, the excitement of the occasion, or other nervous causes may be overcome by elevating the head upon pillows and causing the patient to flex his thighs and knees; continuous deep or rapid breathing is also useful. Tact and address on the part of the physician and suggestion are also to be employed. It is frequently necessary to examine the patient under general anæsthesia before expressing a final opinion as to the nature of the case, and finally, there are serious cases of abdominal disease in which it may become necessary to perform an exploratory operation to arrive at a positive diagnosis. Ticklishness is an obstacle of minor importance, but it may call for the exercise of much patience on the part of both the doctor and the patient.

It often becomes necessary to turn the patient from side to side or to examine him in the knee-elbow posture, or standing. A digital examination by the rectum or vagina with or without bimanual manipulation is frequently required in lesions of the lower portions of the abdomen.

The regions of the abdomen must be in turn systematically explored, the natural rings and accidental sites of hernia examined, and the general outline, contour, and condition of the belly, particularly as to its symmetry and elasticity, carefully determined. Large knowledge of the changes caused by abdominal disease and wide experience are required in this field of diagnosis. Here also a delicate and well-trained touch—*tactus eruditus*—is especially serviceable.

The signs obtained by inspection are confirmed by palpation. Much knowledge is obtained by the latter method. This comprises the following subjects:

The Condition of the Abdominal Walls,
General and Local Fluctuation,
Pulsation, Thrill and Fremitus,
The Respiratory, Postural, and Manipulative Movements of Organs or Tumors,
Peristaltic and Fetal Movements,
The Outline and Relations of Palpable Tumors,
Their Density and Elasticity,
The Nature of the Surface of Tumors.

The Abdominal Walls.—In healthy young persons the belly walls are soft and elastic but neither tense nor relaxed, and the curvature of the abdomen as determined by inspection and palpation is symmetrical and uniform.

Abnormal firmness and relaxation are attended by a loss of healthy elasticity. Local firmness may be caused by inflammatory or carcinomatous infiltration, and general hardness by the massive enlargement of the liver, spleen, uterus, an ovary, or other organ, or diffuse malignant deposits in the intestines or peritoneum. Muscular rigidity is characteristic of the early stage of peritonitis. It may be localized, as in the right lower quadrant in appendicitis or enteric fever, or general. Local rigidity of the bellies of the recti is sometimes observed in neurotic persons and may be mistaken for a tumor, as a thickened or carcinomatous pylorus. Local rigidity with meteorism constitutes phantom tumor. A generalized inelastic doughy sensation upon palpation is often observed in tuberculous peritonitis. The general distention of ascites is associated with dulness save in the upper portions, where there is tympany, and with fluctuation; that of meteorism is associated with tympany everywhere, including the dependent parts, and a balloon-like elasticity quite different from that of the normal abdomen. The anasarcous abdominal wall is doughy, inelastic, and pits upon pressure; dropsical accumulations are seen in the flank and elsewhere in the more dependent parts.

Relaxation follows the resorption of large amounts of fat and repeated childbearing. In such cases the belly wall is often pendulous and remarkably puckered and thrown into folds when the patient lies upon her back. Relaxation also follows ascites of long standing and the removal of large tumors and is usually present in old age and the advanced stages of wasting diseases. In women who have borne many children wide separation of the recti is occasionally seen, the connective tissue of the linea alba being enormously stretched and thinned and the gastric and intestinal peristalsis plainly seen and felt over a large area in the middle of the abdomen. In such cases very large ventral hernia and downward displacement of the abdominal viscera—Glénard's disease—are commonly present.

Local tumors of the abdominal walls are abscess, attended by local induration and central softening; cysts, oval or circular in outline, tense, elastic and fluctuating; enlarged lymph-nodes in the inguinal region; subcutaneous carcinomatous and sarcomatous tumors, which may be movable or immovable, and arranged in irregular masses as is common in the former, or scattered singly over a wide area as in sarcoma; and hernia. The last appears in definite locations, as the inguinal and crural rings, the umbilicus, in the linea alba—*ventral hernia*—and in the sites of scars after surgical operations. Upon palpation the hernial tumor is usually soft, elastic and reducible; omental hernias are doughy and irregular in outline. The hernia which cannot be returned to the abdomen by manipulation is irreducible, that which is tightly constricted and is therefore likely to become or has already become sphacelated is strangulated.

The umbilicus that pouts in ascites or pregnancy is smooth, stretched, and somewhat translucent. In umbilical hernia the ring is usually distinctly felt; when omental the tumor at the navel is often large, firm, irregular in its surface and irreducible and may suggest a malignant growth. The umbilicus, normally somewhat movable, when the seat of secondary carcinoma, usually by extension from the liver, becomes fixed and is indurated and nodular. Tuberculous infiltration of the tissues around the navel has been observed in tuberculosis of the peritoneum. A deeply seated, painful swelling of the navel is usually an abscess.

Fluctuation.—This sign is elicited by combined bimanual percussion and palpation, those methods being employed at the limits of the area examined, as for example at the right and left lateral regions of the abdomen in suspected ascites and at the opposite borders of circumscribed collections of fluid as in pancreatic or other cysts. To elicit general fluctuation the palpating left hand or finger-tips are lightly laid upon the surface of the right side of the patient's abdomen, while with the fingers of his right hand the examiner percusses or taps somewhat sharply upon the left side of the abdomen. If there be ascites a transmitted wave corresponding to each tap is felt upon the opposite side. This wave is also in many cases visible. Very light percussion may bring out this physical sign when the wall of the abdomen is thin. The thin ulnar border of the hand of an assistant must be rather firmly pressed against the abdomen in the middle line to arrest the undulatory transverse movement of the wall, which very often simulates the fluctuation of peritoneal effusion. This sign does not arise unless the fluid is freely movable and sufficient in amount to rise above the pelvis—two or more litres.

The method of determining fluctuation in circumscribed collections of fluid, as pancreatic or other cysts within the abdomen, circumscribed effusions, dropsy or empyema of the gall-bladder, etc., is somewhat different in technic and available only in patients whose belly walls are comparatively thin. The tips of the palpating fingers are lightly placed in contact with the surface at one border of the area under examination while the opposite border is sharply but lightly flicked with the nail—dorsal surface of the tip of the middle or ring finger suddenly disengaged from contact with the palmar surface of the thumb, as one flicks a crumb. By this method not only can fluctuation of limited extent be determined but also the limits of the area in which it is present defined.

Pulsation, Thrill and Fremitus.—Pulsation.—In thin persons the normal pulsation of the aorta may be felt upon deep palpation in the middle line about the level of the umbilicus. Abnormal pulsation of the abdominal aorta is of two kinds, the so-called dynamic pulsation seen in neurotic persons, not expansile and not associated with tumor or other signs of dilatation of the vessel, and the expansile pulsation of abdominal aneurism, in which a tumor that can be grasped between the hands and is the seat of distinct expansile pulsation may be present together with other signs of aneurism. The differential diagnosis between these two forms of pulsation should not be a matter of doubt. Pulsation is sometimes transmitted from the aorta to a tumor overlying it in such a manner as to simulate aneurism, especially as the pressure of the tumor may cause both bruit and thrill. The fact that the pulsation is not expansile and the palpation of the tumor in the knee-elbow posture, when the movement of the aorta is no longer communicated to it, serve to render the differential diagnosis between such a tumor and aneurism a matter of comparative ease. Dynamic pulsation of the aorta is felt in the course of the vessel in the middle line and slightly to the left of it; that of aneurism is usually more extended transversely and may be felt some distance to the left, even reaching almost as far as the iliac crest, as I saw in a case verified by autopsy.

The liver pulsation due to tricuspid incompetency—hepatic venous pulse—may frequently be recognized upon palpation, especially bimanual palpation, when it is not visible upon inspection, and by the former method the difference between the expansive movement of a pulsating liver and the jogging due to the communicated movement of the heart may be appreciated.

Thrill.—This sign is sometimes met with in abdominal aneurism and tumors pressing upon the aorta. It has little diagnostic significance.

Fremitus is the sign of echinococcus cysts—hydatid fremitus or thrill. The tumor is soft, elastic, fluctuating, and in the majority of cases the seat of a peculiar vibration or fremitus, which may be felt by palpation with two or three fingers of the same hand or by placing three finger-tips widely separated upon the surface and lightly percussing the middle finger. Gall-stone fremitus is sometimes elicited upon palpation of the gall-bladder distended with a large number of calculi. It is a comparatively rare but very important sign.

Movements of Abdominal Organs or Tumors.—The movements ol intra-abdominal organs and tumors constitute physical signs of great value in diagnosis. They are *respiratory, postural, and manipulative.*

Respiratory movements are communicated to the organs in close relation to the diaphragm, especially the liver, spleen, and to a less extent the kidneys. Tumors of the stomach are usually but little influenced by the movements of the diaphragm. Conditions which hinder the respiratory movements of the diaphragm, such as pleurisy, emphysema, massive enlargement of the liver or spleen, advanced pregnancy, meteorism and ascites, restrict or wholly arrest the respiratory movements of abdominal viscera. The anatomical relations of the pancreas and retroperitoneal glands are such that they are not influenced by the movements of respiration. Very large cysts of the pancreas may show slight movement on deep breathing.

Intra-abdominal new growths which are influenced by respiratory movements originate in the upper portion of the cavity; those which manifest no respiratory movement upon careful palpation commonly but not always develop from the pelvic organs or from structures directly connected with the spinal column behind the peritoneum—pancreas, retroperitoneal lymphatic glands, aneurism.

Postural Movements.—Free fluid in the cavity tends to gravitate to the most dependent space, while the air-containing intestines float upon the surface of the fluid. Small effusions may cause dulness in the umbilical region when the patient assumes the knee-elbow position. Floating viscera, kidneys, spleen, and in very rare instances the liver, are recognized upon palpation by their size, shape, and general relationships. A liver dislocated has little range of movement, but the kidney and spleen may be found in distant regions of the abdominal cavity, even at the brim of the pelvis.

THE TECHNIC OF PALPATION OF THE KIDNEY.—The recognition of a displaced kidney is not attended with difficulty. Palpation should be bimanual, one hand pressing upward from the lumbar region while the other is gently moved over the anterior surface of the abdomen, which should be as relaxed as possible. The tumor is oval, smooth, firm, and has the oblong shape of the kidney. It is sometimes possible to recognize the hilum and to feel the pulsating renal artery. The tumor is usually sensitive to firm pressure and freely movable. In the knee-elbow posture it advances towards the wall of the abdomen, while it sinks backward and may be pressed into its normal position when the patient assumes the dorsal decubitus. In the lateral and erect postures, it sinks to the lowest point of its range of movement. Except in the case of a much elongated mesonephron, it moves also with the movements of respiration. Wandering kidney is more common in women, in multiparæ, upon the right than the left side and is occasionally bilateral.

THE TECHNIC OF PALPATION OF THE SPLEEN.—The patient should be placed in a position midway between right lateral and dorsal, with his left hand upon his head. The thighs should be flexed in order to relax as far as possible the abdominal wall. The head should be slightly retracted and the patient directed to breathe deeply and slowly. The physician, standing at the patient's right, exerts with his left hand firm pressure upon the infra-axillary region downwards and forwards while, with his right hand, he presses the soft belly wall below the arch of the ribs upwards and inwards to determine whether or not the lower border of the spleen can be felt and in particular at the end of deep inspiration. Too much force must not be employed lest a greatly softened spleen, as in enteric fever, might be ruptured. The data yielded by percussion in the examination of the spleen are rendered uncertain by gastrectasis, meteorism, pleural effusion, and fecal accumulations in the colon and new growths in the splenic region. The results of palpation in moderate enlargement are much more satisfactory and reliable.

The diagnosis of massive enlargement of the spleen is usually a matter of ease and certainty. The contour of the tumor, upon which may be distinctly felt a sharply rounded inner border, often notched opposite the hilum, its firmness, its slight movement upon deep breathing, and the smoothness of the surface are of diagnostic importance.

Wandering spleen is not often difficult of recognition. The displaced organ is readily palpable below the left hypochondrium, less often in the umbilical or left iliac region, and very rarely at the brim of the pelvis, as a smooth oval tumor of the outline of the spleen, notched and freely movable upon change of posture and by manipulation. If the organ occupies a position to which the movements of the diaphragm do not extend, it does not move even upon the deepest respiration.

Movements upon Manipulation.—All abnormal organs and tumors that change their position in response to changes in posture are movable upon manipulation or palpation. The list comprises floating liver, spleen, and kidney; in the absence of adhesions, tumors of the pylorus and less frequently of other parts of the stomach, as the greater curvature, new growths in the intestines, excepting the ascending and descending colon;

ecal accumulations, gall-stones and enteroliths; mesenteric and omental umors. The range of movement is limited in tumors of the gall-bladder and pancreatic cyst, in the upper regions of the abdomen; very limited in tumors of the ascending and descending colon laterally and enlargements of the uterus and ovaries in the lower segment. All malignant and some benign tumors tend to contract adhesions which interfere with movement. The following are immovable: small tumors of the pancreas, retroperitoneal growths, peri-appendiceal infiltration, adhesions and abscess, abdominal aneurism and abscesses.

Peristaltic and Fetal Movements. — The peristaltic movements may sometimes be felt, as they may be seen, in thin individuals in health and when in obstruction of the bowel they become excessive. In chronic, slowly developing stenosis of the gut the musculature of the intestines undergoes hypertrophy and the peristalsis becomes proportionately more powerful. Antiperistaltic or reverse waves may sometimes be felt. Palpable coarse intestinal movements with the formation of knots accompanied by borborygmi may be present in colic and in hysteria. The gastric and intestinal movements are very plainly felt and seen in cases of wide separation of the recti in women who have borne many children.

The movements of the fœtus may be often plainly felt upon palpation, and in advanced pregnancy the position of the fœtus may be recognized by this method of examination. All these movements may be rendered more active by manipulation and the sudden application of cold.

Outline.—By palpation we determine whether an intra-abdominal mass is round, oval, or irregular in outline; whether it is rough, nodular, or smooth; whether it resembles a viscus as the kidney or spleen in shape and has characteristic anatomical features, as the hilum or a pulsating artery. We ascertain its apparent point of origin, as in the epigastrium, the lateral regions of the abdomen, or the pelvis, and whether or not it has direct attachments or relations with another organ, such as may be made out between an enlarged gall-bladder and the liver, carcinoma of the pylorus and the stomach, or a large cyst in the left hypochondrium and the pancreas.

Density and Elasticity.—The signs relating to the consistency of an intra-abdominal mass can be ascertained by palpation alone. We thus determine whether it is fluctuating as in abscess or cyst; soft as in rapidly developing new growths and aneurism; moderately firm as in organs the seat of congestion and hypertrophy, or dense as in slowly developing carcinoma or interstitial overgrowths. In fecal accumulations the tumor is sometimes hard and firm, sometimes soft and doughy and can be indented by the finger.

Surface.—Palpation enables us to study the surfaces of organs and tumors. The smooth surface of an amyloid or fatty liver, the coarse granular surface of the liver in atrophic cirrhosis, the nodular liver with its rounded isolated eminences at the summit of which slight depressions may be felt—Farre's tubercles—in cancer, are examples of surface changes of diagnostic importance. The smooth surface of the distended gall-bladder stands in strong contrast with the irregular outline of carcinoma of the pylorus; the irregular multilocular echinococcus of the liver can hardly be differentiated from hepatic cancer, but is wholly unlike the smooth, elastic, and vibrating single hydatid cyst. The smooth, elastic, and fluctuating cyst in hydronephrosis differs altogether from the firm, nodular and irregularly shaped mass in carcinoma of the kidney; and the smooth, ovoid, nearly centrally placed tumor of early pregnancy is wholly unlike the tumor formed by irregular, coarsely nodular subperitoneal uterine myomata.

MENSURATION.

The use of instruments of precision is of great importance in physical diagnosis. Such appliances vary from a simple graduated tape to the most intricate and delicate hæmodynamometer or polygraph. The writer holds the opinion that simplicity both of method and of instruments yields the most satisfactory results at the bedside, and that intricate and costly mechanical devices which require great technical skill and consume much time are better suited to scientific research than to every-day clinical work.

Measurements of the chest—thoracometry—may be conveniently made by a steel tape graduated upon one side in centimetres, on the other in inches; the diameters are taken by calipers made for the purpose.

The circumference and semicircumferences are taken at the level of the nipples or the fourth costosternal articulation in quiet breathing, in full held inspiration and on full expiration. Care must be taken that the tape is horizontal. The normal chest is nearly but not quite symmetrical, the right semicircumference being in the majority of individuals slightly larger than the left—an average difference of about half an inch. It is well to make a mark with a dermatographic pencil in the median line in front and over a vertebral spine at the same level and measure the semicircumference from point to point on each side for comparison. Two tapes attached to a little wooden saddle which fits over a vertebra are useful to determine the semicircumference on quiet breathing and the differences on forced respiration. The average circumference in men is 34.3 inches (87 cm.); in women 29.5 inches—(75 cm.). The difference in forced expiration and full held inspiration varies in normal individuals between 1.5 (4 cm.) and 5 inches (12.5 cm.).

The main diameters of the chest at the same level as taken by compass calipers with curved arms or slide calipers are: anteroposterior (the depth of the chest) average in repose in men 7.5 inches (19 cm.); in women 6.9 inches (17 cm.); bilateral or transverse (the breadth of the chest), average in men 9.9 inches (25 cm.).

Spirometry.—Mensuration may be employed not only to ascertain the size of the chest and its movements but also to learn the volume of the tidal air. The instrument used for this purpose is the spirometer. Various forms are in use but the results are far from satisfactory. The instruments are cumbersome and require a certain amount of training to obtain constant results. The sex, age, weight and height must be taken into account. Thus for every inch above five feet, eight cubic inches are to be added to the normal standard, which for five feet is 174 cubic inches. The estimated average lung capacity for height in males between sixteen and forty years of age is, according to Otis, twenty-three cubic centimetres for every centimetre of height; in females at nineteen years of age, it is fifteen cubic centimetres for each centimetre of height.

Waldenburg's pneumatometer is an apparatus designed to measure the respiratory energy. Normally the power exerted in expiration is greater than in inspiration by from twenty to thirty millimetres of mercury. In emphysema and asthma the expiratory pressure is greatly diminished, while in certain forms of phthisis the inspiratory power is much lessened.

Cyrtometry.—The determination of the outline of a cross-section of the chest may be made with an instrument called a cyrtometer—measure of the curve. This procedure is of no great use in ordinary clinical work but very suggestive and important in teaching. Elaborate and costly instruments are not necessary for this purpose. The best device consists in a little metal saddle made to fit the spine, to each side of which is hinged a strip of leaden ribbon half an inch in width and thick enough to be easily bent so as to conform to the surface of the chest, yet retain its form when removed. The saddle is set upon the spine at the level selected, the leaden band is carefully adjusted to the surface on each side and made to meet at the median line in front. It is then released, opened at the hinges, removed from the chest and then laid upon a sheet of paper, the ends being brought together at the point of meeting in the median line. The outline is controlled by the fixation of the main diameters by means of the calipers. A soft pencil is then used to make the tracing on the inside of the cyrtometer. The various deformities of the chest described under inspection may be thus depicted.

Circumferential measurements of the abdomen at the level of the umbilicus and vertical measurements from the ensiform cartilage are useful, especially for purposes of comparison in ascites and enlargements from tumor or other conditions. They are best made with the ordinary graduated tape. Measurements from various fixed points upon the surface of the thorax or abdomen are necessary for purposes of record.

The Sphygmograph.—This is an instrument for the graphic registration of the pulse. There are various forms. Vierordt's, 1855, was the earliest. Marey's, 1860, was more practical and as modified by Mahomed and others is still much used. The instruments of Sommerbrodt, Jaquet and Frey are more recent. That of Dudgeon has many advantages and is in general use. Dudgeon makes the following claims for his instrument:

That it magnifies the movements of the artery in a uniform degree, namely, fifty times; that the pressure of the spring can be regulated from 1 to 5 ounces; that it requires no wrist rest and can be used with equal facility whether the patient be standing, sitting, or recumbent; that a tracing can be made with it almost as quickly as the pulse can be felt with the finger; that owing to its great sensitiveness it records the slightest deviation in the form or character of every beat; its construction is so simple that any watchmaker can repair it if broken; that it is so small, 2¼ x 2 inches, and so light, 4 ounces, that it may be carried in the pocket; that it is much less expensive than other forms of the instrument.

The sphygmograph is of great value in clinical work but rarely essential to a diagnosis. That which it adds to the information obtained by the well-trained finger relates to minutiæ many of which are still in dispute.

CYRTOMETRIC TRACINGS.

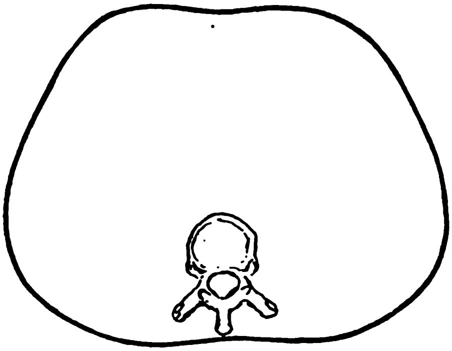

FIG. 48.—Outline of normal chest.

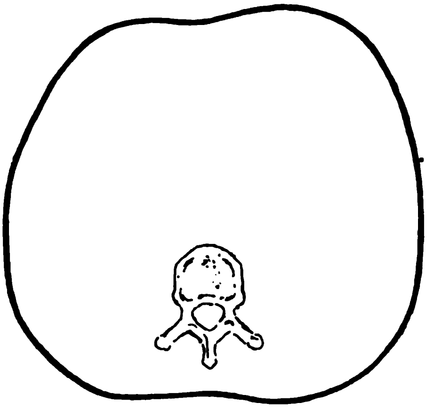

FIG. 49.—Outline of emphysematous chest.

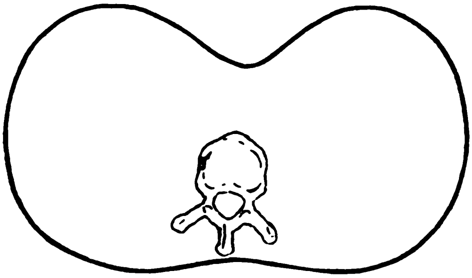

FIG. 50.—Outline of chest showing "funnel-shaped" deformity.

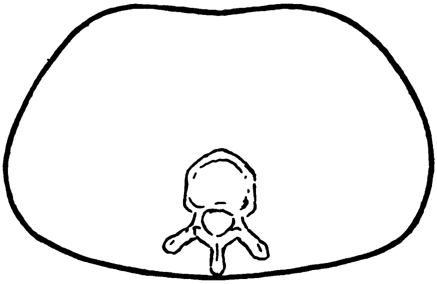

FIG. 51.—Outline of phthisical chest.

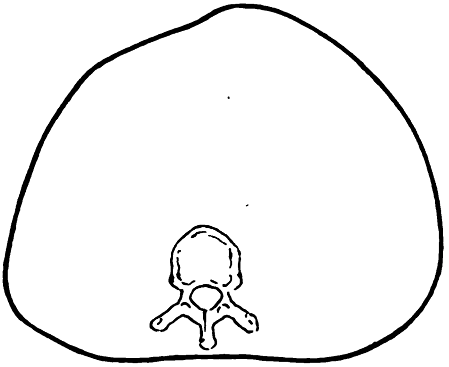

FIG. 52.—Outline of the chest in spinal curvature.

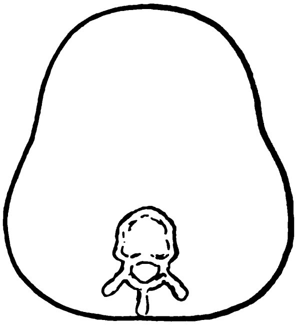

FIG. 53.—Outline of the chest in rickets.

The Sphygmogram under Normal Conditions.—The percussion stroke or ascending limb is caused by the wave-like transmission of the impulse communicated to the blood in the arterial system by the ventricular systole. The elastic walls of the suddenly distended artery contract slightly, causing the aortic or pretidal notch, and again expand as indicated by the tidal wave, after which the pressure again diminishes until the closure of the aortic valves, by which a base of support is presented to the blood column with a resultant recoil which is manifested in the dicrotic wave. Insignificant undulatory curves in the lower part of the descending or catacrotic limb, scarcely shown in ordinary tracings, are caused by minor oscillations of the arterial walls. The intrinsic movements of an extremely delicately adjusted and sensitive writing needle are to be taken into account but cannot be determined. It is evident that the excursion of the needle will be exaggerated when the pulse is quick and of large volume and that under these circumstances the tracing represents the qualities of the pulse plus certain qualities of the instrument not shown in tracings of other pulses, particularly those of small volume, high tension, and tardy development.

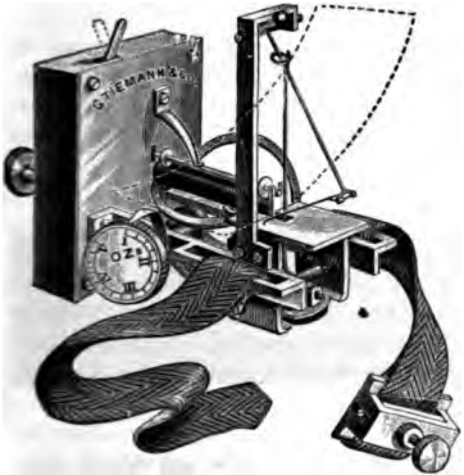
G. TIEMANN & Co.

FIG. 54.—Dudgeon's sphygmograph.

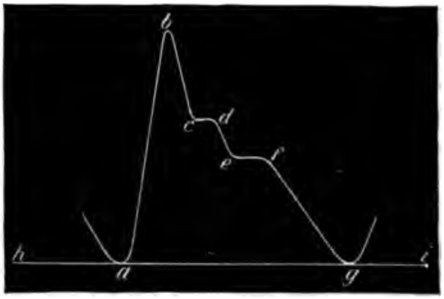

FIG. 55.—Diagram of normal pulse tracing. *a, b,* up-stroke, percussion stroke; ascending or anacrotic limb. *b, g,* descending or catacrotic limb. *a, b, c,* percussion wave; *c, d, e,* predicrotic or tidal wave; *e, f, g,* dicrotic or recoil wave; *b, c, d,* pretidal notch; *d, e, f,* the aortic notch; *h, i,* base line. The period indicated between *a* and *e* corresponds to the ventricular systole; the period between *e* and *g* to the aortic closure.

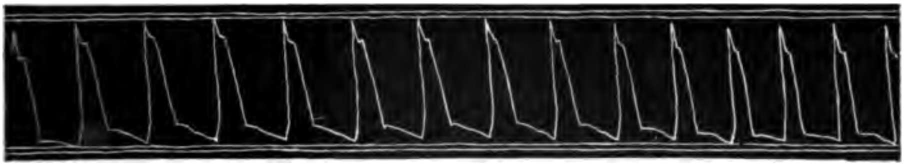

FIG. 56.—Aortic regurgitation.

The Diagnostic Significance of Sphygmograms.—In the normal sphygmogram the up-stroke is straight, almost vertical, of moderate amplitude as compared with the tracings of low and high tension pulses, the apex acute rather than obtuse, the descent gradual, interrupted by a small tidal and well-marked dicrotic wave. Sphygmograms are read from left to right.

Minor departures from the normal in any of these particulars cannot be regarded as of diagnostic value. The tracings are much modified by certain pathological conditions and the following points deserve consideration:

Amplitude.—The length of the percussion stroke varies considerably under normal conditions. It is dependent primarily upon the quickness of the pulse, secondarily upon the relaxation of the peripheral vessels. A long up-stroke indicates a pulse of low tension and large volume. The up-stroke is elongated in pyrexia. A striking character of the tracing of aortic regurgitation is the long up-stroke. On the contrary the percussion stroke is short when the pulse is of small volume or of high tension, as is seen in tracings from cases of arteriosclerosis, aortic stenosis, mitral stenosis and regurgitation and aneurism.

Fig. 57.—Mitral stenosis.

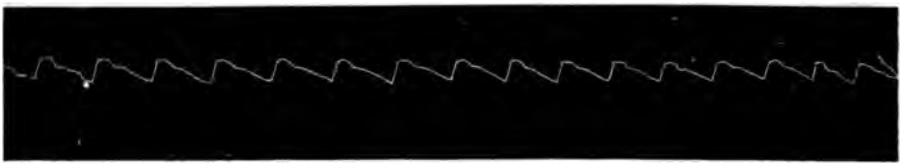

Fig. 58.—Mitral stenosis.

Direction.—The direction of the percussion stroke is dependent upon the quickness of the pulse. It is vertical or nearly so in tracings from cases in which the ventricular systole is sharp, especially in cases of dilated hypertrophy. The up-stroke is vertical in the tracing of pulses of low tension and both vertical and elongated in aortic incompetency. It is oblique or sloping in fat persons, the force of the percussion impulse being taken up by the thick tissue overlying the artery. Tracings of this kind are characteristic of cases in which there is a relatively slow discharge of blood from the ventricle into the arterial system, whether in consequence of lesions of the vessels or lesions of the heart itself. A sloping anacrotic

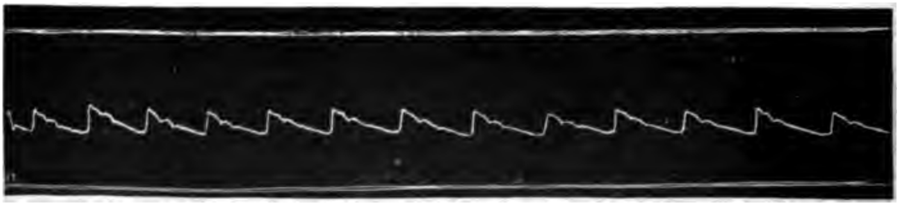

Fig. 59.—Arteriosclerosis.

limb occurs in tracings in arteriosclerosis, high tension from any cause, and aneurism. It is seen also where the left ventricle is weak, or where the mass of blood in the ventricle is divided, as in mitral incompetence, or where there is mechanical obstruction of the flow of the blood through the heart, as in mitral and aortic stenosis.

Apex or Summit.—The apex is sharp or pointed in the great majority of tracings, both normal and pathological. Tracings in which the apices are blunt or broad are frequently the result of improper adjustment of the sphygmograph or too great pressure. Tracings of this kind are sometimes obtained in high tension pulses, arteriosclerosis, aortic stenosis, and especially in aneurism.

Line of Descent.—This in a normal pulse tracing is gradual and undulatory, showing in succession the pretidal notch, the tidal wave, the aortic notch, and the dicrotic wave. Under normal circumstances the blood takes some time to flow from the arterial system into the capillaries and the subsidence of the pulse wave is gradual and the line of descent sloping. The line of descent is relatively abrupt when the outflow from the arterial system into the capillaries is rapid, as in tracings from cases of great relaxation of the peripheral circulation and in aortic regurgitation in which the arteries suddenly collapse in consequence of the defect in the aortic valves; it is more gradual than normal when the outflow is retarded, as is the case in arteriosclerosis, during a chill and in the early stages of peritonitis. Great irregularity in the line of descent is frequently seen in tracings of mitral disease, especially during impairment or rupture of compensation. All forms of intermittence and irregularity of the pulse are graphically represented in properly taken sphygmograms.

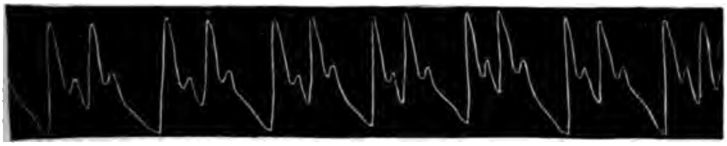

Fig. 60.—Pulsus bigeminus.

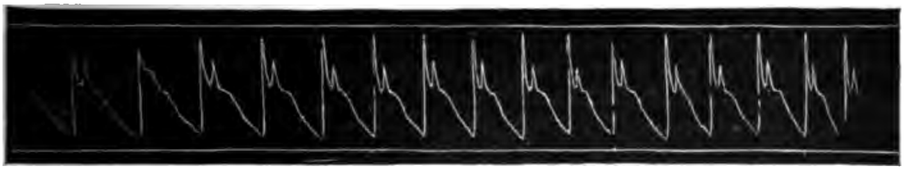

Fig. 61.—Pulsus bisferiens.

Tidal Wave.—The prominence of this curve indicates increase of the arterial tension during the ventricular systole. It occurs in some cases of arteriosclerosis and in aortic stenosis. This wave is sometimes perceptible to the finger—*pulsus bisferiens.* It is small or absent when the heart is very weak, or when with moderate systolic force there is free peripheral circulation. It is absent in mitral and aortic incompetency of high grade. It occasionally happens that the tidal or predicrotic wave is present in some pulse curves of a tracing and absent in others—a condition arising from variations in the amount of blood discharged into the arterial system during the ventricular systole. Tracings showing a marked predicrotic wave upon alternate beats or at other intervals indicate derangement of the nervous mechanism of the heart.

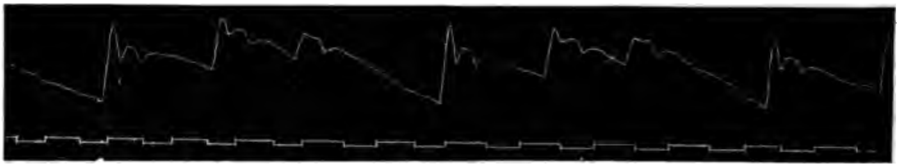

Fig. 62.—Pulsus trigeminus.

Dicrotic Wave.—This wave is usually present in the normal pulse tracing. It corresponds to that period of the cardiac revolution immediately following the closure of the aortic valves. When the vasomotor tone is good and the arterial tension high the dicrotic wave is feebly marked or absent. It is absent or faintly indicated in free aortic regurgitation. In some cases of arteriosclerosis when well marked, it may be recognized by the palpating finger, and the pulse is spoken of as dicrotic. Different degrees of dicrotism are shown in tracings. When the dicrotic wave is well marked but the aortic notch is above the respiratory or base line, the pulse is called dicrotic. When the aortic notch falls to the level of the base line the pulse is said to be fully dicrotic. When the aortic notch falls below the level of this line the pulse is called hyperdicrotic. If the hyperdicrotic pulse is very rapid the second beat is altogether lost and the pulse is said to be monocrotic.

Fig. 63.—Dicrotic pulse.

Respiratory or Base Line. — In the normal sphygmogram the lowest points of the percussion strokes of successive pulse waves are on the same horizontal plane, and a line drawn through these points is called the base or respiratory line. The term respiratory is used to designate this line because the inspiration and expiration to some extent in health, but much more in disease, exert a marked influence upon it. Full and sudden inspirations reduce the arterial tension and lower the base line. Forced expiration on the contrary increases arterial tension and raises the base line. In cases of severe dyspnœa from any cause the base line is undulatory, falling with inspiration and rising with expiration.

The use of the sphygmograph cultivates habits of clinical exactness; but much satisfactory work can be accomplished without it.

Mackenzie's Clinical Polygraph.—By means of this instrument, for a description of which the student is referred to "The Study of the Pulse," London, 1902, graphic tracings of the venous pulsations in the neck or liver are made in connection with sphygmographic tracings of the radial pulse. The simultaneous tracings upon the same strip of paper afford an opportunity for accurate timing of the radial and the venous pulses for clinical purposes by the familiar method of laboratory research. This method may be made use of in hospital work and under certain circumstances in the consultation room, and, by a modification of the instrument, the portable ink-writing polygraph may be used in private practice at the bedside.

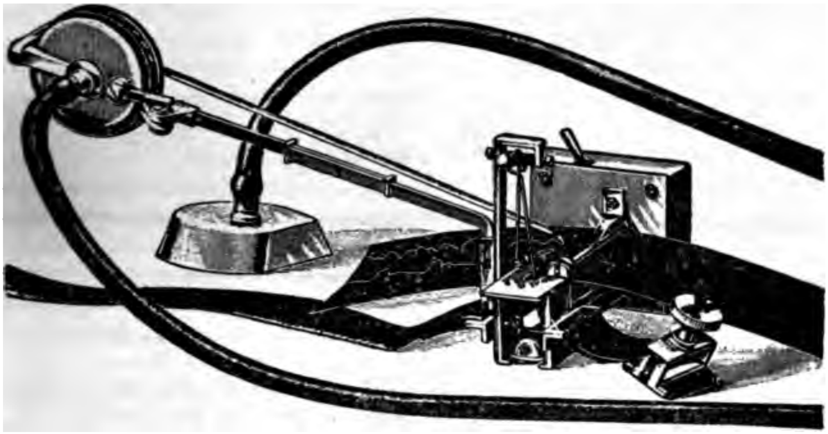

Fig. 64.—The Mackenzie ink polygraph.

THE ELECTROCARDIOGRAPH.

Great as have been the advances made in the knowledge of the origin and course of the cardiac impulse and the derangements of the cardiac rhythm by physiological and clinical studies by means of the polygraph, it early became apparent that this method, like those which had preceded it, has its limitations, since it fails to indicate in which chamber of the heart

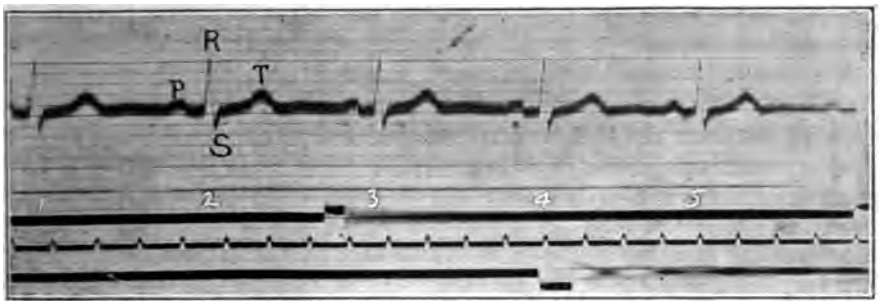

Fig. 65.—Electrocardiogram from a normal man (D1).

a disturbance of rhythm has its origin. The perfection of the string galvanometer of Einthoven, an instrument of sufficient delicacy to determine and record the electrical variations which accompany the phases of the cardiac contraction in the form of the electrocardiogram, affords a new method of investigation of the greatest importance. The cost of the apparatus and the great technical skill required in its use restrict its employment, but insure at the same time accuracy in the results.[1]

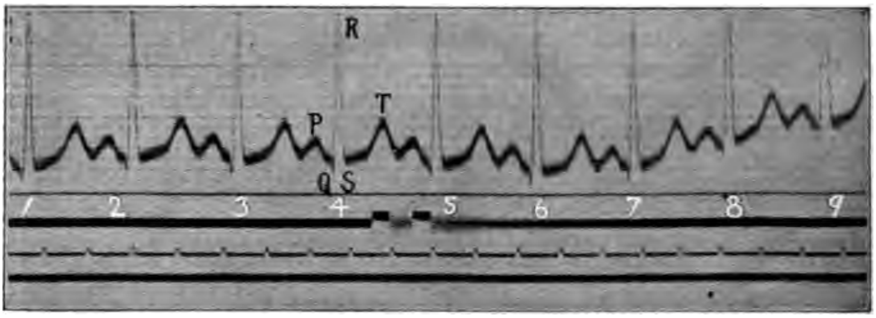

Fig 66.—Tracing taken in D2, showing hypertrophy of the right ventricle.

This instrument depends upon the "fact that a current generates a magnetic field acting at right angles to its course, which varies with the strength of the current and which may thus exert a varying attraction or repulsion upon a second magnetic field in its vicinity." The current is led off from the body by suitable electrodes and represents the difference of potential between the skin underlying the electrodes. It is generated by (a) the permanent difference of potential between these two different areas known as the current of rest and (b) the oscillations caused by the action current of the heart itself. The current of rest is neutralized by a battery current equal to it but opposite in direction. This being accomplished the only current acting upon the galvanometer is that due to the contraction of muscles. The patient being completely at rest the tonic contractions of the skeletal muscles exert an insignificant influence; contraction of the muscles of the extremities and of the respiratory muscles upon deep inspiration exerts a distinct effect, which, however, is slower than that produced

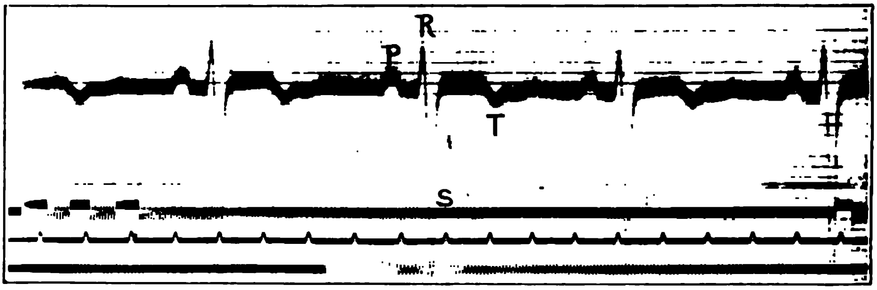

Fig. 67.—Tracing taken in D3, showing hypertrophy of the left ventricle.

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[1] For a full description of the instrument and methods and an analysis of the electrocardiogram, see Hirschfelder, Interstate Medical Journal, vol. xviii, No. 6. There is already a large literature upon this subject.

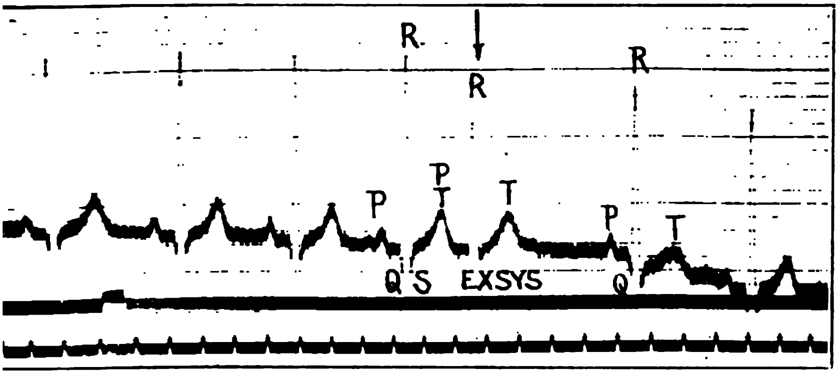
ıtractions of the myocardium. The action current, therefore, is that ꞉ed by cardiac contractions.

he conventional tracings in man are made with the electrodes in three ıns:

ıt (D 1) Lead or Derivation—Right hand and left hand.
l (D 2) Lead or Derivation—Right hand and left foot.
l (D 3) Lead or Derivation—Left hand and left foot.

ecial purposes other derivations are occasionally used.

A curve representing the electrical variations due to the heart-beat vn as an electrocardiogram and that of a normal heart-beat is shown . 409. The first part of the curve is represented by a small wave



ı.—Tracing taken in D1, showing an auricular extrasystole arising near the superior vena cava. EXSYS, extrasystole.

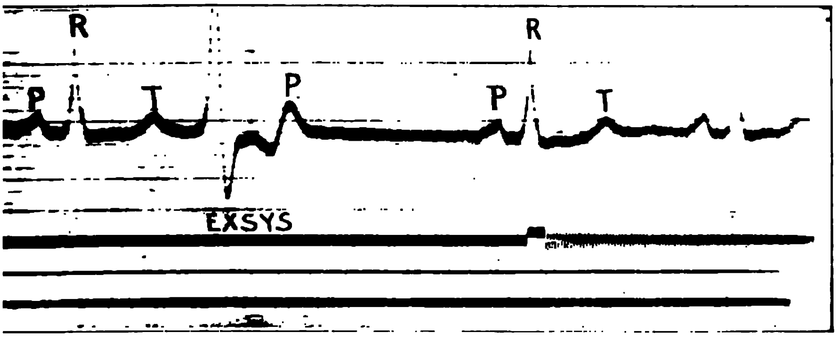


Fig 686b.—Tracing taken in D1, showing an extrasystole arising in the right ventricle.

ı Einthoven designates by P (presystolic wave) equivalent to a variof $\frac{2-4}{10{,}000}$ volt, coincident with the onset of auricular systole. This lowed by a small depression, Q, below the base-line just before the ·ical wave of the ventricle. The ventricular wave (R) is the largest of the curve and represents about $\frac{3-4}{1{,}000}$ volts. It sets in and subwithin .02 second, and then falls below the base-line to form a depres-S, just before the mechanical contraction of the left ventricle sets in. is followed by an instant during which the curve remains near the line, and then it gradually ascends to form a wave, T, lasting about ·ond in midsystole. There are no waves at the end of systole or in the diastolic period until the beginning of auricular contraction." (Hirschfelder, to whom the author desires to express his thanks for permission to reproduce the accompanying electrocardiograms, made by Dr. G. S. Bond.)

A number of similar instruments have been devised, among the more important of which are the sphygmocardiograph of Jaquet and the sphygmotonograph of Uskoff. That precise graphic methods have added much that is new and important to our knowledge of the mechanism of the heart in recent years is known to every student of medicine; but that the practitioner of general medicine should feel obliged to acquire the manipulative skill to make accurate records of the movements of the chambers of the heart and the experience necessary to properly interpret them when made cannot be affirmed. Most of the derangements of the heart met with in practice can be recognized by the direct application of the ordinary means of diagnosis.[1]

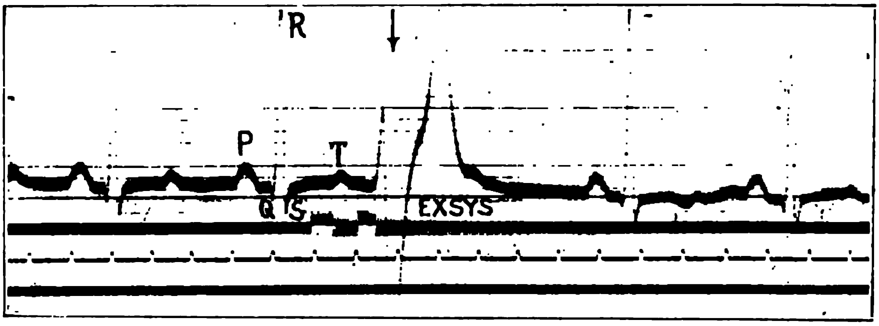


FIG. 68c.—Tracing taken in D2, showing an extrasystole arising on the left ventricle.

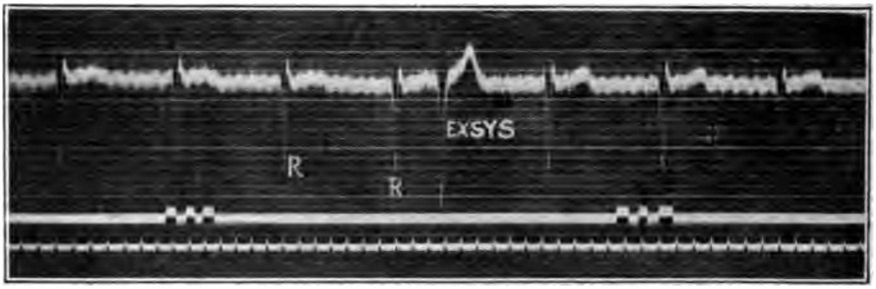


FIG. 68d.—Tracing taken in D3, showing auricular fibrillation, absolute arrhythmia, and hypertrophy of the left ventricle; also one extrasystole arising in the left ventricle.

Lewis has shown that the following disorders of the cardiac mechanism can be generally recognized and identified by careful and painstaking bedside work without the use of recording instruments: 1. *Sinus arrhythmia;* 2. *Heart-block;* 3. *Premature contractions; extrasystoles;* 4. *Simple paroxysmal tachycardia*—pulse frequency 110 to 200 per minute, usually with abrupt onset and offset; 5. *Auricular flutter*—auricular beats varying in rate from 200 to 350 per minute; 6. *Auricular fibrillation*—rapid and haphazard impulses arising in the auricle, giving rise to gross irregularity in the action of the ventricle; 7. *Pulsus alternans.* In the last, however, instrumental means are necessary to a diagnosis in a majority of the cases.

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[1] Consult "Clinical Disorders of the Heart Beat," Thomas Lewis, 2d Ed., London, 1914.

The Sphygmomanometer.—Instruments for measuring the blood-pressure in the arteries not only render it possible to determine the blood-pressure with a reasonable approximation to accuracy, due allowance being made for variations inherent to the instrument employed, but they also reveal changes in pressure not recognizable by palpation. The mechanical principle involved in the instruments thus far devised for clinical use consists in the transference of the blood-pressure within limits nearly constant as regards the apparatus to a properly graduated mercurial manometer. The essential parts comprise a compressing armlet having a breadth of not less than 8 cm., connections of tubing which is practically non-distensible, an inflating apparatus, and the manometer. For practical purposes such an instrument must be capable of measuring both systolic and diastolic pressures; its application must be simple and the results obtainable within a period not exceeding three minutes; it must be not too delicate for ordinary clinical use and sufficiently compact to be readily transported by hand.

The method consists in the close application of the armlet to the arm at the level of the heart, the patient being either in the sitting or the recumbent posture; the application of the finger to the radial pulse, the patient's arm being completely relaxed and the pressure being steadily raised by the other hand by means of the inflating apparatus. When the pulse can no longer be felt, the pressure is gradually released until it is again perceptible. This manœuvre is repeated as a control observation. The height of the mercury at which the pulse is again felt indicates the systolic blood-pressure. As the pressure is gradually released the lowest point at which the maximum oscillation of the mercury occurs indicates the diastolic pressure.

TECHNIC.—"It is wise to form a method by which all pressures are taken. This diminishes the time required and eliminates error. The muscles of the arm should always be relaxed; hence, if the patient is in a sitting posture, the elbow and forearm should be supported, as muscular contractions show themselves on the mercury column. Preferably, the pressures should be taken with the patient recumbent. For most observers it is easier to take the pressure from the left arm, as the necessary manipulations of the manometer are more easily done with the right hand. In nearly all cases the first estimation will be found 10 to 20 millimetres higher than subsequent estimations. This is probably due to excitement arising from fear that the examination will cause pain. Several estimations should be made until the level normal to the individual is obtained. In cases with a very rapid pulse-rate the diastolic pressure is hard to determine because of the inertia of the mercury. Repeated observations may be necessary. With a very slow, strong pulse the oscillations may be so large that it is hard to distinguish the largest ones. In cases showing threatened circulatory failure, especially in cases of high pressure, it will be found almost impossible to get a clear-cut high or low pressure. That is, in spite of repeated estimations, the high pressures will vary from 5 to 15 millimetres. These cases may, at times, show a condition in which an occasional beat comes through at a much higher level than that at which all the beats can be detected. Often this is due to the action of respiration. This should be noted in the estimation thus: High pressure, occasional beat at 170; all other beats at 155. Diastolic level with increasing pressure. Where the diastolic level is hard to obtain, it is of help to get the greatest oscillation with increasing as well as with decreasing pressure."

The modification of the Riva-Rocci sphygmomanometer by Beall and Mason is portable and compact. The manometer tube is in two sections, connected by a rubber cuff, and the scale is hinged at 195 mm.

Janeway's[1] studies show that the upper limit of normal blood-pressure in young adults is about 145 mm. and the lower limit about 80 mm. In the great majority of young males the upper limit is 100 mm. to 130 mm. In females it is about 10 mm. lower. In infants the pressures are lower; after fifty they are higher. Blood-pressures above 160 or 180 mm. are almost always associated with disease. Janeway places normal diastolic pressure at 25 to 40 mm. below systolic pressure in the same individual, that is to say, between 65 and 110 mm. In the aged a difference of 50 mm. is common. A difference of less than 20 mm. between the systolic and diastolic pressure indicates an abnormally small pulse; a difference of more than 50 mm. an abnormally large pulse.

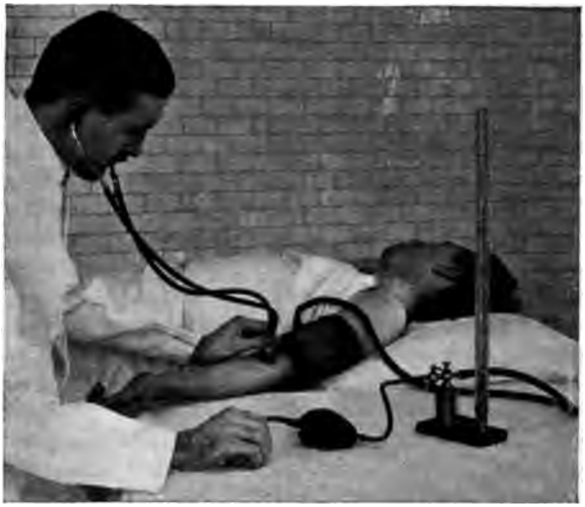

FIG. 69.—Stanton's sphygmomanometer—Auscultatory method.

Pachon's Sphygmometric Oscillometer.—This instrument, constructed on the aneroid principle, is much more convenient and sensitive and fully as accurate as the mercurial devices. In ordinary practice the cuff is applied at the wrist. The details of the technic accompany each instrument.

Auscultatory Method.—With the mercurial manometer instruments and the ordinary cuff applied to the arm the auscultatory method is much more satisfactory than that by palpation and is to be preferred for its simplicity and accuracy. The bell of a binaural stethoscope is applied over the artery below the cuff without pressure and the air gradually released. Goodman and Howell have called attention to the remarkable cycle of auditory phenomena which correspond to the degrees of pressure as shown by the manometer and they have divided them into five distinct phases. When the pressure is normal—systolic 130 mm. and diastolic 85 mm.—these phases are well defined and bear a definite relation to the differences between the extremes of pressure. They are as follows:—

1. A loud, clear, snapping sound ascribed to sudden distention of the artery by the inrushing blood. This persists from the level of systolic pressure for 14 mm.

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[1] Theodore Janeway, the Clinical Study of Blood-pressure. New York, 1904.

2. A series of murmurs ascribed to "whirlpool eddies" (fluid veins?) produced at the level of constriction of the vessel by the cuff. This phase corresponds to a drop of 20 mm.

3. These murmurs suddenly disappear and a sound which resembles the first phase is heard and is the sign of re-established blood flow in the artery. It lasts during a fall of 5 mm.

4. The sound becomes duller during a further fall of 6 millimetres—a transitional phase, and finally—

5. All sounds cease. This phenomenon marks the diastolic pressure. Absence of silence at this period points to aortic insufficiency. These observers believe that variation in sequence relation of these phases, tonal arrhythmias and irregularities in maximal and minimal pressures are of diagnostic importance, especially in the cardiac diseases.

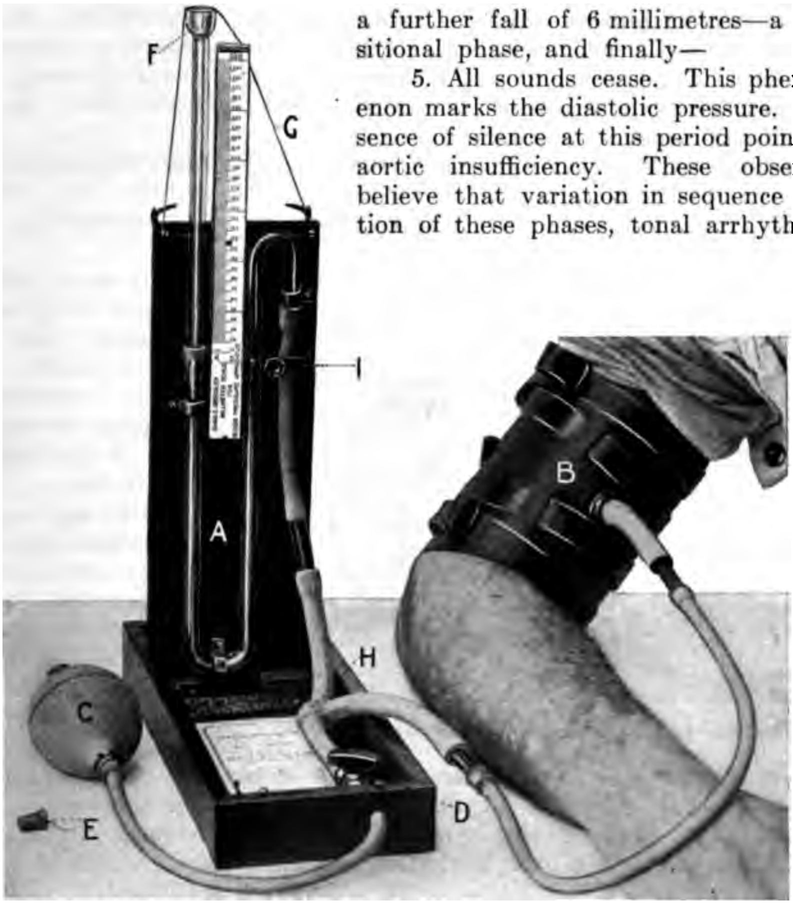

FIG. 70.—Janeway's sphygmomanometer. A, manometer; B, compressing armlet; C, inflator.

The Blood-pressure in Diseases. High Blood-pressure—Hypertension.—The highest recorded arterial pressures have occurred in acute compression of the brain, such as is caused by intracranial hemorrhage or fracture of the base of the skull. A permanently high blood-pressure accompanies persistence in the peripheral resistance with hypertrophy of the left ventricle, in arteriosclerosis and renal disease.

Low Blood-pressure—Hypotension.—This condition is present in wasting diseases and cachectic states, various infections and toxæmias, especially when severe, profuse hemorrhage, collapse and shock, and terminal states—agonal hypotension.

Nephritis.—Permanent high pressure is a conspicuous phenomenon in chronic interstitial nephritis. Systolic pressures of 200 mm. and more are common. Diastolic pressures are usually 60 to 80 mm. lower. The facts have great value in diagnosis. There are cases of interstitial nephritis in which high arterial pressure as shown by the sphygmomanometer does not occur. They are those with associated severe wasting diseases, those in which there is late cardiac insufficiency, and those that have reached the terminal stages of the disease.

In chronic parenchymatous nephritis high tension also occurs, but is by no means so constant as in the interstitial form. In amyloid disease blood-pressure is inconstant, sometimes high, sometimes subnormal.

Uræmia.—The symptoms of this condition, especially in its chronic form, are associated with increased blood-pressure and become more marked as the tension rises, less marked as it falls. Persistent lower tension has followed improvement under treatment. A gradual fall has preceded death.

Arteriosclerosis.—When the larger superficial arteries only are involved the blood-pressure is not markedly affected. Arteriosclerotic processes generally involving the smaller vessels are accompanied by increased blood-pressure, the systolic pressure being increased much more than the diastolic.

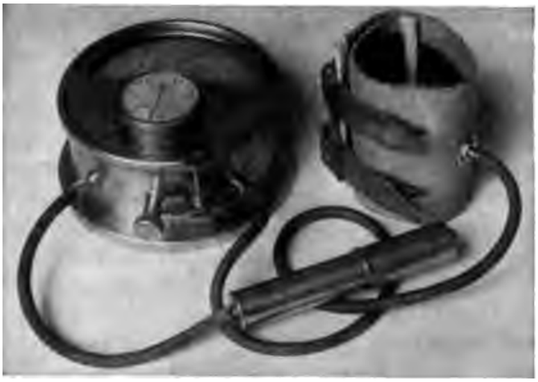

Fig. 71.—Pachon's sphygmometric oscillometer.

Apoplexy.—As a general rule patients who become hemiplegic either in consequence of thrombosis or hemorrhage have previously, if examined, manifested sclerosis of the peripheral vessels with elevation of blood-pressure.

Diseases of the Heart.—In primary, uncomplicated cardiac insufficiency from myocardial changes high normal pressures appear to be the rule. When the cardiac insufficiency is due to failing compensatory hypertrophy in arteriosclerosis and renal disease, the blood-pressure is high. As the myocardium becomes feebler the arterial tension falls.

Valvular Disease.—In aortic insufficiency the sphygmomanometer, to use the words of Janeway, "gives a numerical value to the well-known pulsus celer, which expresses perfectly the mechanical effect of the lesion in the systemic arterial circulation." The systolic pressures are high, the diastolic pressures low. In combined aortic insufficiency and stenosis the blood-pressure determination is of value in indicating the preponderating lesion, a high degree of stenosis being accompanied by a proportionately lower systolic pressure. In associated aortic and mitral insufficiency the degree of the latter defect may be estimated by the systolic as compared with the diastolic blood-pressure. In disease of the aortic valves the systolic pressure is frequently variable in the absence of obvious cause, while the diastolic pressure is more constant. Sphygmomanometric measurements are of less value in other forms of valvular disease.

Angina Pectoris.—Hypertension is an important condition in this syndrome. It is not, however, constant.

The Acute Infectious Febrile Diseases.—The type of this group, namely, enteric fever, shows with great constancy low pressure. Systematic observations at regular and frequent intervals have shown that hypotension is first apparent toward the end of the first or early in the second week and increases as the attack goes on. The daily oscillations are not significant. Crile's statistics, quoted by Janeway, are very suggestive. The mean pressure by weeks in all cases was, first week, 115 mm.; second week, 106 mm.; third week, 102 mm.; fourth week, 96 mm.; and fifth week, 98 mm. A gradually progressive fall indicates increasing failure of vasomotor tonus; a sudden fall, actual collapse or hemorrhage. A sharp rise in pressure attends the occurrence of perforation. If the observations hitherto recorded should be confirmed by further clinical studies, the sphygmomanometer will prove of great value in the differential diagnosis between collapse from hemorrhage or other cause in enteric fever and intestinal perforation. Continuous records are necessary. In the terminal stage of the consecutive peritonitis hypotension becomes extreme—agonal fall of pressure. Pneumonia stands alone among the complications of enteric fever in occasionally causing hypertension. The favorable influence of treatment by systematic cold bathing is manifest in a rise of pressure.

Pneumonia.—Uniform tendencies in blood-pressure have not been observed. Subnormal pressures are common; in severe cases the rule. A rapid fall may precede collapse or the fatal issue. When arterial pressure expressed in millimetres of mercury does not fall below the pulse-rate expressed in beats per minute, the fact may be taken as of excellent augury, while the converse is equally true (Gibson).

Chronic Diseases.—Phthisis in its advanced stages and Addison's disease give low pressures. In the early stages of syphilis when there is fever and the condition is analogous to an acute infectious process, there is hypotension. Diabetes is apparently without direct influence upon the blood-pressure. When associated with arteriosclerosis or chronic renal disease it may show hypertension, and in advanced cases hypotension is common in consequence of emaciation and cardiac insufficiency. The secondary anæmias are attended by low blood-pressures; derangements of pressure in chlorosis are neither marked nor characteristic. Chronic bronchitis, emphysema and asthma are frequently attended with high arterial tension. Pleural effusions show hypertension which falls upon aspiration.

Diseases of the Nervous System.—In locomotor ataxia the lightning pains are attended by a fall in blood-pressure; in the gastric crises the pressure is greatly increased. Arteriosclerosis of the cerebral vessels may exist without similar changes in the general vascular system Blood-pressure estimations are therefore without value as indicating the existence of intracranial vascular lesions. When there is reason to suspect their presence, increased arterial pressure due to cardiac, vascular, or renal causes affords important data for prognosis and treatment. With high pressure there is danger of hemorrhage, with low pressure danger of thrombosis. Cerebral hemorrhage is attended by marked hypertension which continues to rise as the hemorrhage increases, and remains stationary or falls when the hemorrhage ceases. In uræmic coma the pressure is also greatly increased.

In epilepsy, owing to the difficulty of making observations during the attack, there is some uncertainty. During the attack there is said to be a sudden rise in the blood-pressure, followed by a rapid fall to normal as the paroxysm ceases. In coma following an attack of general convulsions the fact that in epilepsy the blood-pressure falls while in uræmia it remains high is of diagnostic importance. In tic douloureux there is a rise of pressure during the pain proportionate to the intensity of the attack. Insomnia may be associated with increased tension on the one hand or normal or diminished tension on the other. In the former condition the pressure falls during sleep. In hysteria and neurasthenia the pressures are variable. Some observers have observed high pressures in neurotic and excitable persons, but this condition is not constant.

MENTAL DISEASES.—In melancholia the pressure is abnormally high and shows rises and falls corresponding to the intensity of the mental symptoms. In mania, on the other hand, the pressure tends to subnormal levels.

PERCUSSION.

Percussion in physical diagnosis is the art of striking or tapping upon the surface of the body in such a manner as to call forth sounds, from the nature of which conclusions are drawn as to the structure of the underlying parts.

This art was first described and systematically employed in the latter part of the eighteenth century by Auenbrugger, a physician of Gratz, who published his observations in a little book entitled Inventum Novum. The subject was widely brought to the attention of the profession by Corvisart in the beginning of the following century.

The practice of this method demands nice training both of the hands and ear in order to secure its best results. Careless and inexact methods yield not only unsatisfactory but also positively misleading results. It is especially true of percussion that they find it most useful who most clearly realize its limitations as an art in diagnosis. Unfortunately too many practitioners, otherwise well trained, fail to acquire proficiency in percussion and equally fail to appreciate its limitations.

Neither percussion nor auscultation requires the possession of much technical knowledge of acoustics nor a cultivated musical ear. It is, however, necessary to be able to discriminate differences in the character, intensity, and pitch of sounds.

The Theory of Percussion.—Reduced to its simplest terms the theory of percussion depends upon the differences in the vibrations produced by blows delivered upon structures which do not and those which do contain air, and in the latter case upon differences in the mechanical arrangement under which the air is present. There is nothing *a priori* in the matter. Our whole knowledge in regard to the signs elicited is the result of observation and experience. It has been found that direct percussion, that is, percussion without the intervention of a finger or other form of pleximeter, practised upon the thigh, which does not contain air, produces a minimum of sound which has a peculiar quality, technically described as dull. The interposition of a pleximeter increases the intensity of the sound and slightly alters its other acoustic properties. From this we infer that in percussion the vibrations of the pleximeter itself constitute a certain factor in the general result. It has further been found that percussion over the liver and spleen, organs which do not contain air, produces a similar dull sound, but that the quality of dulness is modified according to the force with which the act is performed. Upon light percussion over the spleen or centrally over the liver the dulness is much like that of the thigh, but upon powerful percussion over these organs the dull sound is modified, the quality of resonance being added. This fact in connection with others presently to be mentioned leads us to infer that by light percussion a limited region of the wall of the body is set into vibration, but that the area is extended by forcible percussion, so that the sound produced partakes of qualities due to the sound-producing mechanism of adjacent organs or structures, and that if we desire to obtain the percussion phenomena peculiar to an organ we must content ourselves with well-defined but light percussion of the surface overlying the viscus immediately in question. Experience amply confirms this inference. It has been further established that percussion over the distended bladder or a cyst, or any considerable collection of fluid, as a serofibrinous or purulent pleural effusion, produces a dull sound, and that there are degrees in the dulness just as we find differences upon light and heavy percussion over the spleen and liver, the sign having a certain quality of resonance at some parts of the border or edge of the effusion and wholly lacking resonance over the mass or base of the effusion. The recognition of these differences led to the very proper employment of such terms as *relatively dull, dull,* and *absolutely dull* or *flat.* To return to the liver and the flatness upon light percussion and the development of some degree of resonance upon forcible percussion especially near the borders of the dull area, we have attributed the latter to the vibrations of adjacent organs. Pursuing our investigations we find that as we proceed in lines upwards the sign changes somewhat abruptly from dull to a distinctly resonant sound, having qualities hereafter to be pointed out, which, with modifications of intensity and so on, but not of quality, is everywhere present over the chest where the surface or periphery of the lung comes into contact with the wall. For this reason the percussion sound elicited over the chest, and having the peculiar resonant quality spoken of, is known as pulmonary resonance, or briefly and technically as *clear.* Again, when we extend our percussion in lines proceeding downwards from the liver, we pass, under normal circumstances, quite abruptly, about the margin of the ribs, to a region which yields upon percussion a note of high resonance having likewise peculiar qualities of its own, which, because of its being produced by a mechanism remotely analogous to that of a drum, is called *tympanitic.*

A very important fact in connection with these three fundamental qualities of the signs elicited upon percussion, namely, *dulness, clearness,* and *tympany,* is this, that they are constantly related to and dependent upon the absence or presence of air in the examined structures and upon the mode of arrangement of the air when it is present. The constant correspondence between the clinical and post-mortem percussion signs and the post-mortem conditions justifies us in formulating the following dicta: Upon percussion:

1. *Airless viscera and hollow viscera distended with fluid yield* **dulness, flatness.**
2. *The normal lungs contained in the chest under conditions of normal tension yield a* **clear note.**
3. *Air contained in hollow viscera, as the intestines, the walls of which are not tense, yield* **tympanitic resonance.**

These physical signs—namely, clearness, dulness, and tympany—are normal. The percussion sound clearness as such is always normal. It cannot be elicited anywhere save over the chest, and there is no condition of structures other than the lungs by which the physical arrangement essential to its production can be brought about. With dulness and tympany the case is different. The modifications or absence of clear or pulmonary resonance in regions normally occupied by the lungs constitute morbid physical signs. Dulness in regions normally clear or tympanitic and the extension of dulness beyond the limits of airless viscera constitute morbid physical signs, and this statement is also true of the presence of tympany in regions in which the physical conditions essential to its production do not normally exist.

The foregoing facts also warrant the following statements: Upon percussion:

1. *There is no difference in the physical signs by which a distinction can be made between an airless viscus and a collection of fluid.*
2. *The signs do not enable us to determine the line of contact between two airless viscera or an airless viscus and a collection of fluid, or between collections of fluid separated by a membrane.*

Percussion is the application of an every-day art to diagnosis in medicine. The woodsman taps with his axe upon the trunk of a tree to learn whether or not it is hollow, the gauger upon the cask with his mallet to find the level of the wine, and the carpenter with his hammer upon the plastered wall to fix the position of a stud into which he can drive his nail.

The Technic of Percussion.—The patient may be examined in the recumbent, sitting, or erect posture. The outer clothing should be removed. The air contained in thick garments or in several layers of clothing seriously modifies the results of percussion. A single under-garment or a towel is preferable to the bare skin. The limbs should be symmetrically disposed and the muscles relaxed. Errors may arise from forcible percussion when the patient is resting upon a feather bed or very elastic mattress. In general terms much display of energy on the part of the physician is to be avoided. It not only yields misleading results but it also alarms and may even hurt the patient.

Two methods are employed, immediate or direct, and mediate or indirect percussion.

Immediate or Direct Percussion.—The blow is struck directly upon the surface with the palm of the slightly flexed hand, or upon the clavicles or sternum with the tip of the second or third finger, or upon the abdomen with the dorsal surface—nail—of the second finger flicked off from the thumb as one flicks a crumb. The first two of these methods were originally employed. The last is a modern refinement.

With the Palm of the Hand.—The whole hand slightly flexed or the palmar surface of the fingers held closely together may be employed. The blow is delivered chiefly from the wrist, very slightly from the elbow, care being taken to avoid too much force and the over-production of noise. This method is available for a rapid preliminary survey and class demonstration of gross differences between the sides of the chest, or the upper and lower part of one side, especially posteriorly. It cannot often be employed satisfactorily in the examination of the abdomen. The objections to it are that it demands too much force and that the vibrations caused are too extensive. It lacks the nicety of good clinical work.

Direct finger percussion over the clavicles and sternum is often practised, but is here mentioned only to condemn it as mostly inexact, often misleading, and at best yielding results obtained much more satisfactorily by other methods. The results are unsatisfactory because of the elasticity and extensive vibrations of long and flat bones. The resonance produced is that of an elongated or very large pleximeter—so-called bone or osteal resonance, well illustrated upon percussion, in the same manner, of the head with the finger-tip. As there is no intracranial air, it is evident that the resonance is due to the vibrations communicated by the bone to the external air.

Direct Percussion or Finger-flicking. — In this procedure the skin should be bared. Very exact and satisfactory results may be obtained, especially in the examination of circumscribed regions in a thin-walled abdomen. It is by far the most satisfactory method of mapping out the limits of the splenic dulness.

Mediate or Indirect Percussion — Pleximetry. — The blow is delivered not directly upon the surface of the body but upon an interposed plate or disk of ivory or hard rubber—a pleximeter, literally, measurer of the blow. This instrument should be quite flat with rounded edges, about an inch and three-quarters in length and five-eighths of an inch in width, so that it may be closely applied to the surface in the intercostal spaces. There should be at each end a little flange or ear by which it is held in position. The percussing instrument or hammer is called a plexor, and consists of a suitable head of soft rubber, or metal tipped with soft rubber, and a light, stiff handle. The plexor of Wintrich has a handle or shaft nearly corresponding in length to a human hand from the wrist-joint to the first phalangeal joint and a head corresponding in length from the last named joint to the tips of the fingers. Instrumental pleximetry is much used among European physicians.

FINGER PLEXIMETRY—FINGER PERCUSSION.—This method is almost exclusively used by American physicians. A finger of the left hand is used as the pleximeter and the right hand as the plexor, the fingers being flexed as nearly as possible at a right angle at the first phalangeal joint to form the head of the hammer, and the hand from this joint to the wrist forming its handle or shaft. The blow is delivered from the wrist and not from the elbow, and the head of the plexor, made up of the last two phalanges, must fall at a right angle upon the dorsum of the middle or terminal phalanx of the finger used as the pleximeter, the palmar surface of which is closely applied to the part examined. It is scarcely necessary to add that in left-handed persons the fingers of the right hand are used as pleximeters and the left hand becomes the plexor.

The advantages of finger percussion are (a) that the soft palmar under-surface of the pleximeter can be closely applied to the part to be examined and the danger of a thin layer of air between them wholly avoided; (b) that the finger used as a pleximeter is also a palpating finger and receives sensory impressions concerning the firmness or elasticity of the underlying part which supplement the auditory impressions caused by the vibrations occasioned by the blow; (c) that the pleximeter is composed of tissues corresponding in physical composition with the wall of the body, which it protects from the blow without the interposition of an instrument of wholly different composition, and (d) that the instruments are always at hand.

Flicking percussion may also be intermediate, a finger of the left hand being used as the pleximeter.

Superficial and Deep Percussion.—These terms indicate in general the degree of force employed. In superficial percussion the blow is light and the vibrations are limited in extent and depth. This method is essential in the study of conditions in which the percussion signs involve limited areas, as in the heart and spleen, or in which we have to deal with thin wedges of tissues yielding different signs which overlie each other, as the lung surrounding the cardiac ventricles, or dipping down between the wall of the chest and the liver or the wedge-shaped anterior lower border of the liver occasionally seen. Superficial percussion enables us to determine the nature of the structure immediately beneath the surface, and is necessary where, by reason of the thinness and elasticity of the walls, wide areas of tissue are set into vibration by the blow, as in children and emaciated persons, and in elderly persons whose costal cartilages have undergone calcification. Only superficial percussion should be employed in the examination of the chest after recent hemorrhage.

Deep percussion excites vibrations in wide areas and to a considerable depth. It is employed where the chest walls are very muscular or fat and to ascertain the dulness or resonance of the deeper structures, as the actual limits of cardiac dulness, the upper border of liver dulness, pneumonic consolidation not reaching to the periphery of the lung, or a deep-seated aneurism. In the case of a wedge-shaped anterior lower border of the liver superficial percussion enables us to demonstrate the actual limits of dulness, while deep percussion, by acting upon the underlying intestine through the thin wedge of liver, yields a most misleading tympanitic resonance.

The following directions must be carefully observed:

1. Apply the second or ring finger of the left hand accurately and firmly but without undue pressure to the surface to be examined.

2. Raise the other fingers and palm from the surface to avoid muffling the vibrations. The finger used as the pleximeter only should at the moment be in contact with the surface.

3. Deliver a quick, rebounding blow, with the tip of the percussing finger or fingers perpendicularly upon the finger used as a pleximeter, upon the middle phalanx or the terminal phalanx above the nail. The quicker the rebound of the plexor the better and more significant the result.

4. Let the blow be delivered from the wrist held perfectly loose and not from the elbow. The force must be moderate and equal at corresponding points upon the two sides of the chest; lighter where the chest wall is thin, as in lean persons and in the infraclavicular and axillary and infra-axillary regions, and more forcible in the examination of the back of a very muscular man or the mammary regions of one who is fat.

5. The attitude of the patient is important. It must be easy and unconstrained. Rigid and fixed positions are to be avoided. Muscular tension modifies percussion resonance. The arms must be symmetrically arranged. In the examination of the anterior surface of the body let them lie loosely at the sides in the recumbent posture or hang relaxed if the patient is erect; in the examination of the back the patient should bend forward and gently fold his arms. I do not like the hands to be placed each upon the opposite shoulder, since it involves an undesirable degree of muscular tension; while in the examination of the lateral regions of the chest the hands should be placed together upon the top of the head with the fingers lightly interlocked.

6. The patient must breathe gently and regularly. If changes in the percussion signs upon full held inspiration and forced expiration are to be studied—respiratory percussion—give the necessary directions.

7. Perform percussion systematically and in a routine manner, examining corresponding parts upon the two sides of the chest above and below, anteriorly, laterally, and posteriorly, comparing and noting the signs at each step in the proceeding. Comparison and contrast are essential alike in percussion and auscultation. It is often useful to apply two or more fingers widely separated to the surface and lightly percuss one after the other. In this way the border-line between dulness and clearness or tympany can be defined and demonstrated with great exactness.

8. Deliver two or three percussion strokes and then examine the corresponding point upon the opposite side in the same manner. This manœuvre may be repeated as often as is necessary. Dexterity and close attention to the sounds render a wearisome prolongation of the examination unnecessary.

9. To determine the borders of areas of dulness, clearness, or tympany percuss in parallel or radiating lines and note the points in such lines at which the quality of the percussion signs changes. Repeated light percussion is often necessary. These points may be fixed by touches with the dermatographic pencil, which when joined by a line indicate the borders of the areas studied.

Practitioners gradually develop modifications of percussion methods to suit themselves. There are many different methods, but not every one of them is right. Those not based upon a knowledge of the principles upon which this method of physical diagnosis rests and those which are slovenly or careless are positively wrong. It is like playing a musical instrument. Knowledge, aptitude, and training are essential, and there are good performers, poor performers, and those who cannot play at all.

Sources of error especially to be avoided are:

1. Failure to apply the pleximeter accurately to the surface. A thin stratum of air modifies the result and may render it wholly misleading.
2. Applying the other fingers or the palm of the hand to the surface in such a manner as to dampen the vibrations and muffle the sound.
3. Awkwardness, slowness, and the use of too much force in delivering the blow. These may all be readily avoided if the percussion stroke is from the wrist as a centre of movement rather than the elbow.
4. A false attitude on the part of the patient. Many persons on being examined assume rigid and fixed postures with the muscles in tension and the arms in constrained positions.
5. Too much clothing, and setting the air contained in the pillow, bed, or mattress into vibration by powerful percussion.
6. A want of system in conducting the examination. More errors arise from carelessness than from ignorance.

THE SIGNS ELICITED UPON PERCUSSION.

The sounds differ among themselves, as already seen, as follows:

A. **Quality:** (1) Clear, (2) dull, and (3) tympanitic.

A structure containing no air yields upon percussion a minimum of sound due to vibrations in the surrounding air and is said to be completely dull or flat.

Changes in such a structure by which it becomes air-containing or the contiguity of air-containing structures modify the percussion sign, which acquires resonance, and the dulness is no longer flat or complete, but marked, and, as it is a question of degree, moderate or merely slight or relative.

The physical signs by which these modifications of flatness are brought to pass are (a) in the direction of the conditions which underlie tympany, namely, collections of air contained in spaces the walls of which are not too tense, as, for example, the intestines; and (b) in the direction of the arrangement of the air in the lung under normal conditions which involve a certain tension as to the vesicles and as to the whole lung within the thorax—clearness.

The terms used to designate (a) modifications of dulness in the direction of tympany are slight tympany, dull tympany, moderate tympany, and tympany.

Special modifications of tympany are cracked-pot resonance and amphoric resonance.

CRACKED-POT RESONANCE: THE CRACKED-METAL SOUND. — This percussion sign requires for its development a rather forcible abrupt stroke while the mouth of the patient is open. The physical condition is an air-containing cavity communicating freely with a bronchus and sufficiently near the surface of the chest to be compressed by the sudden blow. It may also be elicited, in the absence of cavity formation, in conditions in which by reason of yielding chest walls a certain amount of air contained in the lungs or in the pleural cavity is suddenly forced by strong percussion into the large bronchi. We may encounter the cracked-pot sound therefore in infants not suffering from disease of the lungs, especially when percussion is performed at the time of the full inspiration of crying, in pleurisy, above the level of an effusion, over lung relaxed by the pressure of a large pericardial effusion, sometimes in emphysema and in certain cases of pneumothorax. This modification of tympanitic resonance may be imitated by sharply percussing the cheek while the mouth is open or by striking the two hands held together against the knee in such a way as to cause a sound like that produced when coins are rattled in the hands. For this reason the cracked-pot sound is sometimes called the money-jingle sound. This sound is in many cases only to be heard when at the moment of percussion the patient's open mouth is turned directly toward the ear of the physician or when the patient holds the bell piece of a double stethoscope just in front of his open mouth. As sudden compression of the cavity is essential the blow must be of some force and as the walls of such a cavity are not always highly resilient the peculiar phenomenon in question is often produced only upon the first two or three strokes of percussion and sufficient time must elapse for the full redistention of the cavity before the cracked-pot sound can again be heard.

AMPHORIC OR METALLIC RESONANCE. — This sign has the quality characteristic of the sound produced by percussing a large vessel with a wide mouth—*amphora*, a jar. It is a ringing tympanitic sound and denotes a cavity of considerable size with firm elastic walls which do not vibrate in unison. The pitch varies with the shape and size of the cavity and the degree of tension of its walls. A closed cavity distended with air or gas under pressure so that its walls vibrate in unison yields dulness on percussion. Amphoric resonance frequently occurs without the cracked-pot quality, but the cracked-pot sound is usually also amphoric.

(b) Modifications of dulness in the direction of clearness are slight or relative dulness, impaired resonance, clearness.

But, leaving dulness altogether out of the question, we find that changes in the physical condition in the lung by which the normal or vital tension is relaxed frequently occur. This takes place, for example, in congestion, in œdema and atelectasis from compression, in both of which the residual air is diminished, and the normal, clear or vesicular resonance acquires the tympanitic quality to a varying degree—vesiculotympanitic resonance—and as the lesions upon which vesiculotympanitic resonance depends undergo resolution this sign is gradually replaced by the normal or clear resonance again. These changes can occur only in regions in which we normally find the clear or vesicular percussion resonance, namely, over the lungs. It has been demonstrated experimentally that the extreme distention of a hollow viscus, as a bladder, with air so that its opposite walls upon percussion do not vibrate independently, but as a whole, does away with the tympanitic sound, and causes it to be replaced by dulness.

B. **Volume or Intensity.**—This acoustic property is of minor importance in percussion. It depends upon the volume of air contained in the structures examined, the elasticity of the enclosing walls and energy of their vibrations, and the force of the blow. This term has reference to the loudness or degree of sonority of percussion sounds, which may be on the one hand so great as to obscure their value or on the other so faint as to be without any significance whatever. The duration of percussion sounds usually corresponds to their volume or intensity.

C. **Pitch.**—The distinction between sounds and musical tones must be borne in mind. In percussion we have to do with the former. Nevertheless the pitch of percussion sounds is of great importance. Pitch indicates in music the relative position of notes upon the scale and depends upon the frequency of the vibrations by which tones are produced. In physical diagnosis we find that large air-containing spaces with slight or moderate tension yield percussion resonance of low pitch, while small spaces with high tension yield resonance of higher pitch, and that the vibrations of the pleximeter upon the thigh or over an airless viscus yield a sound of slight intensity and high pitch.

Percussion over the abdomen reveals great variations in the pitch of sounds having the quality of tympany, as over the stomach and large and small intestines. These variations are of some value, but cannot be relied upon in mapping out the positions of those viscera. They serve a purpose in indicating the border-line between contiguous organs, as the stomach and transverse colon and coils of intestines under different degrees of tension.

The quality of a sound is that property which enables us to recognize it whenever heard without seeing the mechanism by which it is produced, as the sound of a bell, a drum, and so on; the volume or intensity of a sound relates to the energy and the mass of the material by which it is produced, as, for example, in great and little bells, the sound of which has the same quality and may have the same pitch while differing greatly in intensity or volume; the pitch depends upon the rapidity of the vibrations by which sound is produced, as in the long strings of the piano which produce low notes, and the short strings which send forth the high notes.

THE LUNG REFLEX (Abrams).—It has been observed that local irritation of the skin of the chest as by cold or the application of mustard has been followed by the evidences of a temporary circumscribed emphysema of the underlying lung. These signs of dilatation of the air-vesicles have in some instances been confirmed by X-ray examination. Cabot has referred to this observation in explanation of the fact, well known to teachers of physical diagnosis, that the repeated demonstration of an area of moderate dulness, as, for example, in incipient tuberculosis, is followed by a modification of the percussion sign, which gradually becomes more resonant. The repeated percussion apparently acts as a local irritant. If the consolidation is dense and extensive this change cannot occur.

Respiratory Percussion.—Differences in the sound are noted upon quiet breathing and full held inspiration. The contrast between the two sides of the chest in slight consolidation, as in beginning phthisis or pleural thickening, is thus accentuated, the dulness upon the affected side remain-

; the same, while the resonance upon the sound side is increased. This thod is serviceable in determining the presence or absence of slight grees of relative dulness, especially in the infraclavicular regions in cipient pulmonary tuberculosis.

Palpatory Percussion.—As has been pointed out in the general con- deration of percussion, palpation is an essential though subordinate ctor in finger percussion, which is gradually taking the place of other ethods. Palpatory percussion is a method in which the attention is pecially directed to the resistance and elasticity of the tissues over which e percussion is performed. It consists in the combined use of palpation nd percussion in a modified form, and is applicable (a) to the determina- on of the outlines and boundaries of the solid viscera under various con- itions, but especially to the study of the deep dulness of the heart both hen the lungs are normal and when they are emphysematous; (b) to he examinations of solid organs of limited size surrounded by air-contain- g and resonant structures, as the spleen, and particularly when there is ympanitic distention of the abdomen; and (c) to the recognition of the xtent and the horizontal levels of fluid exudates in the abdominal and tho- acic cavities in different postures. The physical signs depend to a greater xtent upon the sensation of resistance imparted to the percussion finger han upon the character of the sounds perceived, though both have value. arious methods have been described. Delicacy of touch, a light stroke, nd a lingering rather than a momentary contact with the surface under xamination are essential to success in all of them.

The direct methods of palpatory percussion are:

1. THAT OF THE WRITER.—This consists in flicking the surface ith the nail of the middle finger in the manner described under the ption direct or immediate percussion. This method is painless to the atient and yields very accurate results. The nail should strike the surface ercussed flatly and linger for an instant.

2. MAGUIRE'S METHOD.—The palmar cushion of the tip of one finger employed as the plexor. The stroke is not short and quick but prolonged nd combined with a certain movement of pressure or palpation.

3. METHOD OF HEIN.—The first and middle fingers are employed, e tip of one resting upon the surface while the other, used as a plexor, elivers a light tap upon the adjacent surface, palpation and percussion eing literally performed at the same time. The fingers are alternately sed and the whole surface is gradually examined. Very accurate results ay be obtained by this method.

The Indirect Methods Are: 1. The finger used as a pleximeter is ruck lightly with the fingers of the other hand, which are slightly flexed such a manner that the blow is delivered by the pulps rather than the treme tips. The stroke is not sharp and rebounding, but prolonged d pushing, the so-called palpating stroke, and the percussing fingers main a moment upon the plexor finger before the blow is repeated.

2. That of Ebstein. A glass pleximeter 4 centimetres in length and 3 centimetres in width, with a projecting bar 1.5 centimetres in height used. This is held firmly in place while the finger held as in ordinary nger percussion delivers a gentle but pushing or pressing percussion stroke

upon the flat upper surface of the bar. The pleximeter devised by Sansom consists of a slender rod of square section having at one end attached at right angles a thin plate and at the other end a similar plate parallel to the first. The measurements are about the same as those of the glass pleximeter of Ebstein, but all the parts are made of hard rubber. In use the larger plate is applied to the surface of the chest and held in position by the tips of two fingers, one on each side of the rod. Percussion is then made upon the upper plate, the finger of the other hand being employed as a plexor. Greater attention is paid to the vibrations perceived by the fingers than to the sound. This special pleximeter enables the observer who has acquired skill in its use to recognize slight modifications of the vibrations produced by percussion and to map out more closely than by other methods, but not absolutely, the limits of the deep dulness of the heart and the great vessels.

Auscultatory Percussion.—The binaural stethoscope is applied to the surface of an organ, as the heart, liver, stomach, etc., and held in place by an assistant or the patient himself. Using finger pleximetry with very light strokes, percussion is performed in radiating lines towards or away from the stethoscope as a centre. Direct percussion with the finger-tips may be employed in case the observer himself is obliged to use one hand to hold the stethoscope in place, or a light stroking touch or scratching of the skin will serve the purpose. A stiff brush or the handle of a large tuning-fork in vibration may be used for this purpose. The sounds are greatly intensified and changes in their quality, volume, and pitch are readily appreciated. Of especial importance are the abrupt changes that take place as the line of percussion passes over the border of the organ over which the stethoscope is placed. The points at which the change occurs being marked and these points being joined by lines, an approximate outline of the organ is obtained. The observation must be controlled and errors eliminated by percussing in segments of widening circles and by the employment of the ordinary methods of percussion. This method is much more useful in the examination of the abdominal than of the thoracic viscera. It should be acquired by every student.

Percussion Signs in the Chest.

THE EXAMINATION OF THE NORMAL CHEST BY PERCUSSION.

Pulmonary Resonance.—The sounds elicited vary in different regions. The anterior and lateral surfaces are more resonant than the posterior by reason of the greater thickness of the walls of the last. The resonance in the former is known as normal, pulmonary, or vesicular. The portion of the apex of the lung above the clavicle yields a sound which acquires the tympanitic quality—vesiculotympanitic—as the trachea is approached. Some difficulty in the application of the finger or pleximeter renders percussion less satisfactory in this region than in other parts of the chest. Over the clavicle the sound has the peculiar quality known as osteal resonance; is dull towards the scapular extremity and acquires a distinctly tympanitic quality with heightened pitch at its sternal end of the bone.

In the infraclavicular region, that is, as far downward as the fourth rib, the pulmonary or vesicular resonance is characteristic. There is usually, however, a slight disparity in the two sides, the sound of the right being somewhat less resonant, shorter in duration, and of higher pitch than the left. The recognition of this fact is of cardinal importance. From the fourth rib downwards on the right side the resonance upon strong percussion is slightly diminished, owing to the presence of the dome of the right lobe of the liver. About the sixth rib the pulmonary resonance ceases. During full inspiration the liver is pushed downwards to the extent of an inch or more and the line of dulness is depressed to a corresponding degree.

On the left side the vesicular resonance is impaired by the presence of the heart between the fourth and sixth ribs and to the left as far as the mamillary or midclavicular line. The extent of this area is diminished under any conditions in which a larger wedge of the border of the lung is interposed between the wall of the chest and the heart, as upon deep inspiration and in those who have deep chests and voluminous lungs. At the base anteriorly the clear resonance passes into the tympanitic resonance of the fundus of the stomach—Traube's semilunar space; laterally into the dulness of the spleen.

In the lateral regions, axillary and infra-axillary, percussion yields vesicular resonance modified in the direction of higher pitch and diminished intensity towards the base of the chest by the presence of the liver on the right and the spleen on the left side.

Posteriorly the sound varies markedly according to the region percussed. The greater thickness of the muscles and the presence of the scapulæ are to be considered. The resonance is everywhere diminished as compared with the anterior and lateral regions. It has the quality of clearness but is muffled and merges into dulness over the scapulæ. The percussion sound is clear over the apices but usually slightly less so on the right side than on the left; and in the interscapular regions, which are widened when the patient bends forward and folds his arms. It is also clear from the angle of the scapula on each side to the base of the chest, namely, about the level of the tenth rib, where on the right side the liver dulness begins. On the left side the clear percussion sound may be found a little lower than upon the right; while the resonance upon deep percussion is somewhat diminished on the right by the convexity of the liver and on the left to a less extent by the spleen.

In children and emaciated persons the resonance in the back is often very good and percussion yields results scarcely less satisfactory than in the front of the chest.

Normal Tympanitic Areas in the Chest.—These are at the sternal ends of the clavicles, over the manubrium sterni and at the left base anteriorly. The first and second of these regions owe their tympanitic resonance to the proximity of the trachea and main bronchi and their osteal quality to the large proportion of bone entering into the wall of the chest. In elderly persons with calcification of the costal cartilages this osteal quality is widely present and when combined with tympany as is often the case greatly impairs the value of the percussion signs.

Percussion directly over the trachea at the episternal notch and that over the larynx, *i.e.*, over the plates of the thyroid cartilage, yields amphoric resonance. The normal tympany at the left base anteriorly is due to the presence of the fundus of the stomach when distended with air and the adjacent transverse colon. The curved upper border of this space, the convexity of which corresponds with the convexity of the diaphragm, is of special diagnostic value. The degree of distention of the stomach increases this curve, which is flattened or may even become concave in large left-sided pleural effusions. Traube's semilunar space is bounded to the right by the left lobe of the liver—dull; above by the lung—clear; to the left by the spleen—dull upon light percussion and is itself tympanitic, the tympany being continuous with that of the stomach and transverse colon. It often requires nice work in percussion to map out the borderline between the clear vesicular resonance which forms the upward limit of this space and the tympanitic resonance of the space itself.

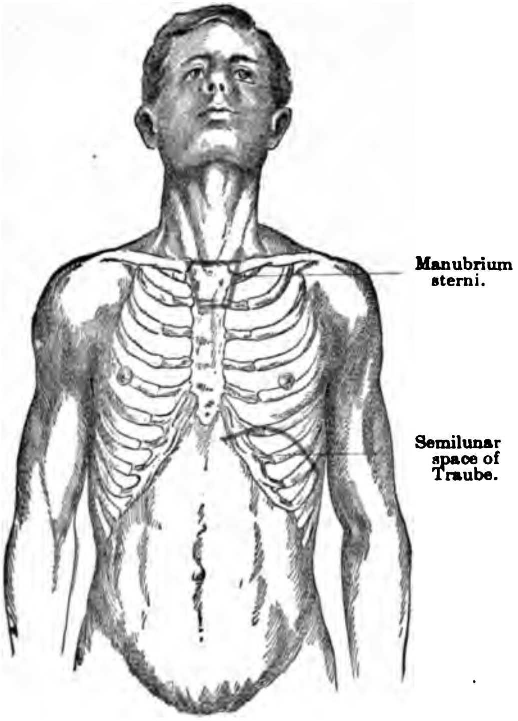

FIG. 72.—Normal tympany.

Dull Areas in the Normal Chest.—These are found to correspond to the scapulæ with their large muscular masses in the chest wall, the liver and spleen reaching up into the chest in the vault of the diaphragm and the heart within the chest itself. The scapular dulness has already been described. The liver dulness extends in the right midclavicular line from about the sixth rib to the border of the ribs and shifts downwards an inch or more on full inspiration; the dulness of the left lobe is continuous vertically with the cardiac dulness, from which it cannot be distinguished by ordinary percussion, although the border-line between them is sufficiently indicated for clinical purposes by the upper border of liver dulness on the right side and the position of the cardiac impulse on the left. In doubtful cases auscultatory percussion may be employed. The heart rests upon the central tendon of the diaphragm and the upper curvature of the liver fits into the vault of the diaphragm.

The area of the splenic dulness extends from the upper border of the ninth to the lower border of the eleventh rib and from a point slightly anterior to the midaxillary line backward towards the spine. It varies normally with the physiological changes in the size of the organ.

Cardiac Dulness.—The cardiac dulness is divided into the superficial and the deep.

SUPERFICIAL.—The superficial cardiac dulness corresponds to that part of the organ constituted by the anterior surface of the right ventricle, which uncovered by lung lies in relation with the chest wall. It begins above about the level of the fourth costal cartilage and extends to the apex, being bounded below by the dulness of the left lobe of the liver and on the right by the resonance of the anterior border of the right lung at the middle line. This irregularly quadrilateral area varies in size according to the expansion of the borders of the lungs, especially the left. It is smaller upon inspiration than upon expiration and in active individuals with voluminous lungs than in sedentary persons with small and narrow chests. It yields upon superficial percussion a flat and upon deep percussion a dull note.

DEEP.—The deep cardiac dulness corresponds to the borders of the heart itself beneath the overlapping margins of the lung and extends beyond the area of superficial dulness. Upon deep percussion over this area the note is dull, but the rounded receding surface of the heart renders a literally exact determination of its limits impracticable. Even the most skilful percussion yields only approximate results. The difficulties in determining the upper and right border of the heart by percussion are increased by the fact that the organ is covered in those regions not only by the borders of the lung but also by the sternum, which modifies to a high degree the percussion sound of the structures underlying it.

Enlargement of the heart gives rise to increase in the diameters of both these areas, the deep area of cardiac dulness being increased by the enlargement of the heart itself; the superficial area by the pushing aside of the margins of the lungs. The determination of the superficial area of cardiac dulness is a relatively easy matter, but the knowledge thus obtained relates rather to the position of the margins of the lungs than to the size of the heart; the determination of the deep area in so far as it is practicable would indicate the actual size of the heart, but the difficulties in reaching exact data are in many cases insuperable. For these reasons we cannot regard percussion as the best method of ascertaining the size of the heart. It has a value as a control method, but the position of the apex-beat, as determined by inspection, palpation, or auscultation, and the extent of the impulse, with associated clinical phenomena, constitute diagnostic criteria at once more convenient of application and far more precise.

The resonance of the normal chest is modified within narrow limits by a variety of conditions, among the more important of which are the following:

1. **Change of Posture.**—In the lateral decubitus the resonance of the lower lung is slightly less than that of the upper by reason of the greater amount of air in the latter. On exchanging the recumbent for the erect posture the pitch of the percussion sound is raised (Da Costa). If the patient turns upon the left side, the heart, under the influence of gravity, swings outwards towards the left axilla, with a corresponding change in the position of the apex and the cardiac dulness.

2. **Respiration.**—The general resonance of the chest is greater upon full held inspiration than on quiet breathing simply because of the increase

of air within its cavity. This increase of resonance may be noted on quiet respiration after great muscular exertion, which is accompanied by a temporary physiological distention of the vesicular structure of the lungs.

The increase in the volume of the lungs upon full inspiration not only augments the resonance but also extends its borders in certain directions, especially over the heart so that the superficial area of cardiac dulness is diminished, and at the base of the chest so that the liver and spleen are carried downwards with the descending diaphragm, and areas at the base, dull on expiration or quiet breathing, yield a clear note. This respiratory excursus of the lower margin of the lungs is observed posteriorly as well as anteriorly, but not to the same extent. It varies in different individuals in health just as the inspiratory expansion varies and is diminished by the presence of pleural adhesions.

3. **Gaseous Distention of the Stomach and Colon.**—This condition may displace the upper crescentic convexity of Traube's half-moon-shaped space and cause tympanitic resonance in the lower part of the left chest or impart a tympanitic quality to the vesicular resonance—vesiculotympanitic resonance. It may also to some extent displace the diaphragm upwards, thus causing the lower margins of the lungs to assume a position slightly higher than normal with a corresponding upward displacement of the limit of pulmonary resonance.

Age.—In children the lungs are relatively small and the dull areas of the heart and liver correspondingly greater. In old age the borders of the lungs are usually emphysematous, even in persons otherwise in normal condition. Hence the area of superficial cardiac dulness is encroached upon and the upper border of liver dulness is slightly lower than at earlier periods of life. Under this circumstance the vesicular resonance acquires a faintly tympanitic quality.

The Condition of the Chest Wall.—The obvious part in this respect played by great muscular development and obesity has already been spoken of. There are persons in whom percussion on account of these obstacles yields negative results. Œdema of the chest wall is also an important obstacle. Highly developed mammæ likewise interfere with the application of this method of examination; so also do the tenderness of inflammation of the chest wall and hyperæsthesia.

PERCUSSION IN DISEASE OF THE THORACIC ORGANS.

Percussion in the different regions of the normal thorax yields (1) vesicular resonance, the sign of normal lung tissue under normal intrathoracic tension; (2) diminished resonance or dulness over the scapulæ and the area of deep cardiac dulness; (3) absence of resonance or flatness over the lower ribs on the right side anteriorly; (4) vesiculotympanitic resonance towards the base of the chest anteriorly on the left; (5) tympanitic resonance over Traube's semi-space and over the manubrium and the sternal ends of the clavicles; (6) amphoric resonance over the trachea and cracked-pot resonance sometimes in the crying infant. While these sounds are normal when obtained in the particular regions of the chest above indicated, they become abnormal or morbid signs in other positions.

Thus vesicular resonance in the area of superficial cardiac dulness may indicate dextrocardia or some other form of malposition of the heart; diminished resonance or dulness in the infraclavicular or mammary regions may be significant of tuberculous infiltration or at the bases of bronchopneumonia; absence of resonance or flatness over a large area on either side which is normally clear denotes pleural effusion, a tumor, or some other airless condition; vesiculotympanitic resonance is the sign of a moderate degree of atelectasis due to compression of the lung and of emphysema; when of high degree it constitutes the percussion sign known as skodaic resonance; tympanitic resonance elsewhere than normal is the sign of a cavity, pneumothorax, or pneumopericardium; and amphoric resonance save over the trachea and the cracked-pot sound except in crying children must in all instances be looked upon as pathological conditions.

The changes which modify the normal resonance affect (a) the borders of the lungs; (b) the structure and tension of the lungs, and (c) adjacent organs. They may be general, unilateral, or local.

Changes in the Relation of the Borders of the Lung to the Wall of the Thorax.

The Apices.—The lungs normally reach about an inch and a half to two inches above the clavicles, the right apex being usually somewhat higher than the left. Normal pulmonary resonance is obtained therefore in both retroclavicular spaces. If absent in one or both and especially when replaced by dulness there is consolidation and retraction of the apex or apices. This sign is significant of tuberculous disease. fibroid phthisis, or local adhesive pleurisy. It is often stated that bulging of the retroclavicular space with tympanitic resonance occurs in emphysema. This is not always true. Owing to the skeletal changes in the thorax in emphysema of high grade there is usually retraction of the spaces immediately above and below the clavicles. Transient prominence with vesiculotympanitic resonance occurs in the acute emphysema of asthma and pertussis.

The Anterior Borders.—The resonance is marked by the osteal quality of the percussion sound over the sternum and the signs are uncertain. Below the level of the fourth costal cartilage the border of the left lung sweeps downward and to the left, and is readily made out by percussion, forming the upper and left lateral boundary of the area of superficial cardiac dulness. Below the clinical apex of the heart and between the anatomical apex and the chest wall a tongue-like projection of the anterior border of the lower lobe called the lingula gives rise to a clear percussion sound over a limited wedge-shaped space. The anterior border of the left lung is pushed aside by an hypertrophied heart or large pericardial effusion so that the area of superficial cardiac dulness is increased. An increase in diameter is, however, much more frequently due to diminution in the volume of the lung as in tuberculous or fibroid disease and consequent retraction of its borders. In substantive chronic emphysema and the acute emphysema of asthma and whooping-cough the left border of the lung is advanced and in extreme cases to such an extent as to obliterate the area of superficial cardiac dulness.

The Lower Borders.—Due allowance must be made for the changes caused by posture, exertion, age, etc. (p. 133). The borders are lowered in pathological conditions in which the volume of the lungs is increased and they occupy a position higher than normal when it is diminished.

In advanced cases of emphysema the lower border of the lung as marked by the transition to liver dulness on the right side and to tympany on the left, may reach the ninth rib and a corresponding level in the lateral and posterior regions. The respiratory excursus of the border is very limited in this disease. It is likewise much restricted by old pleural adhesions. Permanent upward displacement with restricted respiratory movement is a sign of tuberculous or fibroid shrinking, chronic bronchopneumonia, or pulmonary collapse. The lung border may be pushed up by a distended abdomen, or drawn up by its own elasticity in paralysis of the diaphragm. It is also displaced upward and rendered immovable by fluid or air in the pleural cavity—hydro-pyo-hæmo-pneumothorax.

Impaired Resonance; Dulness; Flatness.—The resonance is diminished in proportion as the amount of air is decreased in the part percussed. It is modified according to the changes in the physical structure of the spaces containing the air caused by the lesions of disease. Consolidation of the lung from exudate within its substance, compression, infarct, collapse, renders the percussion sound over the affected area less resonant in proportion to the extent of the lesion. In disseminated lesions, as those of the common forms of ordinary or tuberculous bronchopneumonia, there is usually a local compensatory emphysema which modifies the dulness. The association of heightened pitch and diminished volume with diminished pulmonary resonance must always be borne in mind. In many instances the well-trained ear will recognize a change in the pitch of the percussion sign before alteration in its quality. The sensation of increased resistance—loss of elasticity—which is experienced by the pleximeter finger in fluid exudates and dense consolidations is also to be remembered.

Impaired resonance is a sign of beginning or disseminated tubercle, bronchopneumonia, early croupous pneumonia, small effusions, thickened pleura, gangrene of limited extent, and small abscesses or tumors. Dulness is present when the lesions of the above conditions are close set or extended.

Flatness when no Air is Present.—The percussion sound is dull over the complete consolidation of croupous pneumonia involving a lobe or an entire lung because some air yet remains in the large and middle-sized bronchial tubes; it is flat over a large effusion because the lung with its compressed vesicles and with it the air-containing bronchi are pushed wholly away. The presence of circumscribed consolidations, especially when not directly beneath the chest wall, cannot be recognized by percussion. Their only sign may be a slight elevation of the pitch. Hence central pneumonias and deep-seated aneurisms are frequently overlooked. An effusion into the pleura of serum, pus, or blood which does not reach 500 to 750 cubic centimetres in volume does not often yield definite physical signs upon percussion, and a pericardial effusion of half this amount may escape detection. In pneumothorax when the bronchopulmonary fistula has closed and the air is present under a high degree of tension, the percussion note over the greater part of the affected side may be dull.

Impaired resonance over the apex or upper lobe of one lung with normal resonance elsewhere is commonly significant of tuberculosis. It may, however, be caused by an apex pneumonia or gangrene. Dense pleural thickening is also a cause of dulness in this region. Slight impairment of resonance in this region which passes away upon repeated deep inspiration or prolonged percussion may simply indicate habitual deficient respiratory expansion of the lungs.

Dulness at the base of the chest, always more pronounced and significant posteriorly, may be the sign of pneumonia, œdema, hypostatic congestion, atelectasis, or pleural effusion or thickening. Less commonly it stands for infarct, abscess, gangrene, tuberculosis, or tumor.

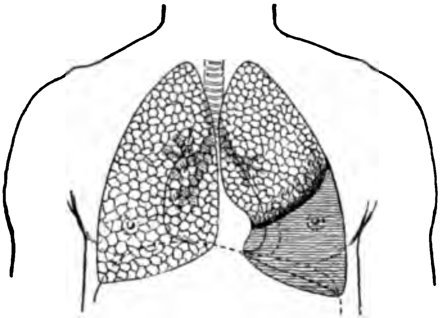

FIG. 73.—Pleural effusion, left side, showing degree of displacement of heart and of obliteration of Traube's semilunar space.

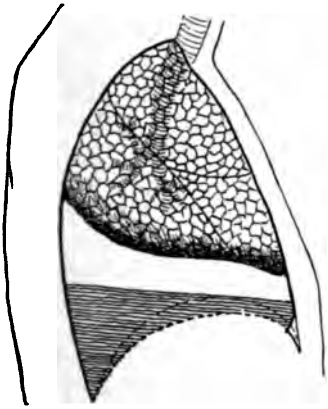

FIG. 74.—Pneumohydrothorax—erect posture.

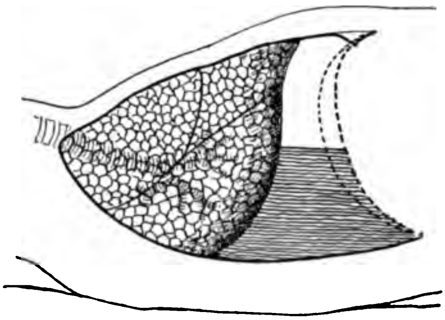

FIG. 75.—Pneumohydrothorax—dorsal decubitus.

Flattening of the convexity of Traube's semilunar space is a sign of moderate pleural effusion ; marked depression, with a concave upper line, occurs in massive effusion.

Vesiculotympanitic resonance of woodeny quality is significant of extensive fibroid changes in the lung.

Dulness at one or the other base, the upper line shifting quickly upon change in posture, is characteristic of pneumohydrothorax. The upper line of small pleural effusions shifts much more slowly and that of large effusions scarcely at all save in prolonged and decided change of posture. It is to be remembered that a pleural effusion which develops insidiously while the patient is up and about occupies the lower part of the chest and causes dulness at the base anteriorly, while one that accumulates in a bed-

Enlargement of the boundaries of precordial dulness to the left, right, and upwards may indicate the presence of a pericardial effusion. The dulness is marked and its outline is pyramidal or pear-shaped, the smaller end being directed upward. The increase in dulness may be first observed in the angle formed by the right border of the cardiac and the upper border of the liver dulness, which becomes at first rounded and then obliterated. The dulness may extend to the second interspace or higher and is sharply defined at its borders. In pericardial effusions of considerable size the compressed left lung yields a vesiculotympanitic or tympanitic resonance —dull tympany; skodaic resonance. The apex-beat may be felt or located by the intensity of the first sound within the borders of dulness. Percussion alone will not always enable us to make a differential diagnosis between a moderate pericardial effusion and extreme dilatation of the heart.

Under normal conditions the impairment of resonance due to the presence of the aorta and pulmonary artery does not extend beyond the manubrium on either side. When it can be made out upon the right side in the first and second interspaces, or in the notch of the sternum, it is usually a sign of dilatation of the aorta or of aneurism of the ascending or transverse portion of the arch. Sometimes it indicates the presence of a mediastinal tumor.

Decrease in the area of cardiac dulness is mostly caused by pulmonary emphysema, by which the heart is covered more completely by the lung and displaced towards the middle of the thorax. The heart like other muscle masses undergoes atrophic changes in acute and chronic wasting diseases, as enteric fever and consumption, with a corresponding diminution in the area of cardiac dulness. This area is diminished in extent, altered in outline, and in extreme cases rendered wholly unrecognizable by percussion in left pneumothorax, pneumopericardium, and emphysema of the mediastinum, such as follows trauma and occasionally occurs in fatal cases of pertussis or after tracheotomy. Extreme meteorism and great distention of the stomach by gas may cause like effects. Under these conditions the modified pulmonary resonance of the deep area and the dulness of the superficial area are replaced by a tympanitic note.

Dislocation of the Cardiac Dulness.—The heart is a very movable organ. The shifting which the apex undergoes upon changes of the posture of the body has already been described. The heart is displaced upwards by great meteorism, ascites, a massive abdominal tumor, or pregnancy; to the left by pleural effusion, pneumothorax, or tumor on the right side; to the right by similar conditions upon the left side. Great enlargement of the right lobe of the liver likewise displaces the heart to the left. Contraction of the lung with adherent pleurisy displaces the heart by traction toward the affected side, as in old pleural effusions that have undergone resorption or been relieved by operation, and in cirrhosis of the lung. Aneurismal or cancerous tumors and diaphragmatic hernia are among the rarer causes of displacement of the heart. Practically speaking, displacements of the heart are the result of diseases of the pleura or lungs. The greater part of the heart and its apex may lie to the right of the median line with or without general transposition of the viscera. Under all these conditions except the last, there are such modifications of the percussion signs relating to the heart as to render that method of physical diagnosis still more unsatisfactory and frequently wholly useless in determining the boundaries of the cardiac dulness, valuable as it remains in the diagnosis of the primary disease. The position of the apex as located by palpation and auscultation constitutes the most reliable evidence of the region occupied by the displaced heart. The diagnosis of congenital displacement of the heart should in no instance be made until all other causes capable of producing such displacement have been shown to be absent.

Percussion in the Examination of the Abdomen.

This method is far less valuable than palpation in the diagnosis of abdominal diseases. It has, however, much usefulness for direct examination and is particularly important in controlling the results obtained by the other methods of physical examination.

The Technic.—The general directions are the same as in the technic of palpation. Direct percussion except flicking percussion cannot be employed, owing to the sensitiveness of the surface and the elasticity of the walls of the abdomen. As in palpation the patient must be examined in various postures, and it is frequently necessary to distend the stomach or colon with air or water. Auscultatory percussion is of service in determining the boundary lines between contiguous viscera, whether they be solid or air containing.

PERCUSSION OF THE ABDOMEN IN HEALTH.

The upper limits of hepatic and splenic dulness are determined by vesicular resonance. With this exception the signs relate to dulness and tympanitic resonance and their modifications.

The normal dull areas in the abdomen are:

(a) **Hepatic.**—The upper border of dulness begins about the level of the sixth rib in the midclavicular line. Its lower border nearly corresponds with the arch of the ribs. This area of dulness shifts with the respiratory movements about two fingers' breadth on quiet and slightly more upon deep breathing. The dulness extends upon corresponding lines in the epigastric zone and its respiratory excursus is less at the back than in front. The dulness of the left lobe extends to the left of the median line and is continuous vertically with that of the heart.

(b) **Splenic.**—The dull area of the spleen occupies the space between the ninth and eleventh ribs, its anterior border being slightly in advance of the midaxillary line. Its respiratory excursus is slightly less than that of the liver. The observation must in all cases be confirmed by palpation.

The above are constant in health.

Dull areas that are not constant also occur under physiological conditions. These are:

(c) **The Distended Bladder.**—The dull area is situated in the suprapubic region in the median line. It may extend half-way to the umbilicus. It is oval and symmetrical in outline and disappears upon micturition or catheterization.

present at the apex. Single small cavities even when they are superficial, and deeply seated cavities even when of moderate size, do not yield a tympanitic percussion sound.

8. Subdiaphragmatic tympanitic resonance when the distention is extreme may be elicited by percussion in the anterior axillary line on the left side as high as the third interspace.

Amphoric Resonance. This is the sign of a cavity of large size or, when very extensive, of pneumothorax. The cracked-pot sound is usually the sign of a cavity of some size with compressible walls and communicating freely with a bronchus. The essential physical requirement is that the walls should be freely compressible so that there may be a free outrush of air at the moment of the percussion stroke.

The following percussion phenomena described in the text-books are of greater clinical interest than practical value. They are very rarely brought out in a manner that amounts to a demonstration.

1. WINTRICH'S SIGN. The tympanitic percussion sound is higher in pitch upon opening the mouth and lower when it is closed. The patient should open his mouth, protrude his tongue, and breathe quietly. This phenomenon is occasionally, but by no means in the majority of instances, observed in large cavities of the lung or pneumothorax with wide communication with a bronchus.

2. INTERRUPTED WINTRICH'S SIGN.—If the foregoing sign is exclusively present in the sitting posture, it is evidence of a cavity containing fluid which in one posture occludes and in the other leaves open the communication with the bronchus. Under these circumstances gurgling or the râle of cavities is usually present.

3. GERHARDT'S SIGN. The pitch changes with change of posture, usually becoming higher in the erect posture but scarcely ever becoming higher in the recumbent position. The alterations in pitch are attributed to the changes in the shape of the cavity caused by the gravitation of the fluid to its lowest part.

4. FRIEDREICH'S SIGN. The tympanitic resonance over a cavity communicating with a bronchus is higher in pitch upon inspiration than upon expiration. The change in pitch is always slight and often too slight to be of value in diagnosis. The inspiratory rise is attributed to the widely open glottis and the increased tension of the air in the cavity.

5. BIERMER'S SIGN. In pneumohydrothorax the tympanitic percussion note is lower in the recumbent than the erect posture, the change being due to alterations in the shape and relative diameters of the air space caused by the gravitation of the fluid. The underlying principle in [illegible]

Coin Percussion. Coin Test. Anvil Test. [illegible] is performed upon [illegible] diametrically opposite [illegible] laid flat upon [illegible] striking with its [illegible] pieces of silver [illegible] sign consists [illegible] may be [illegible] This sign [illegible]

PERCUSSION IN DISEASE OF THE HEART.

The normal superficial and deep cardiac dulness and the method of determining them have been discussed in a foregoing section. By this method of physical diagnosis we ascertain approximately the size, shape, and position of the heart, the relation of the anterior borders of the lungs, especially upon the left side, to it, and the presence of pericardial effusion when it is of sufficient amount. The area of superficial cardiac dulness is ascertained by light percussion in the parasternal line from above downwards until dulness with a distinct sense of resistance is reached, usually about the level of the fourth rib or its lower border. This point is in a transverse or oblique line extending downward and outward from midsternum. Next percuss over the lung upon the right side about the level of the fifth rib and in a transverse line across the sternum to the left. About or just beyond the middle line the sound again becomes dull and the resistance increased. This marks the limit of the anterior border of the right lung. Continuing to percuss in the same line and lightly as before, we reach a point at which the sound again becomes clear and which corresponds to the anterior border of the left lung at the level named. This determines the transverse diameter of the superficial area of cardiac dulness. The left lower angle corresponds to the apex and can be determined by palpation. The lower border is bounded by a line prolonged from the upper border of liver dulness to the apex of the heart.

The area of deep cardiac dulness may be roughly estimated by percussion in corresponding parallel lines from the parasternal line on the right side across the chest to the left and from above downward upon the left side along the sternal border, the parasternal line and the mamillary line. The base line is the same in both areas. The first modification of clear pulmonary resonance as we approach the heart may be accepted as a sign indicating the outline of that organ.

SIGNIFICANCE OF VARIATIONS IN THE CARDIAC DULNESS.

Alterations in the size of the superficial area, as has been heretofore stated, are usually signs of pulmonary rather than of cardiac disease. They correspond to increase as in emphysema, or decrease as in phthisis in the volume of the lung. This area together with the deep area undergoes changes, however, with changes in the volume of the heart.

Increase of Cardiac Dulness.—When the enlargement, as determined by percussion and other methods, extends chiefly to the left and downward, the longest diameter being oblique from above downward and to the left, it is the sign of hypertrophy and dilatation of the left ventricle. When the enlargement is to the right, with an extension of the dulness in the third and fourth intercostal spaces at the right border of the sternum and a rounded blunt apex, the longest diameter being transverse, it is a sign of hypertrophy and dilatation of the right ventricle and auricle. Enlargement both to left and right indicates hypertrophy and dilatation of both ventricles, the dilatation under these circumstances being almost always in excess of the hypertrophy.

Enlargement of the boundaries of precordial dulness to the left, right, and upwards may indicate the presence of a pericardial effusion. The dulness is marked and its outline is pyramidal or pear-shaped, the smaller end being directed upward. The increase in dulness may be first observed in the angle formed by the right border of the cardiac and the upper border of the liver dulness, which becomes at first rounded and then obliterated. The dulness may extend to the second interspace or higher and is sharply defined at its borders. In pericardial effusions of considerable size the compressed left lung yields a vesiculotympanitic or tympanitic resonance —dull tympany; skodaic resonance. The apex-beat may be felt or located by the intensity of the first sound within the borders of dulness. Percussion alone will not always enable us to make a differential diagnosis between a moderate pericardial effusion and extreme dilatation of the heart.

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The upper limits of hepatic and splenic dulness are determined by esicular resonance. With this exception the signs relate to dulness and ympanitic resonance and their modifications.

The normal dull areas in the abdomen are:

(a) **Hepatic.**—The upper border of dulness begins about the level of he sixth rib in the midclavicular line. Its lower border nearly correponds with the arch of the ribs. This area of dulness shifts with the espiratory movements about two fingers' breadth on quiet and slightly ore upon deep breathing. The dulness extends upon corresponding nes in the epigastric zone and its respiratory excursus is less at the ack than in front. The dulness of the left lobe extends to the left of he median line and is continuous vertically with that of the heart.

(b) **Splenic.**—The dull area of the spleen occupies the space between he ninth and eleventh ribs, its anterior border being slightly in advance f the midaxillary line. Its respiratory excursus is slightly less than that f the liver. The observation must in all cases be confirmed by palpation.

The above are constant in health.

Dull areas that are not constant also occur under physiological onditions. These are:

(c) **The Distended Bladder.**—The dull area is situated in the supraubic region in the median line. It may extend half-way to the umbilicus. t is oval and symmetrical in outline and disappears upon micturition or atheterization.

(d) **A Distended Stomach.**—A hearty meal or the large ingestion fluid will cause an area of dulness in the epigastrium which disappea in the course of digestion. The sharp contrast between the lower bord of the dulness and the tympanitic resonance of the transverse col indicates the position of the greater curvature of the stomach.

(e) **Fecal Masses in the Colon.**—In persons of sedentary habits is not uncommon to find areas of dulness corresponding to the course the colon, and especially to the left end of its transverse portion, whi disappear upon brisk and repeated purgation.

(f) **Pregnancy.** — The oval area of dulness gradually developi upward from the pelvis, always central, at first symmetrical, later deflect somewhat laterally, is suggestive. The diagnosis of this physiologic condition under ambiguous circumstances or in a doubtful case must a guarded one.

With the above exceptions the percussion resonance of the abdom is tympanitic. Its pitch varies with the dimensions of the particular spa and the tension of the contained air, being relatively high as the space small and the tension great. The stomach and colon yield therefore percussion note of lower pitch than the small intestines. The structu and functions of these organs are, however, such as to cause great vari tions in the size, tension, and relation of their various parts, and lessen t value of the signs obtained by this method of examination. The percussi signs are furthermore greatly modified by the thickness of the abdomin walls and their general state as to tension and relaxation.

PERCUSSION IN DISEASE OF THE ABDOMINAL ORGANS.

Under ordinary circumstances except as above stated the abdom in health is everywhere tympanitic beyond the borders of the liver ar spleen. Persistent dulness is significant of morbid conditions. It may general or local, continuous with the dulness of the liver or spleen separated from them, fixed or shifting.

General Dulness of the Abdomen.—The retracted abdomen se in the wasting diseases and in œsophageal and pyloric carcinoma, choler and the pernicious vomiting of pregnancy is usually dull upon percussio The areas of tympanitic resonance are limited in extent and of irregul distribution. This is especially true of the scaphoid abdomen so oft observed in meningitis, tumor of the brain, and lead colic. The bow are empty of air and collapsed.

The general distention due to fat in the walls and intra-abdomin fat, fluid within the peritoneal cavity, or abdominal tumor yields dulne upon percussion. The bowels contain air but under conditions whi modify the results of percussion. In the case of an excess of fat in t walls the force of the blow is not transmitted to the underlying gut; excessive omental fat the same is true. Fluid accumulates in the depen ent parts, displacing the coils of intestine, which float upon the surfa and yields dulness upon percussion at the lower levels with tympany abo shifting with change of posture, the line between them tending to mainta its correspondence with the plane of the horizon. Thus, in the recumbe posture there is general dulness save in a limited oval region around the umbilicus, over which there is tympanitic resonance; in the erect posture the resonance of this region is replaced by dulness while there may be demonstrated a broad line of tympanitic resonance in the epigastric zone, previously dull; in the lateral postures the area of resonance seeks the upper spaces and shifts alternately as the patient turns from side to side. The fluid commonly gravitates slowly from region to region and a few moments must be permitted to elapse before the change of note can be demonstrated. Large monocysts, as of the pancreas or ovary, also yield fluctuation and general dulness, but the intestines do not float at the highest level, being, on the contrary, pushed aside, and causing resonance in the flanks, which does not change to any great extent with change of position and does not present the oval area of tympanitic resonance in the umbilical region which is characteristic of ascites. The presence of peritoneal adhesions and a great quantity of fluid sometimes renders fluctuation obscure and the results of percussion uncertain by interfering with the free movement of the supernatant intestines. Tumors of sufficient size to give rise to marked distention and general dulness or flatness usually increase the anteroposterior diameter of the abdomen to a greater extent than the bilateral as compared with ascites and meteorism. The enlargement caused by tumor is not usually symmetrical. The intestines are pushed aside and tympanitic resonance is

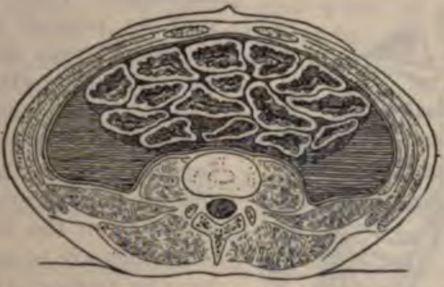

FIG. 76.—Free fluid in abdominal cavity—dorsal decubitus—flatness in flanks and tympany over supernatant coils of intestines.

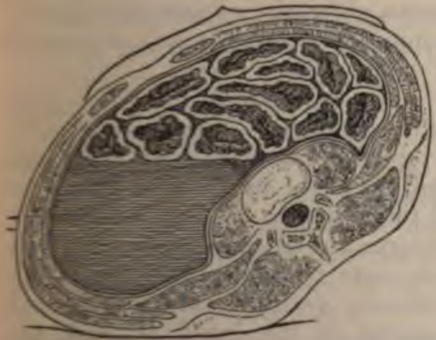

FIG. 77.—Free fluid in peritoneal cavity—lateral decubitus—flatness in dependent side and tympany above.

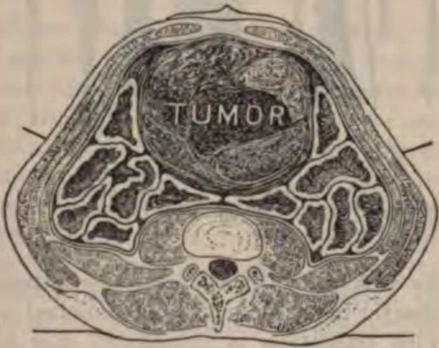

FIG. 78.—Abdominal tumor—increase in anteroposterior diameter—flatness centrally and tympany in flanks.

elicited upon percussion in the flanks and especially upon the opposite part of the abdomen to that from which the growth has developed—on the left side in case of tumor of the liver, on the right in case of tumor of the spleen, above in tumors springing from the pelvic organs, below in those springing from organs in the epigastric zone, and so forth, while over the tumor there is dulness. The list of tumors which attain dimensions sufficiently great to cause general distention of the abdomen comprises cancer, syphilitic and amyloid disease, and hydatid cysts of the liver; malignant disease and multiple cysts of the kidney; cancer of the intestines and peritoneum; ovarian cysts and uterine fibromata and retroperitoneal sarcoma. Very marked distention may be present in pancreatic cyst, hydronephrosis and tuberculosis of the mesenteric glands, and Hodgkin's disease. All these conditions yield dulness upon percussion. An important sign in the diagnosis of large neoplasms of the retroperitoneal glands arises from the fact that, while the intestines are in general pushed aside by the tumor, the ascending or descending colon, according to the side upon which the growth develops, passes obliquely across it and yields tympanitic resonance, at both borders of which dulness begins.

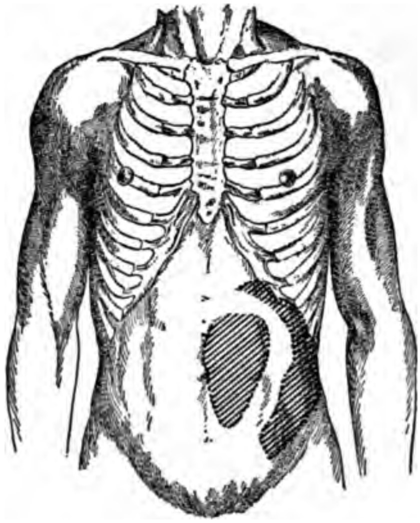

FIG. 79.—Tumor of left side of abdomen—dulness with strip of tympany corresponding to colon.

Gaseous distention of the abdomen—meteorism, tympany—yields tympanitic resonance at all points and increases the vertical diameter of the abdomen by pushing the diaphragm upwards and interfering with its descent. In extreme distention the note becomes higher in pitch, shorter in duration, and diminished in intensity until it finally may be dull. This condition is commonly due to paresis of the intestinal wall and occurs in peritonitis, the advanced stages of the infectious fevers, and hysteria. To a less degree it is present in cretinism, rickets, and pseudohypertrophic paralysis. Great dilatation of the stomach and congenital dilatation of the colon are attended with general abdominal enlargement over which the note is tympanitic.

Free gas in the peritoneal cavity may be the outcome of a perforating ulcer of the stomach or duodenum—peptic ulcer—or of the ileum in enteric fever or of the appendix. The accident which leads to the escape of gas is usually attended with severe abdominal pain, collapse, and meteorism. Rapid obliteration of liver dulness in an abdomen not previously much distended is an important sign. Mere disappearance of the anterior liver dulness at the margin of the ribs or in the nipple line may be a sign of ordinary meteorism. If, however, liver dulness is present in the infra-axillary line while the patient is in the dorsal decubitus and is replaced by tympanitic resonance when he is turned upon the left side, it may be inferred that there is free air in the peritoneal cavity.

Local Areas of Dulness.—Spasmodic local contractions of the abdominal muscles and phantom tumors may yield dulness upon percussion. In obscure cases a somewhat deeply seated tumor may be examined by percussion, if the walls are relaxed, by pressing with the pleximeter hand gently but firmly for a time until the bowel is pushed aside, and the mass may be recognized by palpation and its percussion signs ascertained. Any local tumor or new growth gives rise to percussion dulness in that area of the abdominal wall which overlies it. The variety of such morbid conditions is very great. The nature and point of origin of the most important of them have been indicated under the heading Palpation in Diseases of the Abdominal Organs.

AUSCULTATION.

Auscultation as a method of physical diagnosis is the art by which we recognize and interpret the sounds produced within the body in health and disease.

This is the most important of the methods of physical diagnosis. It is essential to the diagnosis of diseases of the organs of respiration and circulation and of limited service in the diagnosis of diseases of the digestive organs.

The Methods.—Auscultation is of two kinds,—*immediate or direct*, in which the ear is applied directly to the surface to be examined, and *mediate or indirect*, in which a stethoscope is employed. The latter was practised by Laennec, the discoverer of auscultation; the former has since come into use.

Each of these methods has its peculiar advantages in diagnosis. Direct auscultation is useful for a general survey of the chest, including both its respiratory and circulatory phenomena, the study of broad areas and the determination of the presence or absence of abnormal signs. It also enables us to detect the signs of deep-seated lesions, as central consolidation of the lung, which are not audible by the stethoscope. Indirect auscultation, on the other hand, is preferable for the nice study of the signs heard in limited areas, the point of maximum intensity of a murmur or the limits of a friction sound. Just as in palpation we use the palm of the hand to find and estimate the extent of the impulse of the heart and then study its force and characters with the smaller and more sensitive finger-tips, so the experienced diagnostician uses the two methods of auscultation. Like the other methods of physical diagnosis they are not independent and sufficient of themselves, but interdependent and complementary. There is no question as to which should be employed, since both are necessary: the one for one kind of observation, the other for a different kind; the one for clinical research, the other to control its results.

Many experienced auscultators use the direct method in the examination of the back of the chest and the stethoscope for the examination of the anterior surface, the reason for this being found in the difficulty in reaching the supraclavicular and axillary regions by the direct method, the closer study necessary in the examination of cardiac and pericardial conditions, and certain personal considerations which appeal to the user of the stethoscope.

Stethoscopes.—These instruments are made of various materials and shapes. The young auscultator of a mechanical turn of mind is very apt to turn his attention to the stethoscope and there are many inventions. Few only deserve serious consideration. The underlying principle is the conduction of the sound. There are two kinds of stethoscopes, the single and the double or binaural.

The single stethoscope was used by Laennec. The best form is the gun-metal instrument with detachable hard-rubber ear-piece devised by Hawksley of London.

The double stethoscope of Cammann of New York consisted of a chest-piece connected with two tubes fitted with ear-pieces. Many modifications of this instrument have since been made and the double stethoscope has come into general use. The chest-pieces as now made consist of interchangeable bell-like expansions of hard or soft rubber, or a shallow metal cup with a hard-rubber diaphragm held in place by a metal ring, seven-eighths of an inch in diameter so as to be applied to the costal interspaces, or larger; the tubes are long and flexible to enable the examiner to move the chest-pieces freely without changing his position, while the ear-pieces are in some instances attached to metal arms held together by a spring or hinged and held in position by a rubber band. In other forms the soft-rubber tubes are connected directly with the chest-piece and ear-piece, the latter retaining its place in the meatus by its appropriate shape and size.

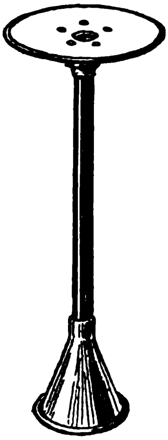

FIG. 80.— Hawksley's single stethoscope.

In selecting a stethoscope attention should be given to the kind. It should be an excellent conductor of sound as tested by comparing several different instruments under similar conditions, and simple in construction, durable, and convenient to carry. Attention must also be given to the particular instrument to see that the ear-pieces fit comfortably, that the pressure is right, and that extraneous sounds are excluded.

With a good instrument, even with the unaided ear, and a fair amount of training the sounds which constitute auscultatory signs may be heard. The problem in diagnosis is their proper interpretation.

The phonendoscope of Bianchi consists of a shallow metallic circular chest-piece with vibrating hard-rubber disks and soft-rubber tubing conductors to the ear-pieces. It is readily applied, and, while it intensifies the sounds, does not produce exaggerated sounds. It is especially useful in auscultatory percussion.

In the Bowles stethoscope the chest-piece is constructed with a vibrating hard-rubber diaphragm with the attachment for the conducting tubes at a right angle to its central axis. Multiple attachments are made for class demonstration. The sounds are intensified and the claim has been made that cardiac murmurs otherwise inaudible may be distinctly heard. The flat chest-piece is especially serviceable in the examination of the back of the chest in persons too ill to be moved, since it may, by pushing the bedclothes down, be slipped under the patient's back at different points without changing his position. Combination forms in which the Bowles attachment is fitted into the chest-piece of an ordinary stethoscope are sold.

The differential stethoscope of Alison has two chest-pieces with separate conducting tubes. This instrument enables the examiner to compare the sounds heard at different parts of the chest and to study differences in their acoustic properties as well as in the time of their occurrence. Notwithstanding its value it has not come into general use.

The Technic of Auscultation. — The following general rules are to be observed:

1. The patient's attitude should in so far as possible be comfortable and unconstrained.

2. Let the chest be bared or covered only with a towel or single layer of undergarment. When the stethoscope is used it is better to have the chest bare. When direct auscultation is practiced it is convenient and fitting, though not essential, to have a layer of thin stuff between the ear of the examiner and the skin of the patient. Auscultation cannot be properly performed through the patient's ordinary clothing. The superimposed layers of several garments, silk fabrics, and the suspenders or corsets not only mask the sounds within the chest but also give forth sounds of their own upon respiratory movements.

3. In indirect auscultation apply the chest-piece of the stethoscope closely to the surface, steadying it by grasping it between the thumb and index finger.

4. If the single stethoscope is used, it must be applied perpendicularly to the surface. If it is tilted, external sounds are not excluded.

5. The stethoscope must be applied very lightly in auscultation of blood-vessels. The rim of the instrument may cause a murmur in the vessels at the root of the neck or in the abdominal aorta by causing the physical condition to which such murmurs are due, namely, sudden narrowing of the lumen—stenosis.

6. Examine the chest in a routine manner first at one apex, then at the other, and at corresponding points upon the two sides from above downwards, in front, behind, and at the sides. Comparison and contrast are essential to auscultation. Equally important are the differences in the sounds upon ordinary quiet breathing, full respiration, and coughing. The respiratory signs are to be considered also in connection with the signs upon auscultation of the voice. In very serious cases, where the patient cannot be disturbed or where the condition can be at once recognized, a complete systematic examination may be omitted.

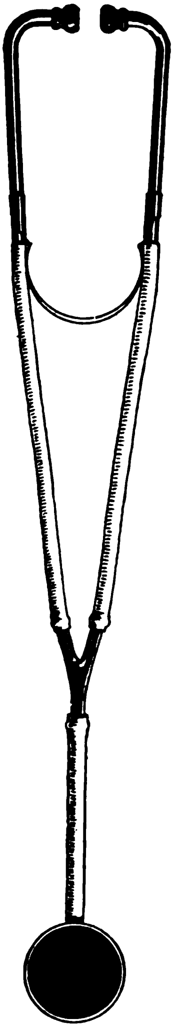

FIG. 81.—Bowles binaural stethoscope.

7. Examine the heart in the same systematic manner, placing the stethoscope over the puncta maxima in turn and noting the direction in which sounds or murmurs are propagated together with the presence or absence of friction sounds, etc.

8. Consider the patient. Do not fatigue him unnecessarily either in mind or body. Do not cause distress by undue pressure of the stethoscope or by insisting upon the repetition of deep breathing or cough when they give rise to pain. Conduct the examination with method, dispatch, and regard for his feelings and do not repeat it with unnecessary frequency.

9. Consider yourself. Assume a position which enables you to place your ear or the stethoscope in accurate relation to the surface to be examined. Use such patience with skill as will render the examination satisfactory to you. If, despite your efforts, the results do not justify a diagnosis, defer expressing an opinion until you have an opportunity of repeating the examination under more favorable circumstances. In dispensary and hospital practice be on your guard against vermin.

In children auscultation is even more valuable in the diagnosis of diseases of the chest than in adults. Owing to the great elasticity of the walls of the chest and the corresponding increase of resonance, percussion is of much less general applicability. Dulness, even when the physical conditions which cause it are present, is not usually so marked nor its limits so easily recognized, nor do we derive the same advantage from comparing and contrasting the two sides, since the acute pulmonary affections of early life are much more frequently double than those after the second dentition.

In children the back of the lungs should be first listened to. The diagnosis may often be made at once upon a careful and systematic examination of the back alone, after taking the history of the illness and noting the symptoms. This is especially true in acute and chronic bronchitis, croupous and bronchopneumonia, and pleural effusion. Crying is of great assistance. The deep inspirations develop the signs characteristic of the lesions which are present, and we also obtain the signs which arise from the character and modifications of the vocal resonance.

The position in which the child is examined by auscultation should vary with its age. Very young infants may be examined in either a lying or sitting posture on the lap of the nurse or upon a pillow; or they may be held in the arms of an attendant who presents one part of the chest after another to the ear of the physician. The physician himself may hold the baby seated upon his left hand and supported by his right hand applied to the front of its chest and listen to its back with his right ear. Older children may be held seated upon the forearm of the mother or nurse with the head resting upon her shoulder while the physician listens to the back.

The difficulty with beginners in auscultation is that they hear too much. They cannot at first discriminate between sounds that are significant and those which are irrelevant. The power to do this comes, however, with practice.

The most important of the sounds which, by a process of selective attention, the young auscultator must learn to disregard are the following:

1. *Outside Noises.*—A quiet room and silence are desirable but cannot always be secured. We must train ourselves not to hear extraneous sounds while engaged in listening to those which are the object of our immediate attention. Properly fitting ear-pieces and accurate adjustment of the chest-piece of the stethoscope are of help in excluding the sounds which we do not want to hear.

2. *Accidental noises* produced by the stethoscope. These comprise friction rubs of the instrument upon the skin, especially when it is dry and harsh or covered with coarse hair; friction rubs of the auscultator's fingers, or his sleeves, or the like, upon the stethoscope; friction or fine snapping sounds caused by the movement of one part upon another of an adjustable stethoscope of several pieces; sounds made by the breath of the examiner upon the rubber cross-piece or steel spring of the stethoscope, and finally the humming or buzzing sound—tinnitus—made by the ear-pieces. Most of these sounds are easily recognized and avoided. The last is to be diminished by very careful adjustment of the ear-pieces and overcome by usage.

3. *Adventitious sounds* conducted by the stethoscope but not properly constituting auscultatory signs. The sounds made by the friction of the clothing and coarse hairs are very confusing. The first are easily obviated; the second by practice, by applying the chest-piece beyond the borders of the hairy patch, or by the use of oil. Sounds produced by the contraction of muscular masses may often be heard, especially upon deep breathing, in various parts of the chest and in particular over the pectorals and trapezii. These sounds are faint and variable in kind but often quite distinct. They can be produced upon the forcible contraction of any muscle. The thenar mass, for example, when contracted with the stethoscope applied over it, affords a good illustration of such sounds. Cabot has suggested that auscultatory sounds described as "crumpling," "obscure," "distant," and "indeterminate" râles are in reality due to muscular contractions. The fact that such sounds are very often associated with distinct or easily recognized râles and other evidences of pulmonary disease and occur in individuals with atrophic chest muscles, should put us on our guard against hasty conclusions.

Auscultation as Applied to the Diagnosis of Diseases of the Organs of Respiration.

It is of practical importance that the movement of the tidal air on quiet breathing is in many persons not sufficient to cause auscultatory phenomena of significance. It becomes necessary then to listen to the chest during deep or forced respiration. If the patient is stupid or awkward, difficulties arise. He holds his breath, or pants, or makes strange noises, or does not appear able to take a deep breath. You show him how to breathe for the examination or you ask him to cough, listening to the respiratory signs during the deep inspiration which follows or precedes, or you ask him to count as long as he can with a single breath. The full breath which follows enables us to ascertain the presence or absence of abnormal signs. These difficulties are usually encountered in subacute and doubtful cases. In acute cases and in chronic cases with advanced lesions the signs are commonly distinctive upon ordinary breathing.

THE SIGNS IN HEALTH.

Auscultation of the normal chest discovers two respiratory sounds which are typical:

1. *Tracheal, bronchial, or tubular breathing,*
2. *Vesicular breathing,* and combinations of these types in varying degree, namely,
3. *Bronchovesicular breathing.*

1. **Tracheal, bronchial, or tubular breathing** is heard when the stethoscope is placed over the thyroid cartilage, over the trachea in the episternal notch, and in the upper part of the interscapular space upon the right side—normal bronchial respiration. Sometimes nearly pure bronchial breathing can be heard in health over the manubrium sterni or the three lower cervical vertebræ.

It has its origin in the larynx, and is sometimes for that reason spoken of as laryngeal, and, from the situations at which it is heard in health, tracheal or bronchial. Since it is conducted along the column of air in the bronchial system and probably also along its elastic walls and resembles the sound produced by breathing through a tube, it is called tubular.

This type of breath sound is heard with inspiration and expiration, these two elements of the sound being separated by a brief interval of silence at the end of inspiration. Its quality is bronchial, tubular, or blowing; its pitch relatively high as compared with vesicular breathing. The expiratory element is slightly more intense, usually of higher pitch, and slightly more prolonged than the inspiratory part. It may be imitated by slowly breathing through the hollow of the hand, closed by flexing the fingers till their tips touch thenar and hypothenar eminences, or through the lips and teeth held in the position to sound the German *ch.*

It is produced at the chink of the glottis where the air upon inspiration and expiration is thrown into eddies or swirls—*fluid veins.* For the reason that a similar mechanism is involved in the production of vesicular breathing and cardiac and vascular murmurs, it may properly be considered at this point.

The Theory of Fluid Veins. — Chauveau pointed out the fact that when a fluid is forced under pressure from a narrow into a wider tube or channel, or through a narrow opening into a large cavity or space, it is thrown into swirls or eddies, the vibrations of which, transmitted to the enclosing substance and to the surrounding air, are recognized as auditory phenomena. These swirls have been called fluid veins. They are currents within currents, and their vibrations are not only transmitted laterally but also longitudinally in the stream in which they exist, so that the sounds are heard over the point at which they are produced and at a distance in the direction of the flow. The extent and force of these swirls and the consequent loudness of the sound by which they are represented depend to some extent upon the composition and density of the fluid but mainly upon the force of the current. The student will realize the nature of fluid veins and the part they play in the production of the bronchial respiration and the vesicular murmur,—for the air acts in the same way as other fluids,—and especially their part in the production of endocardial and vascular murmurs, if he considers the course of a rivulet which flows at one time down a steep and rapid course, and at another along a nearly level bed with even sides and a smooth bottom, and now as a gentle stream and again with considerable force. The quiet current flowing in even banks is smooth and noiseless, while the little torrent in its rocky bed has its surface thrown into countless screw-like swirls, and murmurs or roars, according to the force and volume of the water. The stream is an open channel; the respiratory and vascular spaces are closed tubes; but the mechanism by which the sounds are produced is the same in each. It is evident that the intensity of the bronchial respiration will vary with the quantity of the tidal air, the force with which it passes through the glottis, the distance at which it is heard, and the conducting properties of the media through which it is transmitted. Variations in pitch depend upon the size and shape of the spaces—pharynx, buccal cavity, trachea, etc.—which constitute resonating chambers in relation with the larynx. We are thus prepared to find wide differences in intensity and pitch in the breath sounds which have the characteristic tubular or bronchial quality.

2. **Vesicular Breathing.**—Respiration of this type is heard when the stethoscope is placed elsewhere over the chest where the lungs are in contact with the chest wall, namely, in the front of the thorax with the exception of the area of superficial cardiac and hepatic dulness, in the infrascapular regions and in the axillary and the upper part of the infra-axillary regions. In the right interscapular region the breathing in health is usually bronchovesicular, the vesicular element predominating.

This sound has its origin in the parenchyma of the lung, and is due to the transmission of the vibrations, caused by fluid veins or swirls in the air passing into and out of the infundibula and alveoli, to the surface of the chest. The hypothesis that the vesicular respiration is merely a modification of the bronchial appears to me to rest upon insufficient facts. This type of breathing is heard throughout the whole act of inspiration, and is immediately followed, without an interval of silence, by a short but inconstant expiratory sound. The inspiratory portion is low in pitch as compared with bronchial respiration, of variable intensity, and has the characteristic quality described as vesicular, which is to be learned only by experience. It is sometimes called the vesicular murmur, and it may be of service to the student to note that it possesses the distinguishing peculiarity of murmurs, namely, that they are sounds made up of a multitude of small sounds, all having about the same acoustic properties, as we speak of the murmur of a crowd, of the leaves of the forest, of the sea, and so on. The expiratory part is still lower in pitch than the inspiratory, much less intense, frequently absent altogether, and does not exceed one-third the length of the latter.

The vesicular murmur is not equally intense in all parts of the chest. It is loudest in the infraclavicular, axillary, and infrascapular regions, and fainter at the bases in front and behind. That is to say, it is loudest over large masses of lung tissue and faintest over the thin wedge-shaped borders. But it is also less distinctly heard in the mammary and scapular regions. We conclude therefore that it is not well conducted through thick layers of muscle, bone, and fat. Wherever heard, whether loud or faint, it retains its characteristic breezy quality and low pitch, and the relative duration, intensity, and pitch of the inspiratory and expiratory elements are preserved.

The vesicular murmur is feeble and distant on shallow breathing and intense upon deep breathing, especially after prolonged deep breathing as after exertion. It is intense over the unaffected lung in cases in which the opposite lung has been thrown out of service by disease, and in healthy children, hence it is spoken of, when thus intensified, as "puerile" or "exaggerated" respiration. Intense vesicular respiration somewhat modified is spoken of as "rough"; just as bronchovesicular respiration is often called "harsh."

3. **Bronchovesicular Breathing.** — This form of respiration, as the name indicates, has the characteristics of both bronchial and vesicular breathing and consists in fact of a breath sound in which both are present. It is heard in the normal chest very often, but not invariably directly below the right clavicle, and quite constantly at the sternal borders opposite the lower part of the manubrium and in the upper portions of the interscapular spaces, namely, in situations in which both sounds are within range of hearing. Many of the difficulties regarding bronchovesicular respiration are solved when we recognize the fact that it is made up of the two forms in varying degrees of combination, so that it sometimes presents the traits of bronchial breathing slightly modified by the admixture of faint vesicular breathing and sometimes those of vesicular breathing slightly modified by bronchial, and between these two we encounter every grade of admixture. This gradation by which the breath sound passes from bronchial to the vesicular respiration may be heard in the normal chest by moving the stethoscope from point to point, starting at that part of the manubrium over which bronchial breathing is heard and advancing towards the nipple where the vesicular murmur alone can be recognized. The inspiration becomes, as we proceed, lower in pitch, less intense, and longer in duration, and the expiration also lower in pitch and less intense, but shorter in duration. The interval of silence which is characteristic of bronchial respiration is filled by the vesicular element in bronchovesicular respiration. This interval of silence is present in bronchial breathing because the swirls —fluid veins—by which the vibrations causing the sound are produced, arise at a single point, the glottis, and there is at that point an interval of equilibrium between the flood tide of inspiratory and the ebb tide of expiratory air. The vesicular murmur, on the other hand, is produced at a multitude of different points, and the moment of silence is as variable as the individual little sounds which cover the whole time of the inspiratory act, since vesicles at the distant periphery of the lung are still expanding when those nearer the inlet have ceased to dilate.

The conditions which modify the bronchial respiration as a physical sign and those which modify the vesicular murmur also modify the bronchovesicular breathing. It therefore presents differences in intensity, duration, and pitch, corresponding to variations in the quantity and force of movement of the tidal air, to the size and shape of the resonating chambers formed by the upper air spaces and the tracheobronchial system and the physical condition of the intervening tissues through which the sounds are conducted to the ear. The qualities of the two component types of breathing, though they vary in proportion, are not changed.

It is essential for the student to become familiar with these three ms of normal breath sounds and the localities in which they may be rd in the normal chest. Familiarity with normal physical signs is the t step towards the recognition of those which are abnormal.

Bronchial breathing is heard in the front of the neck and over the per part of the manubrium, vesicular breathing over the greater rt of the chest, as above, because the mechanism by which they are pectively produced is situated in the regions indicated. Broncho- sicular respiration is heard normally over the lower part of the manu- ium and laterally to it and in the interscapular spaces because both its ctors are within the range of hearing. Bronchial respiration is heard in e right interscapular space and bronchovesicular respiration is more ominent (bronchial) over the upper part of the right lung by reason of e larger size and higher origin of the large bronchus on the sight side.

Bronchial or tubular breathing is conducted in the column of air in e bronchial tree to its remote twigs. It is not conducted to the surface the chest because the vibrations are on the one hand lost in the mass cushiony, elastic vesicular tissue which constitutes the lung parenchyma, d on the other hand the bronchial sound is drowned in the vesicular urmur. When this tissue becomes solidified by compression—atelectasis or by an exudate—pneumonia, tuberculosis—the vesicular murmur is ne away with and the vibrations conducted by the bronchial tubes are eely transmitted to the surface.

THE SIGNS IN DISEASE.

The auscultatory phenomena which constitute abnormal or morbid ysical signs are (a) variations in the intensity and rhythm of the breath unds, (b) normal physical signs heard in abnormal situations, and (c) rely adventitious sounds.

(a) Variations in Intensity and Rhythm.—Bronchial Respiration.— has been explained that bronchial respiration heard beyond the limits certain regions of the chest in which it is normally present is usually e to the consolidation of lung tissue—atelectasis; presence of an exudate, in pneumonia, tuberculosis, etc. It may, however, arise in connection ith cavities in the lungs or pneumothorax. Under these circumstances ere are layers and masses of compressed or consolidated lung tissue esent and the peculiar modification of the bronchial respiration is prob- ly due to the fact that the cavity acts as a resonating space. Bronchial spiration varies greatly in pitch. This variation is the outcome of com- ex conditions not fully understood, but has been attributed to the rela- ve size of the tubes or cavities from which the sound is directly conducted rough consolidated tissue to the ear. The pitch is usually high and the unds whiffing or snoring in pneumonia of the lower lobes, especially in ildren, and low and the sound soft and sighing or metallic over cavities.

The following varieties of bronchial respiration are to be especially nsidered:

1. *Feeble and distant bronchial respiration* is often heard in central eumonia and pulmonary infarct and over a pleural effusion. In the

former case the bronchial breathing may be only heard upon deep inspiration and is therefore inconstant; in the latter it is frequently so faint as to be overlooked. The sound is conducted by the chest wall or by tense adhesions, the result of former attacks of pleurisy.

2. *Intense bronchial breathing* usually conveys the sensation of being close to the ear, that is, well conducted. It accompanies dense consolidation of the lung in which vicarious or supplemental respiration is well established.

3. *Absence of bronchial respiration* or its sudden disappearance under conditions in which the mechanism for its conduction exists may be due to the plugging of a large bronchus with a mass of tenacious exudate. The disappearance of cavernous or amphoric respiration often results from the accumulation of fluid within the walls of the cavity. Under these circumstances the bronchial respiration returns after cough and expectoration.

4. *Cavernous respiration* is a variety of bronchial breathing sometimes heard over a cavity. It is low in pitch, soft in quality, and the expiratory element is prolonged.

5. *Amphoric respiration* is a variety which has the peculiar quality heard when one produces a sound by blowing across the mouth of an empty jar or bottle. The pitch is variable, usually low, and the sound is hollow, metallic, and musical. Amphoric respiration is never heard over the normal chest, and indicates a superficial cavity with rigid walls—or pneumothorax —having free communication with a large bronchus. The sound may be imitated by whispering "who" with some force and the lips held rigid.

Vesicular Respiration — The normal vesicular murmur undergoes modifications in intensity and rhythm which are of diagnostic significance.

1. *Feeble vesicular respiration* primarily indicates diminution in the quantity and energy of the movement of the tidal air. Hence it is present in varying degrees in quiet breathing in aged and bed-ridden persons, in paretic conditions of the respiratory muscles, including the diaphragm, when the movement of the diaphragm is impeded by meteorism, ascites, abdominal tumor, or pregnancy. The vesicular murmur is often feebly heard because it is poorly conducted, as in very thick chest walls. In pleural adhesions the expansion of the periphery of the lung may be embarrassed, and with thickening conduction is also impaired. A thin layer of effusion or a tumor acts in the same way. In pneumothorax the lung is compressed and removed from contact with the chest wall, and the vesicular murmur, if heard at all, is faint and distant. In acute bronchitis the swelling of the mucosa and the presence of the exudate interfere with the access of air to the vesicles and proportionately enfeeble the vesicular murmur, especially over the lower lobes. In chronic bronchitis enfeeblement is brought about by the accompanying emphysema and restricted movements of the chest.

In congestion and œdema of the lungs the murmur is enfeebled.

Emphysema by impairing the elasticity of the lungs and restricting the respiratory excursus increases the residual and diminishes the tidal air, thus rendering the vesicular murmur faint and in rare cases almost wholly abolishing it. Pain, as in pleurisy, restricts the respiratory movement and renders the vesicular sound faint.

Occlusion of the upper air-passages, as by spasm, œdema of the glottis, the presence of an exudate, as in diphtheria, quinsy, or retropharyngeal abscess, renders the murmur feeble in proportion to the extent of the obstruction. Pressure upon the trachea or a primary bronchus by aneurism, tumor, or enlarged lymph-gland acts in the same way. A foreign body or a plug of tenacious mucus in a bronchus enfeebles the respiratory murmur in the corresponding region to a degree proportionate to the stenosis. In such conditions the occurrence of râles obscures the enfeeblement of the respiratory sounds and the latter will be overlooked unless made the subject of especial attention.

2. *Absence of the vesicular murmur* may be noted over an area of the chest more or less extensive when the obstruction to a bronchus in any of the foregoing conditions is complete. Marked obstruction of the upper air-passages is at once followed by the signs of asphyxia. The murmur is absent over the greater part of the chest in rare cases of advanced emphysema, and no respiratory sound is heard over a pneumothorax not communicating with a bronchus, a large pleural effusion and locally over limited areas in some cases of cirrhosis of the lung and at the apex in rare instances in beginning tuberculosis.

3. *Intensified or exaggerated vesicular breathing—puerile, vicarious, or compensatory respiration*—is normal in childhood and gradually decreases until some time before puberty the intensity of the sound becomes that of adult life. It occurs in health after exertion and in dyspnœa from almost any cause in which there is no obstruction to the entrance of air. It occurs over one lung when the other is put out of service, as in pneumonia, large effusion, tumor, etc., and in some instances over a portion of one lung under similar conditions, hence the adjectives vicarious and compensatory.

4. *Derangements of rhythm occur in emphysema*, in which the loss of elasticity relatively prolongs the expiratory act and the expiratory sound; in asthma, in which the dyspnœa is expiratory, in the ordinary dyspnœa or panting of great exertion, in which the inspiratory and the expiratory breath sounds are nearly equal, and in various forms of inspiratory dyspnœa which are attended by diminution of the intensity and prolongation of the inspiratory element of the vesicular murmur.

5. *Interrupted or cogwheel inspiration* is characterized by a series of two, three, or four inspiratory sounds instead of the normal continuous murmur. It indicates in some instances a fault in the muscular function and occurs during periods of excitement or during a chill; more commonly it is a sign of early pulmonary tuberculosis, the air entering adjacent lobules in turn as the force of inspiration increases. It is usually heard in limited areas. When restricted to the precordial space it is significant of pressure of the heart upon the borders of the lung—cardiopulmonary murmur. In some instances the respiratory sound is not actually broken, but wavy or jerky, and is then described under these terms. It is not rarely present in tuberculosis before the disease has shown itself by other signs, and individuals who present it should be carefully watched. In other cases it is wholly without diagnostic significance, which it acquires only in conjunction with other physical signs or the symptoms of pulmonary disease.

(b) **Normal Physical Signs in Abnormal Situations.—Normal Sounds Heard in Abnormal Situations.**—Note the relative duration of the inspiratory and expiratory sounds and determine the presence or absence of an interval of silence between them and the quality of the sound, whether soft and breezy—vesicular murmur; blowing and tubular—bronchial breathing; or whether these qualities are both present—bronchovesicular. The most important facts for the beginner in the recognition of bronchovesicular respiration are the prolongation and relatively high pitch of the expiratory sound.

Perfectly normal vesicular respiration is rarely heard in other than its extensive normal domain in the chest. The rare cases of dextrocardia are attended with dislocation of the precordial space, and fibroid contraction of one lung frequently displaces the border of the opposite lung towards the affected side so that it occupies the area of superficial cardiac dulness in whole or in part. The modified respiration of emphysema, faint and prolonged, is sometimes heard in the precordia and over the upper normal area of the liver dulness.

Bronchovesicular and bronchial respiration are on the contrary common and significant signs of disease in the chest. The lesions are commonly progressive, and bronchovesicular usually, both in acute and chronic affections, precedes and progressively develops into bronchial respiration. Pulmonary consolidation either from compression or infiltration is the underlying physical condition and reaches its extreme development whether rapidly or slowly by progressive advance.

These signs are heard over the compressed lung in the following conditions: pleural effusion, the area in which they are present becoming more limited and the respiration more characteristically bronchial as the effusion augments; pericardial effusion; pneumothorax, in which more or less complete compression of the lung, unless prevented by old partial adhesions, takes place rapidly; tumor of the lung or pleura; massive enlargement of the heart, and large aortic aneurism. They are heard over the lung undergoing solidification or already solidified from infiltration in tuberculosis, bronchopneumonia, croupous pneumonia, pulmonary infarct. As already pointed out, distant bronchial breathing may frequently be heard over an effusion. It remains to point out the more important fact that loud, distinct, and well-conducted bronchial respiration is by no means uncommon over pleural effusions of large amount in thin-walled individuals and especially in children. This sign is conducted from the compressed lung by way of the wall of the chest and probably in some cases also along bands of old adhesions tightly stretched between the compressed lung and the chest wall by the force of the accumulating fluid. In pneumothorax the variety of bronchial breathing known as amphoric is heard when there is free communication between the pleural cavity and a bronchus.

Cavernous or amphoric respiration may be heard over cavities, whether due to the breaking down of lung tissue (tuberculosis, abscess, gangrene) or to dilatation of bronchi (bronchiectasis). Deep-seated cavities due to any of these causes may be attended with distinct bronchial respiration yet be difficult to locate with precision.

Bronchovesicular respiration must be distinguished on the one hand from puerile or exaggerated vesicular respiration and on the other from ronchial respiration. The breezy quality, low pitch, short expiratory lement, and absence of a period of silence between inspiration and expiraion are characteristic of the former, however intense. The tubular quality, elatively high pitch, longer expiratory sound, and an interval of silence re distinctive of the latter, and between the two are all degrees of transitional sounds. Normal in the right infraclavicular region, at the upper sternal borders, and in the neighborhood of the upper dorsal vertebræ, bronchovesicular respiration elsewhere in the chest becomes a sign of disease, and denotes partial consolidation of the lung, patches of collapse or infiltrated lung or consolidation with intervening normal vesicular tissue. This sign is present in early pulmonary tuberculosis, at the borders of the exudate in croupous pneumonia, in bronchopneumonia, and in incomplete atelectasis from any cause.

(c) **Adventitious Sounds.**—Purely adventitious respiratory signs are of two kinds: (1) Râles, which are produced by abnormal conditions within the lungs, and (2) friction sounds which originate in the pleura.

1. **Râles.**—Literally a râle is a "rattle" and may be defined as an abnormal respiratory sound heard on auscultation. Râles are grouped as dry or moist according to the impression conveyed to the mind as to the presence or absence of fluid in the mechanism by which they are produced. They are laryngeal, tracheal, bronchial, vesicular, and cavernous, according to the situations in which they occur.

In general râles or rhonchi are generated in the air-passages by the ebb and flow of the air when their lumen is contracted or when they contain fluid—dry and moist bronchial râles. Certain râles originate in the bronchioles and vesicular structure of the lung (vesicular râles), others in cavities (gurgling), and finally the succussion sound and the sign known as *gutta cadens* or *metallic tinkling* have their origin in hydropneumothorax. Râles may be heard upon inspiration or expiration or during both acts. They may obscure the normal breath sounds or entirely replace them.

DRY RÂLES are produced by stenosis of the bronchial tubes. This narrowing may be present at one point only as in laryngeal diphtheria or œdema of the glottis, or a tumor pressing upon the trachea, but is usually present at the same time at many points and in many bronchial tubes of varying diameter. It is brought about by a variety of pathological conditions, as a mass of tenacious mucus adherent to the surface of the tube, swelling of the mucosa or submucosa, spasmodic contraction of the bronchial musculature, and the external pressure of enlarged glands or a tumor. When this narrowing involves the smaller bronchial tubes the râles which result are high pitched—*sibilant;* when it affects the larger tubes the râles are low pitched—*sonorous.* They resemble the cooing of doves, the hissing of geese, and have very often a musical quality. Sometimes they are groaning or squeaking. In asthma they are often heard in great variety of size, pitch, intensity, and quality, both upon inspiration and expiration, and appear and disappear with the most remarkable modifications and great rapidity.

MOIST RÂLES are caused by the passage of air through the bronchi when they contain fluid—mucus, pus, blood. The mechanism consists in he presence of bubbles or diaphragms before the incoming and outgoing air which continuously burst and reform. When this process takes place in the larger tubes, the bubbles are large and the *râles coarse or large bubbling;* when in the smaller tubes, they are finer, *small bubbling or subcrepitant râles.* Large moist râles are usually low in pitch; small moist râles higher, and in this respect moist and dry râles correspond. The tracheal râle or *death-rattle* is an example of a very coarse râle; the small moist or subcrepitant râle heard in bronchopneumonia in both respiratory acts is an example of a fine moist râle.

Both dry and moist râles vary in intensity and locality. The extent of the area over which they are heard depends upon that of the process by which they are caused; their acoustic characters upon the physical changes produced by that process. In bronchitis râles are very often best heard at the bases of the lungs posteriorly; in tuberculous disease of an apex, in the subclavicular region. Râles are very often influenced by the act of coughing and expectoration. Dry râles produced by pressure stenosis, tenacious exudate which cannot be dislodged, or bronchial spasm, do not disappear upon coughing.

Vesicular or crepitant râles originate in the finest bronchioles and air-cells. Notwithstanding the differences of view in regard to the mechanism by which they are produced, the weight of evidence is still in favor of the theory that it is by the inspiratory separation of the walls of terminal structures—bronchioles, alevoli—previously collapsed or held together by a thin layer of sticky exudate or serum. In support of this theory the following facts may be adduced: This râle, at one time held to be pathognomonic of croupous pneumonia, is now known to occur also in other pathological conditions in which an exudate or blood is present in the lung parenchyma, as pulmonary œdema, hemorrhagic infarct, and acute pneumonic phthisis. It is common in partial atelectasis—atelectatic, crepitation of Abrams. Crepitant râles sometimes associated with subcrepitant râles are frequently heard during deep inspiration at the bas of the chest posteriorly and laterally in persons whose respiration is habitually shallow. This is not only the case in bed-ridden individuals but al in many healthy persons, especially after middle age. The crepitant râ is heard only upon inspiration. The subcrepitant râle with which it often associated is usually coarser and slightly moist.

The crepitant râle is usually heard towards the end of inspiratio the individual râles are of the same size and intensity and they oft occur in "showers," a large number of single sounds having the sa acoustic properties following each other in rapid and irregular successio

The crepitant râle occurs in croupous pneumonia at the beginning the process,—*crepitus indux,*—disappears when the exudate underg coagulation, and reappears together with subcrepitant râles when exudate undergoes liquefaction and resorption,—*crepitus redux.* T auscultatory sign may be imitated by placing a little mucilage betw the finger and thumb and making repeated contact and separation. W contact there is no sound, but upon separating the thumb and fing string of tenacious mucilage is drawn out which finally snaps with a sh sound not unlike the râle. It may also be imitated by the crackli fine salt thrown upon the fire, the creaking of a silk garment, or lig rubbing a few strands of hair between the thumb and finger. If the stethoscope is applied over the thick growth of coarse hair found upon the chest of many men, a sound closely resembling crepitation will be heard. Crackling is the term used technically to designate a râle coarser than crepitus but having in other respects similar acoustic properties. This râle consists of a limited number of well-defined sharp crackling sounds often heard in beginning pulmonary tuberculosis or at the borders of an advancing tuberculous lesion and for this reason is of considerable diagnostic importance. The distinction between crepitus and crackling is not always unattended with difficulty. Crepitus consists of a number of fine sounds, heard only upon inspiration and often over a considerable area at the base of the lung; crackling of a few sharp, well-defined, rather coarser sounds heard also in inspiration but over a limited area and commonly at the apex. It is probable that the mechanism is the same in both, but that crackling occurs in limited lesions, hence only a few individual sounds are heard; in wider spaces, terminal bronchi, hence the sounds are coarser; and at a point surrounded by densely consolidated tissue, hence they are better conducted to the ear. Moist crackling and clicking are varieties of crackling which are regarded as indicative of softening tubercle. In certain cases of dry or plastic pleurisy fine, dry friction sounds are to be heard which can scarcely be distinguished from subcrepitant râles. If they occur only upon inspiration they may be mistaken for crepitus.

Gurgling or the râle of cavities is caused by the entrance and exit of air in a cavity containing fluid. Coarse churning sounds are heard resembling those produced by pouring fluid rapidly from a bottle. These very coarse, well-defined râles are known also as cavernous, and sometimes have the metallic or amphoric quality.

Metallic Tinkling—Gutta Cadens.—All râles heard in pneumothorax acquire the amphoric or metallic quality. In some instances single râles having an exquisite metallic or bell-like musical quality may follow deep inspiration or the act of coughing. This sound, which resembles that made by single fine shot dropped into a metal bowl or basin, was at one time thought to be caused by a drop of exudate or pus collecting at the vault of the cavity and falling upon the surface of the fluid collected at its base. It is now known that it may occur in the absence of any such collection of fluid and that it may be due to the bursting of a bubble formed at the pleural orifice of a bronchopulmonary fistula.

Hippocratic Succussion.—This phenomenon, although it is not a râle in the narrow sense, may be best described at this point. It is characteristic of hydro- (pyo-hæmo-) pneumothorax and consists of a distinct loud splashing which may be heard and felt when the thorax is suddenly shaken. It is due to the swash of the free fluid against the wall of the chest, just as a similar sound is produced by the sudden movement of a partially filled cask.

The Bronchopulmonary Fistula Râle.—In hydro- or pyopneumothorax, when the accumulating fluid rises above the pleural opening of the fistula there may be sometimes heard in connection with paroxysmal cough bubbling sounds due to inspired air being forced from the lung and through the fluid. Under such circumstances violent spells of cough apt to be followed by copious expectoration.

Râles may be conveniently grouped as follows:

- Bronchial Râles
  - Dry or Vibrating
    - Low pitched—Sonorous.
    - High pitched—Sibilant.
  - Moist or Bubbling
    - Large bubbling—Mucous.
    - Small bubbling—Subcrepitant.
- Vesicular Râles
  - Crepitus.
  - Crackling (Clicking).
- The Râle of Cavities
  - Very Coarse Bubbling—Gurgling.
  - Cavernous and Amphoric Rales.
- Râles in Pneumothorax and similar conditions. (Hydro-pyo-hæmo-pneumothorax)
  - Metallic Tinkling—Gutta Cadens.
  - The Bronchopulmonary Fistula Râle.
  - The Hippocratic Succussion.

2. **Friction Sounds.**—The surfaces of the normal pleura, being moist and smooth, glide noiselessly over one another with the movements of respiration. When, however, the serous membrane is roughened by the presence of a fibrinous exudate, as in pleurisy, the movement of the opposed surfaces gives rise to sounds known as "pleural friction sounds" or "friction rubs." As the lesions of pleurisy vary from a mere dryness of the surface in the beginning to every grade of exudate in amount, texture, and arrangement, including the fibrinoserous forms, so the friction sounds present great diversity in their acoustic properties, not only in different cases but also in the same case during its course.

The general and almost constant character of pleural friction is, however, that of the sounds produced by the rubbing together of dry or slightly moistened surfaces, and is properly characterized as grazing, rubbing, creaking, leathery. grating, rasping, and the like. Friction sounds are usually jerky and irregularly interrupted, and change in character not only in the course of time but even in the course of a single respiratory act. They are superficial and give the impression of being produced very near the ear. They vary in intensity from a mere graze, scarcely audible, to a coarse, loud, and prolonged creaking like that of new leather and audible to the patient himself or the bystanders. They are described as fine, medium, or coarse. They are as a rule best heard and often only heard in the infra-axillary or inframammary region where the respiratory excursus is widest and the pleura investing the thin wedge of lung is in contact upon one side with the costal and upon the other with the diaphragmatic pleura. Not being well conducted, they are heard where they are produced, so that in cases of diaphragmatic pleurisy the friction sounds may be heard below the level of the lung, in croupous pneumonia opposite the seat of the exudate, and in the earliest days of phthisis at the apex. They may sometimes be heard over the entire lung from the apex to the base. In children and spare persons the intensity of these sounds may be increased by firm pressure upon the chest, and they are often attended by a palpable sign—*friction fremitus*. They occur most commonly during inspiration and especially toward the end of the act, and are frequently heard also during expiration. Less often they are present during expiration alone.

Friction sounds are sometimes inconstant, ceasing after several deep inspiratory acts and being again heard after a period of quiet breathing. They are not modified, however, to the same extent as râles, nor do they disappear upon coughing and the expectoration of mucus. Various postural methods of bringing out friction sounds in suspected cases have been described, as raising the arm upon the affected side or having the patient quickly rise from the recumbent to the sitting posture during held expiration and then take a very deep inspiration.

Deep breathing, coughing, pressure upon the affected side, not only increase the intensity of the sounds, but are also attended with pain. In exceptional cases friction sounds are unattended by pain during these acts. When a plastic pleurisy is followed by a serofibrinous exudate the friction sounds disappear, but recur upon the resorption or removal of the fluid. They are usually present upon one side of the chest only, but may sometimes, especially in disseminated tuberculosis, be heard in circumscribed areas on both sides.

Crumpling friction sounds are the signs of acute inflammation of the pleura. When the process subsides the surfaces become fused, the fibrinous exudate organized. The condition is that of adherent pleura and, unless dense and extensive, does not give rise to physical signs. In old pleurisy at the apex and especially when cavities exist, curious, low-pitched, soft, creaking sounds are sometimes heard. This sound resembles that produced by squeezing soft thick paper together in the hand in irregular folds and is described as crumpling. It is present upon inspiration and expiration and is not affected by cough, nor has it the characters by which we recognize râles.

In some cases of pleural effusion a considerable period elapses between the resorption of the fluid and the formation of adhesions. During this time friction sounds may be heard and the patient may experience annoying grating or rubbing sensations, especially upon deep breathing or coughing.

Sounds closely simulating friction sounds may be produced by rubbing the thumb and finger together near the ear or by holding the hollow of the hand over the ear and rubbing or stroking the back of it with the fingers of the other hand. There is a fine friction sound which cannot be distinguished from crepitus. Both occur in showers at the end of inspiration, both are close to the ear and have the same acoustic qualities, both are accompanied by an expiratory element which may be in one case a friction sound and in the other a subcrepitant râle. By the sound itself the differentiation is impossible, but when concomitant phenomena are taken into account we find the friction sound is usually more limited in extent, attended more commonly by expiratory sounds, is less uniform in character, and disappears when the movement of the chest wall is restricted by compression, while crepitus persists. The distinction between fine friction of this form and the crepitant râle or crackling is rather of theoretical than practical importance when we reflect that in pneumonia, when, as is commonly the case, the exudate extends to the periphery of the lung, the pleura overlying it is the seat of an inflammatory exudate, and in tuberculosis of the apex the early lesions which give rise to creaking are accompanied by a circumscribed pleurisy. In point of fact when we hear one of these signs the other usually is also present.

The friction sound which closely resembles crepitus or crackling is very rarely, if ever, heard in simple, uncomplicated pleurisy.

Friction sounds heard over the chest are significant of pleurisy. Those over the precordial space, having the cardiac rhythm, are usually but not invariably signs of pericarditis. The subject of pericardial and pleuropericardial friction will engage our attention in a subsequent section. Friction sounds heard in the epigastric zone constitute in rare instances the signs of a peritonitis. The effusion in hydrothorax is not preceded by a friction sound. Pleurisy is frequently primary; often secondary to intrapulmonary disease, pneumonia, tuberculosis, cirrhosis of the lung, abscess, gangrene, or cancer; and sometimes, especially upon the right side, secondary to subdiaphragmatic disease, as abscess, cancer or hydatids of the liver, or subphrenic abscess. Friction sounds may therefore be significant of any of these affections.

Riesman has described under the term subpleural friction a fine soft rubbing or crepitation which occurs in the absence of pain or the signs of consolidation in miliary tuberculosis. The difficulty in distinguishing fine pleural friction from crepitus has already been discussed.

AUSCULTATION DURING PHONATION.

Auscultation of the Voice in Health and Disease.—The sounds heard upon auscultation of the chest of a person who is speaking when the face of the patient is turned away or the opposite ear of the examiner closed, or when the binaural stethoscope is employed, constitute the set of physical signs comprised under the general term vocal resonance, and have diagnostic value. The ordinary spoken and the whispered voice are studied. Obstacles to the employment of this method of physical diagnosis consist in want of coöperation, as in children and extremely ill persons, in inability to use the voice, as in mutes, those suffering from aphonia from any cause, and in extremely feeble patients and great obesity.

The Technic.—The patient is instructed to turn his face away and count "one, two, three"; or repeat "twenty-one" or "ninety-nine" in the loud voice or in a stage whisper. The sound is conducted through the bronchi and along their walls in the same manner as in a speaking tube and greatly dispersed and damped in the cushiony vesicular tissue. Changes in the physical condition of the lung parenchyma favor or still further impede the transmission of the voice in such a manner that increase, diminution, or absence of vocal resonance correspond to these changes and thus become signs of disease. The modifications of vocal resonance correspond in general to those of vocal fremitus and have the same significance.

Normal Vocal Resonance.—The voice is heard as a confused inarticulate hum, most distinct in adults possessed of deep voices and tremulous in aged persons. This sound is more intense upon the right than upon the left side and at the apices than at the base. As the stethoscope is carried to a position nearer the main bronchi the resonance becomes louder and more distinct until finally, when it is placed over the bronchi or trachea in the position in which normal bronchial breathing is heard, the audible words may be recognized—bronchophony.

Increased Vocal Resonance.—This sign when heard over the lung—with rare exceptions, presently to be mentioned—denotes an increase in the power of the lung to conduct sound-producing vibrations,—namely, consolidation. It has, therefore, the same significance as bronchial respiration. Fully developed it constitutes bronchophony, and indicates consolidation of lung tissue in the neighborhood of large or medium-sized bronchial tubes. The following varieties are to be considered:

Pectoriloquy.—Laennec used this term to indicate the complete transmission of articulate words. The voice appears to be directly spoken into the observer's ear. This sign occurs in dense consolidation extending from a large bronchus to the wall of the chest, over a cavity communicating freely with a bronchus of some size, in a pneumothorax communicating with a bronchus, and in some instances over the atelectatic lung overlying a large pleural effusion. When pectoriloquy is very distinct and circumscribed it constitutes the distinct physical sign of a cavity, and, as Da Costa well said, deserves the name of cavernous voice.

Amphoric Vocal Resonance. — Over large cavities and in pneumothorax communicating with a bronchus the voice is peculiarly ringing and metallic. The amphoric character is due to the same physical conditions which we find to underlie the amphoric quality in the breath sounds and râles.

Whispering Pectoriloquy.—The whispered voice is heard as a faint, distant, expiratory whiff or puff over the trachea and primary bronchi in front and behind while elsewhere it is almost or quite inaudible. When the physical conditions which cause bronchophony are present, the whispered voice is curiously near and distinct. Whispering pectoriloquy is an important physical sign, indicating, when distinct and circumscribed, a cavity, and in varying degrees of intensity consolidation of lung tissue. It is of value in the diagnosis of limited areas of consolidation and in determining the boundaries of large ones. The more dense the consolidation the more distinct the whispered voice. Whispering pectoriloquy may be present over the atelectatic lung in pleural effusion and occasionally over the effusion itself. Increased whisper in the intrascapular regions—D'Espine's sign—occurs in tuberculosis of the bronchial glands.

Diminished Vocal Resonance. — This sign indicates impaired conduction in the lung and is present in emphysema and the occlusion of a bronchus. It also denotes the interposition of substances between the lung and the chest wall, which leads to the diffusion and weakening of vibrations passing from one medium to another, and occurs in pleural effusion, pleural thickening, and tumors. The more massive the effusion, the greater the thickening, or the larger the tumor, the more marked the diminution in the transmitted voice resonance. It may be completely absent in closed pneumothorax. Absent vocal resonance is most common in large pleural effusion.

Ægophony.—Literally, the bleating of a goat. A peculiar quavering quality of the voice with a distinctly nasal tone is heard when the patient speaks in a natural voice. This sign is best brought out by using repeated rather than single syllables, as "twenty-one" or "ninety-nine." It may be heard at or just below the upper limit of moderate-sized pleural effusions in the region of the angle of the scapula; less frequently in the front of the chest. It is in rare instances heard over consolidated lung tissue. It is not an important physical sign.

Baccelli's Sign.—Upon direct auscultation in the anterolateral region of the affected side the whispered voice is said to be distinctly transmitted through a serous but not through a purulent effusion, the difference being attributed to variations in the density of serofibrinous and purulent effusions. This sign is not constant, since in large effusions there is commonly absence of vocal resonance in both kinds of fluid.

Auscultation as Applied to the Diagnosis of Diseases of the Circulatory Organs.

The Technic.—This method is of cardinal importance in the examination of the heart. Upon it in most instances the diagnosis depends. Inspection, palpation, and percussion may be used to amplify and control the signs obtained by auscultation, but in a considerable proportion of the cases they contribute no essential facts. Before we apply the stethoscope, we inquire into the history of the case and place the patient as far as possible at his ease. The examination is best conducted when the patient is in a comfortable position, leaning back in a chair or propped up with pillows in bed. We note the facial expression, the appearance of the eyes, the state of the capillary circulation, the presence or absence of dropsical swellings, whether or not there is cough, the character of the respiration and any abnormal impulse or movement that may be present at the root of the neck or in the chest. The signs elicited upon inspection, palpation, and percussion are then ascertained. Finally we employ auscultation.

In women the breast is drawn aside and held by the patient herself or her nurse. In young children inspection and palpation should precede auscultation. Percussion is useless. Very often the auscultatory signs must be caught in the intervals of crying and struggling. Many difficulties may be overcome by tact and gentleness.

The increase in the frequency of the heart's action and the accompanying change in the character of the first sound that occur in nervous persons under examination (*le cœur medical*) must be borne in mind. A few minutes' chat upon indifferent subjects will usually cause the excited action to subside. If on the other hand the action of the heart is weak and the sounds too faint to be well studied, or there is a doubt as to the presence of a murmur, the patient should be asked, unless his general condition forbids, to take a series of very deep breaths, or quickly stoop and rise several times, or take a few brisk turns up and down the room. The increase in the force of the heart's action will often render the sounds distinct and dispel any doubt as to the presence of a murmur. In cases of acute disease or profound general or cardiac asthenia such diagnostic measures are strictly contraindicated.

Faint and distant sounds and obscure murmurs may become more audible if the patient leans slightly forward and to his left, thus bringing the heart under the influence of gravity into closer relation with the wall of the chest. It is important also to request the patient to stop breathing for a moment now and again during the course of the examination, since the breath sounds may mask the normal and abnormal

ASSOCIATION OF THE PHYSICAL SIGNS.

Inspection.	Palpation.	Percussion.	Auscultation.	Physical Condition.
Normal signs	Normal vocal fremitus	Clear	Vesicular murmur or its modifications; normal vocal resonance	Lung tissue normal or nearly so.
Negative	Vocal fremitus increased	Vesiculotympanitic	Bronchovesicular respiration; small mucous râles; vocal resonance increased	Lung tissue relaxed; loss of normal tension. Moderate atelectasis; œdema; deep congestion.
Diminished respiratory excursus on affected side or locally	Vocal fremitus increased	Dull	Bronchial respiration; increased vocal resonance; bronchophony	Lung tissue consolidated by compression, complete atelectasis or exudate; pneumonia; tuberculosis.
Diminished movement on affected side	Vocal fremitus diminished or absent	Flat	Absent respiration; sometimes distant bronchial breathing; absent voice; ægophony	Pleural effusion or tumor.
Respiratory movement restricted generally or locally	Vocal fremitus diminished	Vesiculotympanitic; tympanitic	Respiration feeble or cavernous; vocal resonance feeble or cavernous	Air increased within the vesicles; local or general emphysema or at particular points; cavities.
Diminished excursus over lesion	Vocal fremitus diminished	Amphoric; metallic; cracked-pot sound	Respiration amphoric or metallic; cavernous; amphoric or metallic voice; whispering pectoriloquy	Large cavity with elastic walls communicating with a bronchus.
Greatly diminished movement	Absent vocal fremitus	Tympanitic; metallic; amphoric	Respiration metallic, amphoric; vocal resonance the same	Air in pleural sac; open pneumothorax.
		Coin test	Absent respiration	Closed pneumothorax.
		Dulness	Absent respiration; faint metallic voice	Air under great tension.
Greatly diminished movement	Absent fremitus; succussion	Flatness below. Tympany. Amphoric resonance above	Respiration and vocal resonance as above. Succussion sound; metallic tinkling	Air and fluid in pleural sac; hydro-pyo-hæmo-pneumothorax.

sounds of a feeble heart. The heart sounds should also be studied with full held inspiration and forced expiration.

Auscultation has for its object the determination of the character, intensity, and rhythm of the normal heart sounds, and their modifications within the range of health, the recognition of modifications which transcend those limits, and the detection of abnormal or adventitious sounds.

THE SIGNS IN HEALTH.

The Normal Heart Sounds.—When the stethoscope is placed over the heart two sounds are heard. Of these one is found to correspond in time with the cardiac impulse, and the other to follow it after a short interval of silence. After a longer, but still short interval, these sounds are repeated in the same order. For this reason they are spoken of respectively as the first and second sound of the heart.

The Characters of the Sounds.—The first sound is not only comparatively long, but it is also low in pitch and muffled. The second sound, on the contrary, is comparatively short and is high pitched and clear. The two sounds are therefore in sharp contrast in regard to their duration, pitch, and quality. The respective characters of the two sounds may be roughly imitated by the repetition of the syllables "*ubb dup.*"

Causes of the First Sound. — The first sound is due to vibrations caused by the simultaneous tension of the mitral and tricuspid valves in closure, the muscular contraction of the ventricles and the vibration of the blood contained within the ventricles at the moment of systole.

The Cause of the Second Sound.—The second sound is due to the vibrations caused by the simultaneous closure of the semilunar valves of the pulmonary artery and the aorta at the beginning of the ventricular diastole.

A Third Sound of the Heart.—Gibson has recently described a wave in the jugular pulse in healthy young adults occurring after the closure of the semilunar valves and before the auricular contraction, and accompanied by a low-pitched, clear sound at the apex, more distinctly audible in the cardiac revolutions which occur in the intervals between expiration and inspiration than at any other stages of respiration. This sound is not easily appreciated and is only audible in a certain proportion of the diastolic periods. It corresponds in time to the first element of the reduplicated second sound heard only at the apex, long familiar to clinicians. The explanations of this sound are at present purely hypothetical.

The Cardiac Cycle or Revolution.—Each revolution of the heart consists of an auricular systole, the instantly succeeding ventricular systole, and a period of repose of the whole heart. The relative time occupied with these events varies with the frequency of the action of the heart. With a pulse-rate of 74, that is, a cardiac revolution of about 0.8 second, the cardiac revolution comprises an auricular systole of 0.1 second, a ventricular systole of 0.3 second, and a period of repose of the whole heart of 0.4 second. With increased pulse-frequency the diastole of the ventricles is shortened much more than the systole; it is also, with slowing of the pulse-rate, lengthened to a greater extent. The statements which assign regular proportions to the duration of the sounds and silences of the heart are misleading, since these vary in length, not relatively with varying heart frequency, but absolutely, the second silence being very fluctuating, since it corresponds to the fluctuating ventricular diastole, while the first sound and the short first silence together, which nearly correspond to the ventricular systole, are much more constant.

The Valve Areas or Puncta Maxima.—With the stethoscope applied over the apex of the heart the first sound is heard much more distinctly than the second sound and has a booming character which is in sharp contrast with the short and "valvular" quality of the latter. The temptation to rely upon the rhythm of the sounds for the recognition of the systolic sound or the first and second sounds is to be avoided. The auscultatory sign must be verified by inspection or palpation. This is especially important in the rapidly acting heart and in all morbid conditions. The systolic or first sound corresponds to the impulse as determined by sight or touch, or in default of these by the pulsation of the carotid. The radial pulse cannot be depended upon as a guide. The recognition of the first and second sounds is of especial importance in the diagnosis of valvular diseases.

When the stethoscope is carried to the base of the heart, either to the right or the left border of the sternum, the first sound becomes less distinct than at the apex while retaining its acoustic properties, and the second sound more distinct and prominent with an intensification of its snapping or valvular quality.

The sounds may be further analyzed by placing the stethoscope at the following principal points or areas:

1. **The Mitral Area.**—At or above the apex in the fifth intercostal space and upon the parasternal line. At this point that factor of the first sound made up by the closure of the mitral valve and the contraction of the left ventricle is best heard.

2. **The Tricuspid Area.**—At the juncture of the ensiform cartilage with the sternum and at the right border of the base of the sternum. In this region that factor of the first sound caused by the closure of the tricuspid valve and the contraction of the right ventricle is most distinctly heard.

3. **The Aortic Area.**—In the second right intercostal space near the sternum or directly over the second right costal cartilage at its sternal articulation—the aortic cartilage. At this point the aortic element of the second sound is best heard.

4. **The Pulmonary Area.**—In the second left intercostal space near the sternal border. At this point the pulmonary element of the second sound is best appreciated.

These areas do not correspond to the position of the respective valve systems, but they do correspond to the anatomical relationship to the wall of the chest of the structure in which the mechanism producing the sound exists, or in which the sound is conducted. That is to say, the anatomical apex of the heart formed by the left ventricle comes nearest to the chest at the apex; the tricuspid valve system at the right border and base of the sternum; the aorta just above its origin at the second right interspace, and the pulmonary artery above its valves at the second left interspace.

At the apex the first sound and its modifications in health and disease are best studied; at the base the second sound. In the former position this first sound is louder and more distinct; in the latter the rhythm is changed and the stress falls upon the second sound. The rhythm is the same in the mitral and the tricuspid areas and the quality of the first sound is similar, though in health the first sound is usually less intense in the tricuspid area. The rhythm is likewise the same in the aortic and the pulmonary areas, and the quality of the second sound is similar upon the right and left sides.

Modifications in the Normal Heart Sounds.—Variations in character, intensity, and rhythm are to be considered. There are marked differences in the sounds in different individuals and in the same individual at different periods of life and under varying conditions of activity and emotion.

Character.—The heart in children is less covered by the lungs than in later life and the chest wall is far thinner and more elastic. It follows that the sounds though feeble are more distinctly heard. As the muscle is smaller and thinner the valvular element of the first sound is more in evidence, and as the frequency is greater the long pause is shortened so that the rhythm, which at birth has the characteristic tic-tac of the fetal heart, like the ticking of a watch, only gradually changes to that above described as occurring in later life.

Embryocardia is a common condition in which the rhythm suggests that of the fetal heart, the long pause being shortened and the first and second sound presenting nearly the same acoustic properties. This modification of the cardiac rhythm occurs in tachycardia, the cardiac asthenia of the later periods of exhausting diseases and in extreme dilatation.

The first sound at the apex is not only somewhat louder in powerful persons with well-developed muscles but it is also more prolonged than in feeble persons who lead sedentary lives—a difference due to an increase of the muscular factor entering into the production of the sound.

A similar increase in the duration and intensity of the first sound occurs under conditions of bodily exercise and mental excitement. Under these circumstances the sound is occasionally attended by curious metallic reverberations, the *cliquetis metallique* of the French.

Intensity.—In young persons with thin, elastic chest walls the sounds of the heart are louder and more distinct than in older persons, in whom the walls are thicker and the costal cartilages more rigid. Thick layers of subcutaneous fat may render the sounds faint and distant. The interposition of the thick edge of a voluminous lung may have the same effect. There are marked differences in the intensity of the sounds in repose and activity.

THE FIRST SOUND AT THE APEX.—The first sound is louder and more distinct in the mitral area than in the tricuspid, but in young persons under conditions of excitement or after great muscular effort it may be heard with equal clearness and intensity over the whole front of the chest.

THE SECOND SOUND AT THE BASE.—The peculiarity of the second sound is its valvular quality. Its intensity varies in health with the energy of the heart's action. It has been assumed that the intensity of the aortic sound under normal conditions is greater than that of the pulmonary second sound. Vierordt, however, in 1885 first called attention to the fact that the relative intensity of these two components of the second sound varies at different periods of life, an observation that has been confirmed by other recent clinicians and especially by the investigations of Creighton in 1899. This observer found that in 90 per cent. of healthy children under ten years of age the pulmonic second sound is more intense than the aortic; in 66 per cent. between the tenth and twentieth years the pulmonic sound is more distinct; in about half in the following decade, and after the thirtieth year the proportion gradually declines until after sixty, when the aortic second sound is more intense—accentuated—in almost every case. It thus appears that the relative intensity of the two elements of the second sound depends upon the age of the individual, the sound in the pulmonary area being more intense in early, and that in the aortic area more intense in later life, while in middle life their intensity is much the same. Cabot suggests that "it is therefore far from true to suppose that we can obtain evidence of a pathological increase in the intensity of either of the sounds at the base of the heart simply by comparing it with the other." The difficulty lies in the failure on the part of the auscultator to recognize the difference between mere *loudness* or *intensity* which may be normal, and *accentuation*, which is a morbid physical sign.

In elderly persons the second sounds are frequently heard more distinctly in the third or fourth interspace than in the second.

Rhythm.—The derangements of rhythm which may occur in health are:

1. *Gallop Rhythm in which the Diastolic Pause is Shortened with the Addition of an Extra Sound of the Heart.*—The rhythm suggests the cadence of the footfall of a cantering horse. It is expressed by the repetition of the syllables "rat-ta-ta." The mechanism of its production is not clear.

G. Canby Robinson has summarized the results of recent studies of gallop rhythm as follows: "Gallop rhythm of the heart is a fairly frequent clinical phenomenon, and consists in the presence of a group of three cardiac tones, none of which are murmurs. It occurs under variable clinical conditions. That form of gallop rhythm which is best heard at the apex or over the central part of the precordium may be divided into the presystolic, protodiastolic, and mesodiastolic types, depending on whether the extra tone falls at the end, at the beginning, or in the middle of diastole. Each form is associated with a characteristic cardiogram. There are a number of factors which probably combine in various ways to produce the various forms of gallop rhythm.

"Presystolic gallop rhythm is heard in two classes of cases. It is heard in strongly acting hearts in which a muscle sound produced by a strongly acting, hypertrophied auricle is probably the cause of the extra tone; and it is also heard in weak, rapidly acting hearts at the height of acute febrile diseases, at which time there is possibly a delay in the conduction of the heart-beat from the auricles to the ventricles. Under these circumstances the sound produced during the contraction of the auricles becomes distinguishable from that produced during the contraction of the ventricles. In both classes of cases, the extra tone seems to be produced in the auricle rather than in the ventricle. Protodiastolic and mesodiastolic gallop rhythm are caused by the production of an extra tone in the ventricles. The factors that probably combine to produce this extra tone are an increase in the amount and velocity of the flow of blood from the auricles into the empty ventricles and a loss of tone of the heart muscle of the ventricles. The longer silent period in cases of gallop rhythm does not as a rule occur during diastole, but is usually a systolic silence."

This derangement of the cardiac rhythm may sometimes be observed in the normal heart when rapidly acting under conditions of great exertion or excitement.

2. *Reduplication of the Second Sound at the Base of the Heart.*—Splitting of the second sound may be heard at the base of the heart at the end of full inspiration, especially if the breath be held or after active muscular exertion. Its mechanism probably consists in the asynchronous closure of the aortic and pulmonary valve systems as the result of heightened pressure in the pulmonary circuit.

3. *Reduplication of the First Sound at the Apex.*—An impure first sound may occasionally be heard at the apex, especially at the end of expiration under normal circumstances. This modification varies from a mere blur or prolongation of the sound to a distinct repetition, constituting a form of the gallop rhythm. It may be represented by the syllables ."trupp" or "turrupp." In health it is not constant in the same individual. It has been attributed to conditions temporarily giving rise to an increase in the *vis-a-fronte* of one or the other ventricle.

MODIFICATIONS OF THE HEART SOUNDS IN DISEASE.

Variations in the character, intensity, and rhythm which transcend the borders of health, together with wholly abnormal or adventitious sounds, are to be considered.

Character.—The acoustic properties of the heart sounds are modified not only by changes in the heart itself and in the arteries but also by pathological conditions in the adjacent parts and the state of the chest walls as regards elasticity and thickness. Finally the character of the heart sounds is modified by constitutional conditions. Changes in character are commonly associated with changes in intensity, but it is well for the student to train himself to appreciate modifications of character and of intensity as constituting distinct groups of physical signs.

1. **The Heart.**—The first sound is prolonged and dull in hypertrophy; when the associated dilatation is marked it is sometimes very clear and sharp. A metallic clinking—*tintement metallique*—is occasionally heard to the right of the apex-beat. The second sound is loud and distinct, often ringing in character and doubled. When valvular lesions are present the sounds are greatly modified and replaced or accompanied by murmurs.

In hypertrophy of the right ventricle the first sound at the lower part of the sternum is louder and fuller than normal; but with much associated dilatation it is clearer and sharper. Accentuation of the pulmonary second sound is frequently present.

In dilatation the first sound is shorter and sharper, in other words, more valvular in character than normal. The muscular element is diminished. With progressive thinning of the walls these changes become more marked. The second sound when heard in aortic insufficiency may be distinct, or faint and obscure; when there is dilatation of the aortic arch it may be ringing and prolonged.

The second sound is rarely heard in the aortic area in aortic stenosis for the reason that the deformity of the cusps is such as to prevent their free play.

Accentuation of the pulmonary second sound is an important sign in mitral insufficiency.

The first sound is unusually sharp and clear in mitral stenosis, while the second sound in the second left interspace is strongly accentuated and sometimes reduplicated.

2. **The Arteries.** — Accentuation of the aortic second sound occurs in arteriosclerosis, — especially that form which accompanies chronic nephritis,—in atheroma and dilatation of the aortic arch, and in aortic aneurism. It is the sign of increased arterial tension and is associated with hypertrophy of the left ventricle.

3. **Diseases of Neighboring Organs.**—In pericardial effusion the heart sounds are not only indistinct but they also have a peculiar muffled and distant quality, due to diffusion. Accentuation of the pulmonary second sound is frequently an early and persistent sign.

In some cases of pneumothorax the heart sounds acquire a metallic quality; in pneumopericardium they are feeble, distant, and muffled.

They are distant and muffled in pulmonary emphysema, well transmitted in consolidation of the lung and in chronic interstitial pneumonia and pulmonary phthisis, and sharp and ringing during cardiac overaction, especially in young persons and in the periods of excitement and palpitation which occur in exophthalmic goitre, chlorosis, and anæmic states.

4. **Different Conditions of the Walls of the Chest.**—As in health so in disease, remarkable differences in the heart sounds occur as the result of differences in the chest wall. Through the thin and elastic tissues of the young the sounds are conducted with great distinctness; they are faint and diffuse when the chest walls are thick and fat, and when the cartilages are calcified, the sternum thickened, or when deformities of the chest derange the normal relation of the heart to the wall, or finally when a new growth is interposed.

5. **Constitutional Conditions.** — The first sound is shortened as well as faint in conditions of general asthenia such as result from actual starvation and wasting diseases. In enteric fever the first sound becomes progressively shorter, more indistinct and valvular in quality—a change due to the progressive wasting of the myocardium.

Intensity.—The significance of increase or decrease in the intensity of the heart sounds as morbid physical signs has already to some extent been indicated. It is important to note that as a rule increase in the intensity of the first sound is associated with its prolongation, while decrease in intensity is attended with decrease in duration. The loud first sound is in strong contrast with the short second sound; the faint first sound resembles it. As the feeble heart is commonly also a rapid heart, in which the long pause is shortened, it may become difficult to tell which is the first and which the second sound. The first sound corresponds to the impulse at the apex or to the carotid pulse.

Accentuation. — It is important at this point to emphasize the distinction between "loudness" and "accentuation"—a matter not always made clear in the books. Loudness or sound intensity has to do with the volume of a given sound; accentuation is that acoustic property which indicates suddenness in the application of the energy by which the sound is produced. The first sound of the heart is often loud, even booming, but never, according to my belief, accentuated. It may have a slapping quality as in mitral stenosis, but that is something altogether different from accentuation. The second sound of the heart at the base may be loud and distinct without being accentuated. It may become accentuated without becoming louder. Accentuation is then something quite different from loudness. The word conveys the idea of suddenness, sharpness, a certain vibrating quality due to quick and sharp tension. Loudness is a matter of degree; accentuation a matter of quality. From this point of view accentuation becomes a physical sign of great importance.

The first sound is increased in intensity in conditions which cause the heart to act with unusual energy. In intense emotional states the first sound is greatly increased and may sometimes be heard all over the chest. Such overaction may be pathological, as in mania and acute febrile states. The first sound is louder than normal in hypertrophy of the left ventricle, but less constantly so than has been assumed; even with a considerable degree of associated dilatation the sound may still be quite intense.

The first sound is enfeebled in conditions of general asthenia such as result from starvation, long-continued fevers, wasting diseases, hemorrhage, shock, and profound exhaustion from over-exertion; in dilatation of the ventricles, myocarditis, fatty heart, and rupture of the compensation in chronic valvular disease; in chlorosis and anæmia and in all conditions that interfere with its transmission to the ear of the auscultator, such as fat in the chest walls, emphysema, pleural and pericardial effusions, and certain mediastinal tumors. In conditions in which direct pressure is exerted upon the wall of the heart by effusion or tumor, its action is impeded and its sound enfeebled.

The second sound is increased in intensity in nervous overaction of the heart and in all conditions in which the lungs are retracted so as to bring the aortic arch and the conus arteriosus into more extended relation with the wall of the thorax. An apparent increase in the loudness of one or the other elements of the second sound is produced by the retraction of the anterior border of the lung upon the corresponding side. The second sound is diminished in intensity by those conditions, both general and cardiac, which weaken the action of the heart and diminish the intensity of the first sound.

The significance of changes in the intensity of the aortic and pulmonary elements in the second sound demands consideration.

It has already been pointed out that in normal individuals after middle life the aortic second sound is more intense than the pulmonary. A mere increase in the volume of the sound may be the result of increased cardiac action. An increase associated with that change of quality designated by the term accentuation constitutes a morbid physical sign and becomes more significant in proportion as the accentuation becomes more marked.

Accentuation of the aortic second sound occurs in all conditions in which the arterial blood-pressure — *vis-a-fronte* — is increased, namely, arteriosclerosis, chronic nephritis, and in aortic aneurism and dilatation of the aortic arch. In conditions characterized by habitual increase in arterial tension there is usually cardiac hypertrophy.

Diminution in the intensity of the aortic second sound occurs in conditions in which the blood thrown into the aorta by the ventricular systole is reduced in amount as in aortic and mitral stenosis and to some degree also in mitral insufficiency. Under these circumstances the aortic second sound may be so diminished as to be no longer heard at the apex. Weakening of the wall of the heart, as in fibrous and interstitial myocarditis, fatty degeneration, and extreme dilatation, likewise gives rise to enfeeblement of the aortic second sound. Relaxation of the peripheral arteries produces the same effect. The aortic second sound is extremely faint in collapse from any cause.

The pulmonic second sound is louder than the aortic in children and in young adults. A pathological increase in the loudness of this sound has the same significance in regard to the pulmonary circulation that an increase in the aortic second sound has in regard to the general circulation, namely, an augmentation in the resistance to the flow of the blood. This occurs in chronic valvular disease of the heart, especially in mitral stenosis and insufficiency, and in various pulmonary diseases, particularly emphysema, chronic bronchitis, phthisis, interstitial pneumonia, and compression atelectasis. These conditions are associated with hypertrophy of the right ventricle, compensatory in nature; when the compensation fails, the pulmonary second sound becomes faint and indistinct. Under all these conditions the more intense pulmonary second sound is also accentuated.

Weakening of the pulmonary second sound is the sign of a weakened right ventricle or tricuspid insufficiency. This sign is of great value in pneumonia as indicating failure of the right ventricle. The pulmonic second sound should therefore be systematically studied, since it affords at once indications for treatment and data for prognosis.

Rhythm.—Allorrhythmia is the general term used to designate deviations from the normal rhythm of the heart. See Arrhythmia, p. 468 *et seq.* under Radial Pulse.

Theories of the Mechanism of the Heart.—The neurogenic or older theory was based upon the assumption that the rhythmic contractions of the heart muscle are due to stimuli originating in the nervous system, either in the motor cells of the brain or, as modified by Volkman, in the cardiac ganglia. The myogenic theory, which we owe to the brilliant work of Gaskell and Englemann, is of recent origin and ascribes the contractions to automatic impulses originating in and conducted by the muscle. The subsequent studies of His, Keith, Tawara, Erlanger and others have shown that these properties are possessed not by the myocardium as a whole but by certain highly specialized muscle fibres embryonic in type, which differ in structure from the muscle fibres of the adult heart and are present in the remains of the primitive cardiac tube. These muscle fibres constitute a system beginning at the junction of the superior vena cava and the auricle as a small net-like mass recently described by Keith and Flask and now regarded as the seat of the primary impulses, continued in the right auricle as the atrio-ventricular or Tawara's node and passing thence into the septum and branching into both ventricles as the bundle of His. The myogenic theory during the short period since it was first formulated has aroused widespread interest among anatomists, physiologists and clinicians and been singularly productive in its influence upon biological investigation and practical medicine. Among other matters it has brought about an orderly classification of the cardiac arrhythmias and shed much light upon their significance.

Properties of the Heart Muscle.—The studies of Gaskell and Englemann have shown the following properties to be inherent in the myocardium and to constitute the basis of the physiology and clinical pathology of the heart: 1. *Stimulus production*—the capability of the heart to generate automatic rhythmic impulses. 2. *Excitability*—the ability to respond to an adequate stimulus by contraction. 3. *Conductivity*—the power of transmitting the impulse from one place in the heart to another. 4. *Contractility*—the capacity to react to more favorable conditions, in respect of rest, nutrition, temperature and so on, with stronger contractions. 5. *Tonicity*—the degree of contraction which the heart maintains during diastole, by virtue of which the total volume of the organ and the size of the cavities are less than if full muscular relaxation were to occur in diastole.

Two other properties of the myocardium are essential to the myogenic theory. The first is known as the *All or None Law.* It is the formulation of the fact that while a skeletal muscle reacts in proportion to the vigor of the stimulus, the heart has only one kind of response to all degrees of stimulation. A stimulus barely strong enough to cause a contraction will cause the most powerful contractions of which the heart is capable under given conditions at the moment. No increase of stimulation will increase the force of the contraction.

The second is the *Absence of Tetanus.* A skeletal muscle reacts to continuous rapid stimulation by continuous contraction, whereas the vertebrate heart responds with a single contraction and to continuous stimulation by a series of distinctly separate contractions. This is attributed to the refractory period, the reaction to the stimulus failing to occur until the effect of the previous contraction has ceased.

The influence of the vagus and the accelerators upon the action of the heart is important in connection with the myogenic theory. The rate of beats, the conductivity, the excitability and contractility are decreased by the influence of the vagus fibres and increased by that of the accelerator fibres. Englemann holds that there are separate nerve fibres which influence each of those functions and designates those which regulate the rate chronotropic; those which influence the conductivity dromotropic; those which modify the irritability bathmotropic and those which act upon the contractility inotropic. Finally, the influence of the elasticity and contractility of the blood-vessels upon the heart through the action of the vasomotor system enters largely into pathological considerations and has an important bearing upon diagnosis.

thods.—Formerly the arterial pulse and apex beat were the guides study of the cardiac rhythm. The sphymograph and cardiograph omething to our knowledge but less than was hoped. Auscultation d the sounds and rs which attended inning and the end ventricular systole ognizes ventricular tions which fail to he superficial arte- pulse beats. The f all of these meth- that they give no e sign of the condi- the auricles. Trac- the jugular pulse he condition of the uricle. The poly- ells the whole story contractions of cles and ventricles, he sphygmogram and cardiogram supply definite data in regard pening of the aortic valve system. That this story has been fully ally interpreted cannot be said.

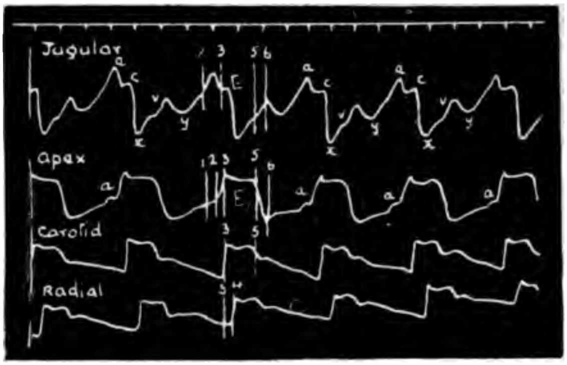

FIG. 81a.—Tracings of the jugular pulse beat, carotid and radial pulses. The perpendicular lines represent the time of the following events: 1, the beginning of the auricular systole; 2, the beginning of the ventricular systole; 3, the appearance of the pulse in the carotid; 4, the appearance of the pulse in the radial; 5, the closing of the semilunar valves; 6, the opening of the tricuspid valves. (MacKenzie.)

ABNORMAL OR ADVENTITIOUS SOUNDS.

on auscultation over the heart and great vessels sounds are heard in gical conditions which differ from the normal heart sounds. These bear a definite relation to the cardiac cycle and are dependent upon ion of the heart. Those which have their origin within the heart are of as endocardial, those which arise outside of the heart as exocardial.

Endocardial adventitious sounds are called murmurs. They are:

Organic;	Functional, Accidental or Hæmic.

Exocardial adventitious sounds, sometimes called pericardial murmurs, include:

Pericardial Friction;	The Precordial Râles of Emphysema;
Pleuropericardial Friction;	Pericardial Splashing;
Cardiopulmonary Murmurs;	The Murmurs of Aneurism.

Endocardial Murmurs.—Much confusion has arisen from attempts in auscultatory phenomena in musical terms. Neither the sounds of the sometimes erroneously called "tones"—nor cardiac murmurs, with ons presently to be mentioned, are musical phenomena. They both om irregular sound-producing vibrations which lack, as a rule, the y necessary to the production of musical tones. A "sound" of the produced by a single sudden derangement of the equipoise of sounding structures, which are thrown into vibration; a murmur by the con- action of forces which maintain such vibrations. The sound presently ceases; the murmur continues so long as the force which causes the vibrations continues to act. The sound corresponds in a way to a single blow upon a drum; the murmur to the continuous, rapidly repeated, but less intense sounds known as the roll of the drum; or the sound to the picking of the violin string, the murmur to the continuous note made by the drawing of the bow. But both these comparisons have the fault of likening musical phenomena to those which usually lack the musical quality. Furthermore the mechanism by which sounds and murmurs are produced is different.

The Mechanism of Endocardial Murmurs.—The heart sounds arise from the contraction of the heart muscle, the vibration of the blood mass, and the sudden tension of the auriculoventricular and semilunar valve systems. When murmurs arise a new set of physical conditions comes into play, namely, fluid veins (see p. 152). These swirls, or currents within currents of the blood, are attended with vibrations, which, first communicated to the wall of the heart or vessels and thence by way of the intervening tissues to the surface of the chest, are recognized by the auscultator as auditory phenomena—murmurs.

The Mechanism of Organic Murmurs—Lesions.—In by far the greater number of instances the fluid veins are due to actual lesions of the heart, and for this reason the murmurs are known as organic. The lesions mostly involve the valves, a fact which is indicated by the descriptive adjective valvular. They are on the one hand inflammatory and proliferative or adhesive, on the other sclerotic. Those that occur in early life are usually inflammatory; those which develop later are mostly sclerotic; but the inflammatory lesions of the valves undergo sclerotic changes, and old sclerotic valves are frequently the seat of recurrent inflammatory processes—recurrent endocarditis. As the result of each of these processes involving the valves, deformities arise. Inflammation causes vegetations, thickening, adhesions, and in extreme cases necrosis; sclerosis gives rise to thickening, retraction, crumpling; both result in loss of elasticity and freedom of movement. In cases of long standing lime salts are deposited and the rigidity and deformity are correspondingly increased.

STENOSIS AND INSUFFICIENCY.—The impairment of function is twofold. That function of the valves by which they yield before the blood stream and permit it to pass unhindered from auricle to ventricle or from ventricle to artery may be deranged. The condition is known as stenosis or narrowing, and the fluid veins are developed in the normal direction of the blood stream. Or that function by virtue of which the valves close their respective orifices is at fault, and there is valvular insufficiency or incompetency, the fluid veins developing in the reverse direction. Very often both these functions are impaired, and the condition is that of combined stenosis and insufficiency, with double murmurs.

RELATIVE INSUFFICIENCY. — Again, the orifice guarded by a valve system may be enlarged in consequence of the dilatation of the heart, so that the edges of the valves may be unable to meet and close it. This condition is known as relative insufficiency or incompetence, and is dependent not upon lesions of the valves, but upon nutritive or degenerative lesions of the heart muscle. Acute relative insufficiency such as sometimes accompanies the heart failure of violent exertion is due to relaxation of the wall of the heart and papillary muscles.

Roughening of the surfaces of the valves or of the parts immediately adjacent to them and sudden dilatation of the artery just beyond the valve system may lead to the production of a murmur.

The deformity which gives rise to an endocardial murmur may be of all degrees, from such as only slightly impair the function of the valve system to a stenosis which leaves a tiny orifice or mere chink for the passage of the blood or an incompetence that is almost complete and transfers the pressure of the blood column to the wall of the chamber of the heart which is immediately behind the defective valve, namely, the left ventricle in aortic insufficiency and the left auricle in mitral insufficiency. A projecting firm vegetation or rigid spicule or the inelastic edge of a sclerotic valve may be the cause of a systolic murmur, where there is practically no actual narrowing of the orifice. One of the first lessons for the student of heart murmurs to learn is that by no means every systolic murmur having its point of maximum intensity in the aortic area is the sign of aortic stenosis.

STENOSIS OR NARROWING OF AN ORIFICE GUARDED BY A VALVE SYSTEM.—There is impairment of the function by which the valves open at the physiological moment. The flow of the blood is obstructed and under ordinary circumstances a murmur is produced, which is spoken of as an obstructive murmur. If the heart be very feeble, marked obstruction may exist without producing a murmur that can be recognized. If the left auriculoventricular orifice is involved, the condition is known as mitral stenosis or obstruction; if the aortic, as aortic stenosis or obstruction.

INCOMPETENCE OR INSUFFICIENCY.—The function of the valves by which they close the orifice is impaired and a portion of the blood which has just passed through the orifice escapes from the main stream and flows back into the chamber of the heart whence it came. This pathological event is known as regurgitation, and the murmur which attends it is called a regurgitant murmur. We then have mitral and aortic incompetence, insufficiency or regurgitation as one or the other of these valve systems is affected.

Valvular lesions of the right side of the heart are of infrequent occurrence. They are sometimes the result of developmental defects or prenatal endocarditis. However produced they cause similar impairment of the valve functions, manifest by murmurs—tricuspid and pulmonary stenosis and incompetence. Stenosis is always due to deformity of the segments of a valve system. Incompetence is mostly due to the same cause, but not always. The deformity which prevents a valve from fully opening also generally prevents it from fully closing.

COMBINED STENOSIS AND INCOMPETENCE arises under the conditions just indicated. The lesion is a "double" one and manifests itself by a "double" or "to-and-fro" murmur.

Incompetence may, however, arise in the absence of stenosis as the result of (a) a lesion by which a valve segment has been destroyed by ulcerative endocarditis or has contracted adhesions to the wall of the heart, or (b) of relaxation of the cardiac muscle, as in relative insufficiency.

Stenosis without incompetence is comparatively infrequent; incompetence without stenosis is not very uncommon.

Valvular lesions exert their effect (a) upon the blood stream within the heart, (b) upon the walls of the heart, (c) upon the viscera, and finally (d) upon the peripheral circulation.

(a) The Effect of the Valvular Lesions which Produce Endocardial Murmurs upon the Blood Stream within the Heart.—The beginning of evil in stenosis and incompetence is the same. It consists in a reduction of the quantity of blood which eventually passes the diseased valve system with each revolution of the heart. In stenosis a portion of the stream corresponding to the extent of the pathological barrier is held back; in incompetence a portion corresponding to the degree of the pathological defect returns into the chamber whence it came—regurgitates. The result is a tendency to retardation of the flow, diminution in the volume of blood entering the arteries, and increase in the volume retained in the veins, with progressive transference of blood-pressure from the arterial to the venous side of the circulation. Were this tendency unchecked every case of valvular disease would in a short time terminate in death, the venous pressure rising and the arterial falling until the circulation becomes no longer possible. This result, which is the usual cause of death in valvular disease, is postponed for an indefinite period by compensatory changes in the muscle of the heart itself. It is true these changes are consecutive to the lesion, but as the latter is progressive, the former are correspondingly progressive. When the one advances at the same rate as the other a physiological balance is again established, the stability of which depends upon the tardiness of the valvular disease on the one hand, and the ability of the hypertrophied heart muscle to maintain its nutrition on the other. When extensive valvular defects develop suddenly or are rapidly progressive, compensation is not established and death occurs in a short time.

(b) The Effects upon the Walls of the Heart.—The immediate effects of the separation of the blood stream into a major part circulating under physiological conditions and a minor part held back under pathological influences are exerted upon the walls of the chamber behind the affected valve system. They are first dilatation, then hypertrophy. These changes may affect the whole organ, the heart acting as a single muscle and undergoing a general enlargement in response to the increased work required of it; more commonly they affect one or more of the chambers and in particular that chamber immediately subjected by the valvular lesion to increase in its blood contents in diastole and to a necessary increase in its energy in systole in order to overcome the obstacle in stenosis or propel an augmented volume of blood in incompetence. In mitral stenosis the left auricle cannot empty itself and receives blood from the pulmonary circuit; in mitral incompetence it receives blood at the same moment from the pulmonary circuit and the left ventricle; in combined mitral lesions some blood is retained and some regurgitates, while the physiological supply enters by the pulmonary veins. Consequently the left auricle is first dilated and then hypertrophied. In aortic stenosis the left ventricle cannot empty itself and in diastole the blood received from the left auricle is augmented by that retained at the time of the previous systole; in aortic incompetence blood enters the left ventricle in diastole the same moment from two sources, the left auricle and the aorta; combined aortic disease the blood coming from the left auricle is gmented by that retained at the previous systole and that returning om the aorta. Under these circumstances the left ventricle is first dilated d then hypertrophied. It is important to bear in mind the cardinal ct that all these changes, like the lesion itself, are slight at first and adually progress, and that in the early stages neither the dilatation or the hypertrophy can be recognized by the ordinary methods of physical iagnosis. Even at this period the murmurs indicative of the respective sions are commonly quite audible, often loud or harsh, and months ay elapse before the signs of enlargement of the heart or venous stasis n be determined. The diagnosis rests upon the presence and characters the murmur. Nevertheless it is an error to speak of this stage as pre-ding compensation. There are, however, cases of rapidly developing sions in which complete compensation is only gradually attained; some which it is never reached. If the quantity of blood held back in stenosis regurgitated in incompetence be represented by x, it is evident that ere must be an increased capacity of the affected chamber, represented y the same symbol, and that the chamber must be dilated to that tent. While if the resulting hypertrophy of the wall of the chamber be ch as to enable it to propel the normal quantity of blood plus x, it is vident that a condition is established in respect to the volume of blood aintained in circulation, which is practically normal despite the valvular sion, and this condition is known as compensation. This condition exists, owever, by virtue of an abnormal increase in the nutrition and work of he heart muscle and at the expense of the normal reserve power of the eart, and is therefore unstable. It consists in a degree of dilatation and ypertrophy combined and in ratio to the valvular defect, but demands r its maintenance a hypertrophy slightly in excess of the dilatation. The utrition of the overgrown and overworked muscle ultimately fails and ilatation develops in excess of hypertrophy. The compensation under hese circumstances is said to be at first "deranged" or "failing," later broken" or "ruptured." It is a question of degree. In a small proportion f the cases failure of compensation occurs in the absence of marked crease in the size of the affected chamber or chambers of the heart nd has been ascribed to derangement of the innervation of the heart.

(c) Effects upon the Viscera.—Compensation, while adequate to e maintenance of a fair degree of health for an indefinite period, is never omplete. There is always increased resistance to the onward flow of the rterial blood and a corresponding increase in the blood-pressure upon the enous side of the circulation. This results in increased fulness of the ulmonary circuit, manifest by accentuation of the pulmonary second und, hypertrophy of the right ventricle and a *tendency* to passive hyper-mia of the viscera in general; hence, accentuation of the aortic second und, dyspnœa upon exertion, a peculiar liability to bronchial catarrh nd the occasional occurrence of blood-streaked sputum or slight hæmop-sis—phenomena which are common in mitral disease even while the mpensation remains fairly good. Upon failure of compensation there is arked venous engorgement of the viscera, with grave derangement of function, shown on the part of the lungs by marked dyspnœa or orthopnea, cyanosis, cough, and the occasional expectoration of frothy blood; of the liver and gastro-intestinal organs by loss of appetite, deficient digestion, nausea, slight jaundice and constipation; on the part of the kidneys by scanty urine and albuminuria. Dulness, stupor, somnolence with inability to sleep are symptoms of the derangement of the cerebral circulation.

(d) EFFECTS UPON THE PERIPHERAL CIRCULATION.—While compensation is maintained, dropsy, as the result of valvular lesions, does not occur. There is sometimes to be found slight pretibial œdema, especially after fatiguing exertion or long standing. When compensation fails, however, the diminished arterial pressure and the increased venous pressure interfere with the circulation of the blood in the capillary zone and give rise to œdema. Under these circumstances, there is an accumulation of extravascular serum about the capillaries and a retardation of the lymph-flow. Hence the visceral derangements are not only hyperæmic, they are also œdematous. The action of gravity renders this accumulation of extravascular fluid early manifest in the dependent parts, namely, the legs and feet. As it increases, the thighs, genitalia, and loins become involved, and finally there is general œdema with effusion into the serous sacs. The dropsy of heart disease is often irregularly distributed, but its presence in particular localities may usually be explained by the relatively loose arrangement of the subcutaneous or other tissues involved, postural influences, and the action of gravity.

Compensation in mitral disease commonly fails by degrees, with periods of improvement following rest and treatment, and the ultimate catastrophe usually occurs after impaired health of prolonged duration.

Compensation in aortic disease is chiefly maintained by the left ventricle, which often becomes enormously hypertrophied,—*cor bovinum.* There is some increase in the venous pressure, since the ventricle receives its blood in diastole not only from the auricle but also from the aorta, but so long as the mitral valve remains competent, the visceral engorgement and general œdema which characterize the dyscrasia of the *stadium ultimum* of mitral disease do not occur. Precordial pain, angina pectoris, and momentary faintness upon rising or at stool are common, and in many cases the rupture of compensation is immediate and instantly fatal, death occurring with the heart in asystole.

The Mechanism of Functional, Accidental, or Hæmic Murmurs. — The murmurs designated by these terms are not signs of disease of the valves or orifices of the heart. The frequency of their occurrence enables us to determine with precision that they do not correspond to anatomical changes in the organ found upon examination after death. The mechanism by which they are produced has been the subject of much controversy, but none of the explanations advanced has been generally accepted. Functional murmurs are almost exclusively systolic and are heard over a limited space in the pulmonary area. They have been ascribed to dilatation of the conus arteriosus, to the fact that in anæmia and similar conditions there is lowered tonicity of the arterial walls which undergo an abnormal dilatation at the time of the systole, and to the lowered blood-pressure of anæmia in the aorta and pulmonary artery, which, in connection with the relatively high intraventricular pressure at the beginning of systole, prevents the closure of the semilunar valves and the systolic tension of the vessels, with the result that a systolic murmur arises upon the passage of the blood into the large vessels at the moment of physiological stenosis—contraction of the cardiac sphincters.

Diastolic functional murmurs, which are so rare that they scarcely demand consideration for diagnostic purposes, are probably venous murmurs transmitted to the innominata or vena cava and heard at the base of the heart.

The Significance of Endocardial Murmurs.—In order to determine the diagnostic meaning of a murmur heard upon auscultation of the precordial area, we must first answer the following questions: Is it endocardial or exocardial? (See page 177.) If endocardial, is it organic or functional? (See page 188.) Having found it to be an endocardial murmur of organic origin we must then ascertain (1) its time in the cardiac cycle; (2) its point of maximum intensity; (3) the direction in which it is propagated and the area over which it can be heard; (4) its relation to the normal sounds of the heart; (5) its acoustic properties, and (6) the effects of exercise, respiration, and posture upon it.

(1) THE TIME OF MURMURS IN THE CARDIAC CYCLE.—The determination of this point is of primary importance. For diagnostic purposes the systole may be regarded as lasting from the beginning of the first sound of the heart until the second sound; the diastole from the beginning of the second sound until the beginning of the first sound in the next revolution of the heart. A murmur heard at the time of the first sound or replacing the first sound or extending into or developing in the period between the first and the second sounds is *systolic*. Murmurs which develop in the latter period are designated *late systolic*.

A murmur which occurs at the time of the second sound or replaces it is *diastolic*. Murmurs which occur during the last portion of the diastole and run up to the first sound are known as *presystolic*.

When the heart is acting moderately there is no difficulty in recognizing the first and second sounds by their respective characters, and the long and short silences by their relative duration. But when the heart is rapid the different acoustic characters of the two sounds cannot always be made out and the rhythm is so deranged that the difference between the long and the short silence is less marked. Under these circumstances the systole may be determined by palpation with the finger over the apex or the carotid artery during auscultation, or by inspection if the double stethoscope is used. The interval between the time of the cardiac impulse and the radial pulse renders the latter a misleading guide for this purpose.

(2) THE POINT OF MAXIMUM INTENSITY OF MURMURS.—The area in which a murmur is best heard is likewise of cardinal importance in diagnosis. The murmur is loudest at the point of its production and is best transmitted in the direction of the blood stream in which the fluid veins which produce it are developed. It is in accordance with these laws that a murmur having its point of greatest intensity in the mitral area has its origin at the mitral valve. It is necessary in this connection to bear in mind the fact that the mitral area is not constant in the normal position

but that it shifts with displacement of the heart. In a limited proportion of cases of mitral disease this murmur is best heard to the right of the normal position of the impulse, and in rare instances at the left border of the sternum higher up, even as high as the punctum maximum of the pulmonary second sound.

It is also in accordance with the above laws that murmurs heard in the pulmonary area, namely, the second left intercostal space, have their origin in the conus arteriosus or at the pulmonary orifice; that murmurs having their maximum intensity at the right lower border of the sternum, at the level of the fourth and fifth intercostal spaces, or at the base of the ensiform cartilage are produced at the tricuspid orifice, and that murmurs whose maximum intensity is in or near the aortic area have their origin at the aortic orifice. Murmurs arising at this point very often, however, are best heard over the upper part of the body of the sternum near its left border or. less frequently, at the apex or over the lower part of the sternum—aortic insufficiency.

(3) THE PROPAGATION OF MURMURS AND THE EXTENT OF THE AREA IN WHICH THEY CAN BE HEARD.—Murmurs are very often heard over limited areas and transmitted in definite directions. This is especially but not exclusively true of the murmurs produced by lesions of single valve systems. Thus the murmur of mitral stenosis—the presystolic murmur—is heard over a circumscribed area just above the apex and is not propagated, while the systolic murmur of mitral incompetence is heard over a considerable area to the right of the apex and upward and is transmitted distinctly in the direction of the left axilla and to the back. On the other hand the systolic murmur of aortic stenosis is usually prolonged and loud, heard over an extended area and transmitted into the carotid and subclavian artery; it is in some instances heard at a distance from the chest. The diastolic murmur of aortic incompetence may also be heard over an extensive area of the chest both in front and behind. A murmur distinctly heard over two or more valve areas may be due to one or to several valve lesions. If it is systolic in time, it may be the sign of mitral insufficiency or of aortic stenosis, or the murmur may be a compound of two murmurs, each representing one of these lesions. The difficulties are greatly increased when there are to-and-fro murmurs representing double lesions—stenosis and incompetence—of the respective valves. A correct diagnosis rests upon the application, in the study of individual cases, of the knowledge, obtained by clinical experience and post-mortem examination, that the murmur produced by each valvular lesion has its characteristic point of maximum intensity and definite line of propagation along which its intensity gradually diminishes as the stethoscope is moved away from that point. A murmur which fulfils these requirements in regard to a particular valve area and line of propagation and is unaccompanied by any other murmur may be regarded as the sign of a lesion of that valve. When, however, two or more murmurs are heard which differ in their acoustic characters, as pitch, quality, and duration, and present each its point of maximum intensity, and are propagated respectively in different directions, a correct diagnosis can only be reached by the separate study of each as though it alone were present, the others being for the time being

disregarded. In this analytical study too much importance cannot be placed upon the differences in pitch and quality and the evidences of the influence of the lesions which cause the murmurs, upon the size of the heart, the viscera and the pulmonary and peripheral circulation. In complicated cases of cardiac disease the diagnosis cannot be made by an analysis of the murmurs alone. A systematic study of the associated physical signs is essential to success.

When several murmurs are present, it is best not to attempt an over-refined diagnosis in regard to the lesions which underlie all of them. We may be content when we have determined with accuracy the two which are most important, and we may be reconciled to our inability to satisfactorily do more than this by the knowledge that greater nicety of diagnosis, though it might gratify our technical ambition, is not demanded by the requirements of therapeutics and prognosis, and has been discredited by the experience of the post-mortem room.

In this connection it is important to call the attention of the student to the fact that the intensity of a murmur does not necessarily gradually and progressively diminish in its line of propagation, but may be modified by the presence of a viscus or the interposition of a new growth. Thus an aortic murmur may be distinctly heard in the aortic area and near the apex and only faintly in the intervening space. This phenomenon has been attributed to the interposition of the right ventricle, while the transmission of a mitral regurgitant in the direction of the left axilla may be abruptly interrupted by a pleural effusion or neoplasm.

(4) THE RELATION OF MURMURS TO THE SOUNDS OF THE HEART.—A murmur may accompany the sounds of the heart or may replace them. The systolic apex murmur of mitral incompetence wholly or in part replaces the first sound. The systolic basic murmur of aortic stenosis accompanies the first sound, but when compensation fails the first sound is greatly weakened, and with enfeeblement and dilatation of the ventricle or upon the supervention of relative mitral incompetence it may no longer be heard. In aortic stenosis the second sound is not often heard at the aortic cartilage, because the deformity of the valve usually prevents its closure. In aortic incompetence, the second sound may be well heard or it may be replaced by the murmur. In some cases it may be absent in the aortic area but heard over the carotid artery.

A murmur which accompanies a sound also follows it, since the time of the murmur is longer than that of the sound. A murmur may run up to a sound, as the presystolic murmur of mitral stenosis. In rare cases murmurs occur between the sounds. The persistence of the sound along with the murmur may be of favorable prognostic significance, as, for example, in aortic incompetence, where it indicates partial closure of the damaged valve cusps with corresponding preservation of function.

(5) THE ACOUSTIC PROPERTIES OF MURMURS.—Under this caption the (a) intensity, (b) quality, (c) pitch, and (d) duration of murmurs are to be considered.

(a) *Intensity.*—The intensity of cardiac murmurs is extremely variable. A murmur may be so loud that it may be heard at a distance of some feet, or so low as to be scarcely audible when the patient holds his breath.

Not infrequently a loud murmur is heard by the patient himself. Such very loud murmurs are rare. The intensity of a murmur is by no means proportionate to the gravity of the lesion by which it is produced. On the contrary, since its intensity depends upon the energy with which the blood is propelled through the affected orifice, that is, upon the compensation, a loud murmur is, other things being equal, more favorable than a faint one. As compensation fails, the murmur becomes fainter and it not infrequently happens that in patients coming under observation with greatly impaired compensation no murmur can be recognized upon careful auscultation, but after rest and suitable treatment have brought about inprovement in the general condition and in compensation a murmur appears which becomes more intense as the patient grows better. This is especially the case in mitral disease. There are, however, cases of acute rheumatic endocarditis, especially in children, and of malignant endocarditis in which the changes in the valvular lesions develop rapidly while the power of the myocardium is still maintained, in which increasing loudness of the murmur constitutes a most unfavorable sign. The intensity of an organic, endocardial murmur is important less from its degree at any one time than from its decrease or increase during the progress of the case.

A murmur is not usually of the same intensity during its brief course. In general it is louder at the beginning than at the end. Presystolic murmurs are, however, usually louder at the close. The cause of the increase in intensity is here due to the fact that the blood flows gently through the auriculoventricular orifice at the beginning of the ventricular diastole, but with increased force under the stress of the auricular contraction later.

(b) *Quality.*—Endocardial murmurs vary in quality from a soft blowing sound—bellows murmur, *souffle*—of little intensity, to a coarse, harsh, rasping sound of considerable loudness. In rare instances they are musical. The musical quality is usually manifest during a part of the murmur only, the remainder having the ordinary blowing or rasping character. The musical quality indicates an organic lesion, but does not enable us to define its nature and is without significance in prognosis except that it indicates a certain degree of power in the heart muscle. The presystolic murmurs which are produced by mitral and tricuspid stenosis and the "Flint murmur" of aortic insufficiency have a peculiar "rumbling" or "blubbering" quality not heard under other conditions. These murmurs have been compared to a short roll of the drum, but they are much less regular.

(c) *Pitch.*—This attribute of murmurs is also variable. Blowing murmurs of soft quality are commonly low pitched, while the coarser murmurs are often high in pitch: to this general statement the exception that the very coarse, blubbering presystolic murmurs are usually of low pitch. It is the high-pitched murmur that tends to assume the musical quality.

(d) *Duration.*—A murmur may occupy the whole period of the systole or the diastole or any part of either of these periods. The systolic murmur of mitral incompetence is sometimes prolonged, the diastolic murmur of aortic incompetence almost always so. Presystolic murmurs are of shorter duration. The length of murmurs is not of itself of prognostic significance

(e) *Variation in Murmurs.*—Rapid changes in the acoustic properties of murmurs, even their disappearance and reappearance, may occur in acute endocarditis when vegetations are forming upon the valve segments, and especially in the malignant form, in which the vegetations grow with rapidity upon the valves and adjacent walls and ulcerative lesions occur. Similar changes may occur in the course of acute endocarditis as the result of rupture of chordæ tendineæ or the formation of adhesions between valve segments or between a segment and the wall of the heart.

(6) EFFECTS OF EXERCISE, RESPIRATION, AND POSTURE UPON ENDOCARDIAL MURMURS.—Faint murmurs usually become more distinct upon moderate exercise, as walking rapidly two or three times across a room, or stooping and rising several times in succession, and these movements are sometimes necessary in the examination of doubtful cases. When, however, a murmur has grown indistinct or disappeared as the result of rupture of compensation, movement simply increases the cardiac dyscrasia.

Organic murmurs are usually more distinctly audible upon quiet breathing, or while respiration is momentarily suspended, and at the close of expiration, when a larger area of the heart is uncovered. In this respect they differ from functional murmurs, which are frequently best heard upon inspiration.

Posture exerts an important influence upon the intensity of certain murmurs. Systolic murmurs not heard in the upright position may be distinctly audible in recumbency; on the other hand, murmurs not heard in the recumbent posture may be recognized when, by the patient leaning forward, the heart is brought into closer relation with the wall of the thorax. Presystolic murmurs are sometimes much better heard in the erect than in the recumbent posture.

The Significance of Functional, Accidental, or Hæmic Murmurs.—A large proportion of endocardial murmurs, much larger than was formerly supposed, are not associated with anatomical cardiac lesions. Certainly murmurs are not rarely heard *intra vitam* in cases in which no corresponding valvular lesions are found *post mortem.* Systolic murmurs arising in conditions of cardiac asthenia from relaxation of the cardiac sphincter—relative incompetence—and having all the characters of incompetence from actual lesions at the mitral orifice, though often transient are not usually described as "functional." Short, whiffing, systolic murmurs, sometimes heard in the mitral area directly after violent or prolonged physical effort, are probably due to relative insufficiency resulting from acute dilatation. They disappear in the course of a little time.

Functional murmurs are almost always systolic in time. By far the greater number of them have their point of maximum intensity in the pulmonic area; occasionally only are they most distinctly heard in the aortic or mitral areas. They are commonly well heard to a little distance from the point of maximum intensity in all directions rapidly diminishing in loudness, and are not distinctly transmitted in a definite line, as is usual with organic murmurs. They are as a rule soft and blowing in character. A loud coarse murmur, whatever its other points of resemblance to functional murmurs, is likely to prove to be organic, especially when persistent. Functional murmurs are usually most distinct at the close of inspiration.

They are commonly transient and disappear when the condition with which they are associated improves. They are not associated with the signs of enlargement of the heart or with accentuation of the pulmonary second sound.

Functional murmurs are significant of the various forms of anæmia. For this reason they are spoken of as "hæmic murmurs." They occur in secondary anæmias, chlorosis, pernicious anæmia, leukæmia and Hodgkin's disease. A distinct, prolonged systolic murmur in the pulmonary area is common in chlorosis, and, in consequence of the retraction of the borders of the lungs, is frequently associated with a loud pulmonary second sound. In the *stadium ultimum* of pernicious anæmia the hæmic murmurs often disappear.

The differential diagnosis between organic and functional endocardial murmurs rests upon the following facts:

Organic murmurs occur at any period in the revolution of the heart; functional murmurs are practically always systolic. It becomes necessary, therefore, to contrast the characters of organic systolic murmurs with those of functional murmurs.

Systolic organic murmurs are usually well propagated in the case of mitral insufficiency toward the left axilla and to the back; in aortic stenosis, to the carotids and the subclavians, especially upon the right side. They are often soft and blowing, not rarely coarse and loud, sometimes musical. The point of maximum intensity corresponds to the respective mitral and aortic areas as above described (see page 169), and only in exceptional cases is to be located in the neighborhood of the pulmonary area. Organic murmurs, except in the case of relative insufficiency, are persistent, diminishing in intensity and ultimately disappearing only when the compensation fails and is finally ruptured. They are sooner or later associated with the signs of enlargement of the heart and increase of the blood-pressure in the veins, as accentuation of the pulmonic second sound, visceral engorgement and anasarca. The anamnesis commonly points to an acute infection, rheumatic fever, hard work and worry as causal factors.

Functional murmurs, on the other hand, are not propagated in definite directions; practically always soft and blowing, very exceptionally loud or coarse; never musical. Their point of maximum intensity is almost invariably in the pulmonic area. They are transient and not associated with the signs produced by the effects of valvular lesions, as manifest in retardation of a part of the blood stream; accentuation of the pulmonic second sound, enlargement or distention of the walls of the heart; visceral derangements—venous engorgement; or disorders of the peripheral circulation —dropsy. Anæmia is almost always present.

The rare diastolic functional murmur, so rare as to be unimportant in diagnosis, has been observed only in anæmia of very high grade and in association with a venous hum.

B. **Exocardial Adventitious Sounds.**—Morbid physical signs not having their origin within the heart are frequently heard upon auscultation in the precordial region. Important in themselves, they acquire additional diagnostic importance by reason of their occasional close resemblance to endocardial murmurs. Of these the following are the more important.

(a) **Pericardial Friction.** — The friction rub of dry or fibrinous peri-rditis is heard upon auscultation as a grazing, creaking, or rasping sound cupying some part of the time of the cardiac revolution. It is sometimes stolic, sometimes diastolic, often to-and-fro, but rarely corresponds to e systole or diastole as endocardial murmurs do. It occurs irregularly d with momentary interruptions, commonly changes in character and ne from one examination to another, and may appear, disappear, and pear again in the course of a few hours. Pericardial friction is usually st heard in the area of superficial cardiac dulness, but may be heard at y part of the precordial region and sometimes over the greater part it. In some instances it is confined to the base of the heart. It is always, wever, distinctly circumscribed and never propagated in any direction yond the borders of the heart. It conveys the impression of being pro-iced close to the ear and is intensified by the pressure of the stethoscope, iich also increases the pain which is present. It is apt also to be increased ien the patient inclines his body forward. It is little influenced by the spiratory movements, except that in some instances its area is slightly tended in expiration. Pericardial friction is frequently associated with docardial murmurs, the signs occurring as the manifestations of an dopericarditis or the pericarditis developing in an individual already e subject of chronic valvular disease. Under these circumstances the ction sound is usually more conspicuous than the endocardial murmur d at times may mask it altogether.

The differential diagnosis between an endocardial murmur and a ricardial friction rests upon a critical analysis of the signs in the light the history of the case.

(b) **Pleuropericardial Friction.**—Cases occasionally occur in which fibri-id exudate upon that part of the pleura which is in relation with the ricardium gives rise to a friction sound having the cardiac rhythm, the ughened pleural surfaces being moved in apposition to each other by the ovement of the heart. The differential diagnosis between pericardial d pleuropericardial friction rests upon the following facts:

The pleuropericardial friction is commonly heard in connection with friction sound having also the respiratory rhythm—pleural friction. is apt to be increased by forced respiratory movements and to be more stinct upon inspiration, whereas pericardial friction is best heard at the se of expiration. A positive diagnosis cannot always be made.

(c) **Cardiopulmonary Murmurs.**—Murmurs, hitherto known as cardio-spiratory, having the cardiac rhythm, are occasionally produced in e borders of the lung in relation with the heart by the traction or ilsion of the heart upon the lung tissue in systole or diastole. The mur-urs are pulmonary but not respiratory, and are due to the sudden dis-acement of a certain volume of air from a mass of lung tissue confined adhesions. They are most commonly heard near the apex of the heart id over the projection of the left lung which overlaps it, known as the igula; less often under the left clavicle or about the angle of the left apula. These murmurs are mostly systolic—traction murmurs; very rely diastolic—pulsion murmurs—and are heard over circumscribed eas. They are much influenced by active respiration and cough. They occur during inspiration and are scarcely, if at all, audible during expiration, a fact which is of importance in distinguishing them from endocardial murmurs, which are usually better heard when the breath is held in expiration and a larger cardiac surface left uncovered by the retracted lung. Cardiopulmonary murmurs have the soft, breezy quality of the vesicular murmur and suggest an inspiratory act broken by successive movements of the heart, which in point of fact they are. In rare instances these murmurs are accompanied by crepitant or subcrepitant râles. Their importance from the standpoint of the diagnostician consists in their superficial resemblance to endocardial murmurs, from which they may be differentiated without difficulty.

(d) **The Precordial Râles of Emphysema.**—In rare cases of emphysema, in consequence of the rupture of the walls of vesicles, air finds its way along the interstitial tissue to the root of the lung and thence to the connective tissue of the anterior mediastinum. The superficial cardiac dulness, if not previously obliterated by the borders of the emphysematous lung, disappears with weakening of the heart sounds and the occurrence of high-pitched metallic or crepitant râles which have the rhythm of the heart. These signs are to be differentiated by their acoustic properties from the tricuspid regurgitant murmurs, due to the dilatation of the right ventricle, so frequently heard in emphysema. They are also to be differentiated from the râles having the cardiac rhythm, which are heard in rare cases of infiltration of the lungs or cavity formation in the neighborhood of the heart, by the persistence in the latter of superficial cardiac dulness and the heart sounds, the character of the associated respiratory sounds, and by the fact of their occurrence in pulmonary emphysema.

(e) **Pericardial Splashing.**—In pneumohydro- or pneumopyo-pericardium there may be heard peculiar splashing sounds of metallic character similar to the succussion sounds of pneumohydrothorax, but having the cardiac rhythm. The heart sounds under these circumstances are usually feeble and distant. The cardiac dulness is in the recumbent posture replaced by an area of tympany, the borders of which shift with changes in the posture of the patient. These splashing sounds can under no circumstances be mistaken for murmurs, but they may closely resemble the splashing of the gastric contents sometimes produced by the movements of the heart, or that of pneumothorax or of a large vomica, from which they may, however, be distinguished by the gravity of the symptoms, the concomitant signs of pericardial perforation or inflammation, the examination of the patient when the stomach is empty, or a systematic routine examination of the lungs.

(f) **The Murmurs of Aneurisms.**—Aneurism of the thoracic aorta more commonly involves the ascending portion of the arch. On auscultation at the base of the heart or sometimes in a wide area there may be heard a systolic murmur, transmitted like the murmur of aortic stenosis in the direction of the aorta itself and the vessels of the neck. A diastolic murmur is sometimes also present. The latter is due to the reflux of blood into the sac, and may easily be mistaken for the murmur of aortic insufficiency with which it is not infrequently associated, as the manifestation of relative incompetence in consequence of the dilatation of the aorta or of valvular deformities resulting from sclerotic changes. The differential diagnosis rests upon the presence or absence of the signs of aneurism, as determined by systematic inspection, palpation, percussion, and the anamnesis.

SOUNDS HEARD OVER THE PERIPHERAL VESSELS.

Auscultation of the Arteries. Normal Conditions.—When the stethoscope is lightly placed over the larger arteries where they run superficially, sounds may be heard which correspond to the sounds of the heart. These sounds are produced (a) in the heart and (b) in the arteries themselves.

The carotid may be studied at the angle of the jaw or at the inner border of the sternocleidomastoid muscle; the subclavian directly above the clavicle and external to the sternocleido muscle or directly below it, in the arm between the pectoralis major and the deltoid; the brachial at the inner border of the biceps or at the bend of the elbow, the arm being slightly flexed; the radial just above the wrist, and the femoral immediately below Poupart's ligament.

(a) The normal heart sounds are transmitted to some distance along the course of the main arteries at the root of the neck and may be heard in adults over the carotids and subclavians, more distinctly upon the right than upon the left side. In infancy and childhood only the second sound is thus transmitted, the first being very indistinct or wholly inaudible.

(b) A systolic sound, due to the sudden tension of the arterial walls, may in some cases be heard over the abdominal aorta and the femorals. In the majority of cases no sound is heard over these vessels nor over the smaller superficial arteries so long as the stethoscope is applied without pressure. When, however, some degree of pressure is exerted upon the wall of the artery by the rim of the stethoscope, a systolic murmur is produced, often intense and high-pitched, the so-called *compression murmur.* This murmur is due to sudden narrowing of the lumen of the vessel at the point of pressure with the production of fluid veins. If the pressure is increased to such a degree as to obliterate the lumen of the artery a systolic sound is produced by the increased force of the tension of the arterial wall—*pressure sound.* These phenomena are physiological and without other clinical significance than that which attends the risk of attaching erroneous importance to them.

(c) *Intracranial Murmurs.*—In children up to the sixth year there is sometimes to be heard upon auscultation over the cranium, and especially over the anterior fontanelle and in the parietal regions, a distinct systolic murmur, which apparently originates in the internal carotids from some unknown cause and is without diagnostic significance.

(d) *The Uterine Souffle.*—A soft blowing systolic murmur is heard over the pregnant uterus. It is first heard about the end of the sixteenth week and increases in frequency until the eighth month, after which it remains stationary. This murmur is subject to great variation as regards quality, intensity, rhythm, and point of maximum intensity. It is usually most distinct low down and upon one or the other side of the uterus, sometimes at the fundus, but very rarely over the entire uterine body. It is attributed to the circulation of the blood in the arteries of the uterine wall. The diagnostic importance of this sign is impaired by the fact that a similar murmur is occasionally heard in chronic metritis, uterine myomata, and ovarian cysts.

Single or double murmurs corresponding in time to the fetal heart-beats are sometimes recognized in auscultation in pregnancy. They have in some instances been found to be associated with defects of development or endocardial lesions of the fetal heart—*cardiac souffle.* In other cases murmurs have originated in the umbilical cord—*funic murmurs.*

Auscultation of the Arteries. Pathological Conditions.—(a) It is obvious that abnormal sounds—murmurs—heard in the aorta will be transmitted into the carotids and subclavians. The systolic and less intensely the diastolic murmurs of lesions of the aortic valve system are transmitted along the course of these vessels.

(b) In any condition in which the pulse is quick—*pulsus celer*—the arteries may yield upon auscultation a systolic sound. This sign is sometimes present in fever and is common in aortic insufficiency, and may be heard over the radials as well as over arteries of larger calibre. In aortic insufficiency of high grade a double sound is sometimes heard over the femorals, the systolic dilatation and the diastolic contraction of the artery being alike attended with an audible sound. Systolic and diastolic sounds in the femoral artery have also been observed in pregnancy and in chronic lead poisoning.

(c) *Double Murmurs in the Arteries. Duroziez's Murmurs.*—In well-marked cases of expansile pulse in which the blood wave rises rapidly and rapidly recedes there may frequently be detected over the femoral or brachial artery at a certain point in the gradually increased pressure of the stethoscope a double—namely, systolic and diastolic—murmur. Some care is necessary to exert the degree of pressure under which this sign is best heard. It may be observed in aortic insufficiency, chlorosis, and other conditions in which there is well-marked *pulsus celer.*

(d) *Subclavian Murmurs.*—Systolic murmurs occurring independently of pressure by the stethoscope are common. Heard upon one side only when the attitude of the patient is unconstrained and the arms hanging at the sides, such a murmur is very suggestive of apex disease of the lung with pleural adhesions implicating the artery in its course. They are commonly louder upon inspiration, exceptionally upon expiration. Such murmurs are occasionally to be heard upon one or both sides in normal individuals, and there are those who are able to produce them at will by assuming certain attitudes, with fixation of the arms and the muscles of the upper part of the chest.

(e) *Thyroid Murmurs.*—Systolic murmurs are very common over the enlarged and tortuous arteries in goitre and especially in Graves's disease.

(f) *Murmurs in Local Arteriosclerosis in Superficial Arteries.*—Systolic murmurs due to this cause are occasionally observed. They are audible in some cases without pressure by the stethoscope; in others upon a minimum pressure. They are most common in the carotids.

Auscultation of the Veins. Normal Conditions.—In healthy individuals the blood flows in the veins without sound or murmur. In rare instances the occurrence of a venous hum constitutes an exception to this rule.

Auscultation of the Veins. Pathological Conditions.—(a) The venous pulse in tricuspid insufficiency may by sudden tension of the valves and walls of the jugular, and in particular the valves of the bulb, give rise to a systolic sound which is scarcely to be distinguished from the almost synchronous systolic carotid sound, except by the fact that it slightly precedes it.

(b) *Venous Hum—Nun's Murmur.*—This is the single venous murmur of practical diagnostic importance. Since the return flow of the venous blood is to all intents uninterrupted, venous murmurs are continuous.

The patient should assume the upright posture with the head straight. The stethoscope should be placed over the space between the sternal and clavicular portions of the sternocleidomastoid muscle without pressure. The murmur in question when present is heard as a peculiar, sometimes blowing, sometimes coarse and humming or again musical continuous sound, with rhythmical systolic, diastolic, and inspiratory intensifications. It is best heard upon the right side and diminishes in loudness or entirely disappears when the patient assumes the recumbent posture. Rotation of the face toward the opposite side increases the intensity of the sound. Pressure with the stethoscope at first increases then enfeebles the murmur until it wholly ceases and the systolic sound of the carotid is heard. In some cases a feeble murmur loses its continuous character and is perceived only at the moment of systole, diastole, or during inspiration. Under these circumstances the murmur becomes continuous upon light pressure with the stethoscope or if the head is rotated toward the opposite side—manœuvres which enable the diagnostician to distinguish the murmur from arterial and inspiratory murmurs. In other cases the diastolic portion of the murmur may be transmitted to the base of the heart and heard there as a diastolic murmur apparently of endocardial origin. The differential diagnosis may be attended with difficulty unless auscultation is practised from point to point from the heart along the course of the jugular, when it will become clearly apparent that the diastolic murmur heard over the base of the heart is in point of fact the transmitted venous hum. The venous hum occurs in anæmic and chlorotic persons and occasionally in healthy individuals with normal blood. In the last it has been assumed that the phenomenon is due to some anatomical peculiarity, as, for instance, sudden and unusual widening of the jugular at the bulb.

In general terms the causes of the venous hum are the more rapid flow of the blood current by reason of its lowered specific gravity and deficient hæmoglobin, and the sudden widening of the jugular at the bulb. The fact that this murmur is louder in the erect posture is rightly ascribed to the influence of gravity in hastening the flow; the increased intensity upon inspiration to the aspiration exerted by the chest movement at that time, and the greater loudness upon the right side to the more direct and unrestrained flow of the blood arising from differences in the anatomical arrangement of the veins of the two sides.

III.

THE EXAMINATION OF THE STOMACH AND INTESTINES.

General Considerations.—Diseases of these organs are (a) primary or organic and (b) secondary or symptomatic, and in each of these groups there are cases in which recognizable anatomical lesions are present and cases in which there is merely derangement of function. Thus, carcinoma and ulcer are examples of organic disease with characteristic lesions, and hyperchlorhydria and pyloric spasm are functional affections; while loss of appetite, eructations, and vomiting occur as symptoms of phthisis, often without actual lesions of the stomach, and erosion and ulcer are not infrequent in chlorosis. It is of cardinal importance that the differential diagnosis between organic and functional disease of the stomach and intestines should in all cases be made, particularly as the former only require direct local treatment and the latter are frequently made worse by such treatment.

The Anamnesis.—It is very common for patients to attribute to "stomach trouble" or "bowel trouble" symptoms due to diseases of other organs or to constitutional disease. A careful and systematic anamnesis is therefore necessary in all cases.

Status Præsens.—The actual condition is ascertained by, (a) physical examination, with special modifications, as inflation, transillumination, the Röntgen rays; (b) chemical and (c) microscopic examination of the gastric contents and alvine discharges. Differences in the methods, the substances to be examined, and the results render it convenient to consider the stomach and intestines separately.

EXAMINATION OF THE STOMACH.

(a) **Physical Examination.**—INSPECTION.—The recumbent posture and exposure of the whole abdomen as well as the thorax and neck are necessary. In the neck may sometimes be seen the enlarged left supraclavicular lymph-node in cases of carcinoma of the subdiaphragmatic viscera; the general conformation of the thorax and abdomen may be studied and in some cases the flaring of the left costal arch from the pressure of a distended high-placed stomach. When the abdominal wall is thin and relaxed the outline of the distended stomach, the slow, large movements of gastric peristalsis from left or right, far more rarely reverse peristalsis, the presence of tumor masses in the gastric wall or at the pylorus, or of metastatic growths about the umbilicus, in the liver, or elsewhere, may be observed.

PALPATION.—This method is useful in determining localized or general tenderness, the presence of a tumor, enlargement and displacement of the stomach, and "splashing." The stomach should be palpated systematically with both hands and for several minutes. By this means peristalsis may be aroused and a tumor which is not otherwise recognizable brought within reach. In stenosis of the pylorus and gastroptosis the wall of the stomach may sometimes be felt to become tense, with the sensation of the passage of fluid in the region of the pylorus, and in persons with thin-walled abdomens the tip of a bougie may be recognized as it is cautiously moved along the greater curvature. Succussion or "splashing" may be recognized both by the palpating hand and by the ear. It occurs in motor insufficiency and is of diagnostic value when elicited after a fasting period of several hours. The patient should be in the dorsal posture with the head and shoulders slightly elevated and at the moment the examination is made the stomach should be depressed by deep-held inspiration. The examiner exerts pressure upon the epigastrium at the level of the xyphoid appendix and with the finger-tips of the other hand makes quick pressing movements immediately followed by relaxation. This method may be used in the determination of the lower border of the stomach after the administration of half a litre of water. The outspread fingers of the palpating hand are used with the short pushing stroke of palpatory percussion, beginning below the level of the umbilicus and proceeding upward until the splash is felt.

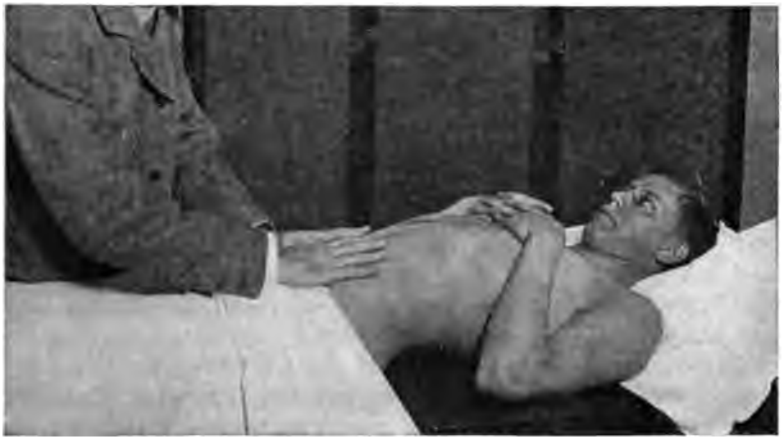

FIG. 82.—Palpating the abdomen.—Cohnheim.

PERCUSSION.—This method is of little value by itself. The stomach and adjacent coils of intestines yield tympanitic resonance or, when they contain fluid or solid matter, dulness, and their boundary lines cannot be defined by ordinary percussion. Auscultatory percussion, especially when the stomach is inflated, is of use. The percussion should be performed in lines radiating from a centre over the stomach and the points of change in the auscultatory phenomena marked upon each line in turn. Those points are then connected. Control observations may be made by changing the centre and repeating the observation.

Auscultation has only a limited application in the examination of the stomach. It is employed in the study of deglutition phenomena, "splashing," the *Spritzgerausch* of pyloric stenosis and hour-glass contraction of the stomach. In the last, if the narrowing be of high degree, there may be an audible sound produced by the passage of water from the cardiac to the pyloric loculus, especially if pressure be used.

THE SWALLOWING TEST.—This test is used to determine the patency of the cardiac orifice and is performed in the following manner: The ear or the bell of a stethoscope is placed at the left of the tip of the ensiform cartilage or, better, at the left of the seventh thoracic vertebra of the patient, standing, who is directed to take a mouthful of water and swallow. There is at once heard the rumble of the contracting œsophageal and cervical muscles brought into play and in about seven seconds the trickling of the fluid entering the stomach through the cardiac orifice. Delay or absence of the latter sound suggests more or less complete stenosis in the course of the œsophagus or at the cardia.

INFLATION.—The stomach may be distended by means of a stomach-tube and an ordinary rubber bulb syringe or by carbon dioxide evolved within the organ itself. To this end 3 to 5 grammes of tartaric acid dissolved in half a glass of water are swallowed and this is followed by an equal amount of sodium bicarbonate dissolved in the same quantity of water. The stomach first distends at its least resisting part and in the case of moderate enlargement and thin abdominal walls its greater curvature and inferior border may be made out by inspection or auscultatory percussion. The gas may be expelled as it is formed either at the cardia or at the pylorus, and in large stomachs the introduction of a sufficient quantity of air may cause much discomfort or even pain, or the gas evolved may be insufficient. The chief value of this method lies in the determination of the size, outline, and position of the stomach. It is of importance in the differentiation between tumors of the anterior wall and those situated in the posterior wall or behind the stomach. It is evident that upon inflation the former will become more prominent, the latter less obvious. Inflation is of some value also in bringing into prominence obscure tumors of the pylorus.

POSITION OF FLUIDS INGESTED UNDER OBSERVATION.—The position of the lower border of the stomach is ascertained as closely as possible, the patient standing. He is then directed to swallow a glass of water and the resulting dulness is determined by percussion. This manœuvre being repeated two or three times at short intervals, changes in the level of the dulness may occur, which are indicative of the position of the lower border of the stomach. In normal stomachs of good musculature the increased amount of fluid enlarges the area of dulness in an upward direction; in relaxed and dilated stomachs the dulness sinks with the increasing weight. The method is of no great value except in dilated stomachs and gastroptosis in individuals with thinned abdominal walls.

TRANSILLUMINATION.—The gastrodiaphane of Einhorn consists of a flexible tube carrying at its tip a small electric light. The patient drinks two or more glasses of water; the tube is introduced and the circuit completed. The light shows through the abdominal wall in the normal stomach as a triangular area having its apex and focus of intensity somewhat to the left of the median line and above the umbilicus; in gastroptosis or dilatation the point of illumination is lower and the light more diffused. Changes follow movements of the bulb. This procedure shows the lowest mit of the stomach at one point. There is no certainty that the lamp es not push the greater curvature into positions it would not otherwise cupy, or that the position of the illuminated area affords positive data regard to the size and shape of the organ.

Direct Gastroscopy.—Direct inspection of the mucous membrane the stomach through a rigid metal tube requires the patient recum- nt, with his head extended in such a manner that the trachea and sophagus approach nearly a direct course. General anæsthesia is recom- ended for the best results. The tube used is fitted with a small electric ht, mirror, and obturators, and in general resembles those employed in e diagnosis of rectal and sigmoid disorders, being, however, longer and ghtly thinner. Many conditions of the stomach are readily recognized direct inspection. An empty or even well washed stomach is preferable the time of examination. Ulcerations and local thickenings can be adily located. Inflammation, catarrh, or atrophy of the mucous mem- ane can be determined. Some care is necessary to ensure inspection of e whole interior, particularly if the stomach is enlarged. The cardia first inspected. Inflation aids in bringing other parts into view. y some manipulation of the upper end of the gastroscope and simul- neous palpation and manipulation on the part of an assistant the whole omach can be gone over and outlined. The readiness with which the d of the tube can be felt through the abdominal wall is of some rvice. The use of the œsophagoscope and gastroscope demands consider- le technical skill.

The Röntgen Rays.—Thickenings, the puckerings caused by dense atricial masses and tumors at the pylorus or elsewhere cast shadows hich often afford confirmatory evidence of great importance in diag- sis. By the aid of the bismuth meal—two ounces of bismuth subnitrate ixed in six ounces of rice pudding or suspended in eight ounces of kéfir— e outline of the greater curvature, hour-glass contraction, the position the pylorus, and the motor power of the stomach can be determined. e tracing of such a meal through the alimentary canal by serial obser- tions during the course of several hours frequently yields diagnostic ta of great value in diseases of the small and large intestines, while portant facts are sometimes to be acquired in disorders of the lower wel by X-ray studies made after the injection of bismuth suspension r rectum. Recent improvements in apparatus and technic, which nder possible instantaneous radiograms, have led to more accurate owledge in regard to gastric and intestinal peristalsis under normal d pathological conditions, much of which may become available for agnostic purposes.

The Stomach-tube.—Various styles are in use. The simplest is the st. They are made of soft red rubber with a lumen of about .50 to .75 ., walls not too thick, and about 70 to 90 cm. in length; near the gastric d one or two large lateral openings. Whether there should be an open- g at the end is a matter of opinion. The upper end is fitted to a glass nnel of a capacity of 500 c.c. At the middle there may be a bulb which rves for inflation or suction and permits free siphonage. As the distance m the incisor teeth to the cardia is on the average 40 cm., or slightly less than 16 inches, there should be an encircling mark at this point. It is important to note that many of the tubes supplied at the shops are marked at a point 51–60 cm. from the tip, about the distance to the lowest point of the greater curvature. For infants a soft catheter may be used.

Introducing the Tube.—The sitting position is easiest. It is best not to elevate the chin, since stretching the neck seems to occlude the upper œsophagus. Plates and false teeth should be removed. Soaking the last several inches of the tube in hot water makes the first contact of the tube with the pharynx less irritating. Holding the tube in the hand, as one would a pen, with five or six inches projecting, the examiner instructs the patient to open his mouth moderately wide, with the tongue touching and against the teeth. The tube is then passed straight back to the middle of the posterior pharyngeal wall and directed downward. If the patient can swallow at this moment the tube is usually engaged at once in the upper œsophagus and can be rapidly pushed in, reaching the stomach in several seconds, and is securely in place before the first expulsive coughing efforts begin. By ordering the patient to breathe deeply several times one usually succeeds in quieting most of the discomfort and can proceed with the examination. To avoid the doubling up of the tube or the unpleasantness of the coughing or vomiting which often ensues, many prefer to introduce the forefinger of one hand along the side of the mouth as a guide and to pass the tube along this guiding finger. Many attempts are often necessary to overcome the spasmodic expulsive efforts of the pharynx. At times the distress is such that it is necessary to forego the attempt. Cocainization of the pharynx has been recommended; it does not, however, seem to have been very successful in difficult cases.

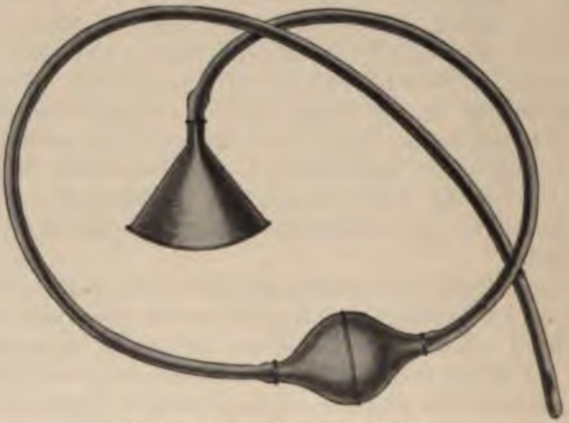

FIG. 83.—Stomach-tube.—Cohnheim.

The great value of the stomach-tube in gastric diagnosis lies in the ease with which the stomach may be inflated and its contents removed. Much time will be saved by having the patient take one or other of the various test-meals whenever the tube is to be passed. After removal of the meal the patient's clothes are loosened and he is directed to lie down with the tube still in position. Inflation can now be performed and in conjunction with some of the various methods already mentioned will be found to be the most satisfactory way of estimating the size and position of the stomach.

The stomach is inflated until the patient indicates the beginning of discomfort. The examiner then clamps the tube or can ask the patient to hold it firmly in his teeth, thus giving the examiner the free use of both hands. During the inflation the examiner should carefully watch for the area where the stomach first manifests its presence. In a normal stomach this will be just below the left costal margin and in the epigastrium between the ensiform and navel. The stomach will stretch easily and its greater curvature can be followed to the umbilicus before overdistention is complained of. The lesser curvature must be outlined as well, either by inspection, percussion, or auscultatory percussion, and marked in pencil. Its position should be under the costal arch as high as the sixth and seventh ribs, and just below the ensiform cartilage in the midline. The fundus may distend high up toward the axilla. A distinct stomach-shaped outline can usually be obtained. It is only by outlining both lesser and greater curvatures that the difference between displaced and dilated stomachs is determined.

The great advantage of the use of the stomach-tube in inflating is that the process can be repeated as often as may be desired without undue discomfort or delay, since after the first few minutes the patient experiences little or no uneasiness.

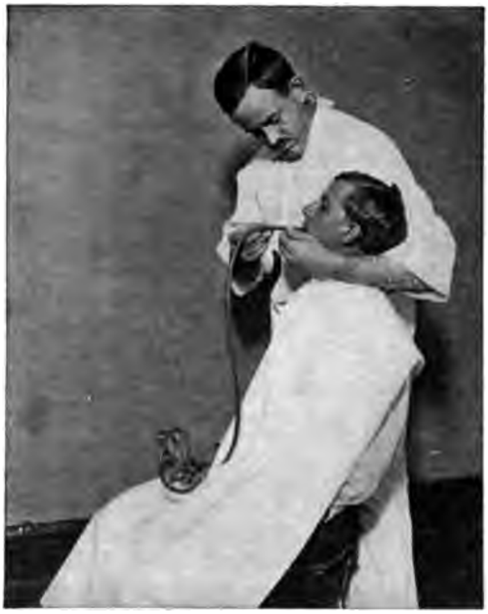
FIG. 84.—Method of introducing the stomach-tube.—Cohnheim.

In addition to the estimation of the size, shape, and position of the stomach, the stomach-tube is used to determine conditions of hypersecretion and retention. To test for hypersecretion the stomach-tube is passed in the morning before any fluid or food has been ingested, or the stomach may be washed out and the tube again passed after several hours' fasting. The recovery of more than 10 to 20 c.c. suggests disturbance of the gastric functions. In testing for retention the examiner washes the stomach clean, administers certain solid articles of diet, and passes the tube to obtain samples of the stomach contents, seven or eight hours later, or, according as marked conditions are suspected, twelve, twenty-four, or even forty-eight hours after the taking of the meal.

Contraindications for the Use of the Stomach-tube.—Those who for repeated diagnostic or therapeutic purposes have become accustomed to the tube take it without difficulty and many learn to introduce it themselves. Its first introduction is often attended with great gagging, straining, and congestion, and is not wholly without danger in elderly persons with arteriosclerosis, myocarditis, and emphysema of high degree. It is also hazardous and unjustifiable in hemorrhagic cases, especially hæmoptysis, hæmatemesis, or cases of marked anæmia with tarry stools, and in aneurism of the aorta, in great debility from acute or chronic illness, and in pregnancy. Even in the absence of any of the foregoing conditions the retching, gagging, and distress of the patient may be so great that the attempt to pass the tube must be temporarily abandoned.

(b) **The Chemical Examination.**—The further examination of the stomach consists in the administration of certain test-meals or substances, their removal by means of the stomach-tube after a given period of time, and the application of various chemical tests for the digestive agents of the gastric juice.

Previous to the giving of a test-meal the stomach should be perfectly free from food remains; hence in cases of suspected dilatation or retention lavage must first be employed. Whatever the form of test-meal some difficulty may be met with in removing it. Frequently the tube is not in the stomach, either not reaching beyond the cardia, or doubled up in the œsophagus or pharynx. In other cases the tube may have reached the greater curvature and turned upward upon itself until the openings are above the level of the stomach contents. Not infrequently plugs of mucus will close the eyes of the tube. In marked displacement and dilatation the end of the tube may not reach the ingested test-meal unless introduced many inches beyond the average distance. Forcing in a little air, partly withdrawing and advancing the tube, will usually clear the opening and enable one to find the proper distance and the point at which the material best flows out or comes out by suction. Some little information is gained by the way in which the material comes away. In atonic conditions of the gastric muscles suction must often be used, even when large amounts of fluid material are present. With a musculature of fair or active tone the gushing forth of the stomach contents is not uncommon.

Test-Meals.—Several standard test-meals are in use.

THE EWALD TEST-BREAKFAST.—This consists of 35 grammes of stale bread or toast without butter and 200–400 c.c. of weak tea or water. Two small slices of toast without butter and one cup of weak tea without cream or sugar represent these amounts fairly well. This meal is to be removed at the end of one hour, or not later than an hour and a quarter, since in the normal stomach the height of acidity is reached by this time and the stomach has already begun to empty itself. Not infrequently the stomach is found empty at this time and one must repeat the meal and remove it earlier. Attention to the details of managing the tube will usually enable one to obtain some part of the gastric contents. With this meal after one hour's time 40 to 60 c.c. of stomach contents should be obtained, brown or white according as toast or bread has been used, of the consistency of thin porridge, or if the absorption of fluid has been rapid much thicker, the bread or toast finely minced by the stomach's activity and without an odor. The glairy saliva produced in excess by the tube's presence in the mouth and the whiter plugs of mucus from the posterior nares, are often mixed with the contents and can be readily distinguished and at once removed. As the contents settle mucus from the stomach will gather together and remain upon the top. Little or none should be present, nor should there be a layer of froth. On standing, if in sufficient amount, the removed test-meal and gastric juice show two layers: the upper fluid, gray, milky, or light green in color; the lower brown or white, composed of the remains of the bread or toast. The tea or water is usually quickly absorbed, and the overlying fluid consists largely of gastric juice.

Objections to the Ewald test-meal are, first, that even stomachs whose motive power is failing can get rid of most of the fluid of the meal within the stated time, and hence relaxed conditions in their early stages may be overlooked; secondly, that the mild character of the meal does not bring out certain conditions of hyperactivity of the gastric mucosa as a more stimulating one would tend to do.

RIEGEL'S TEST-MEAL—200 c.c. mutton broth, 150–200 grammes beefsteak, potato purée 50 grammes, one roll 35–50 grammes—has been proposed in order to overcome these difficulties. The gastric contents are removed at the end of four hours for the various tests of gastric secretion, or at the end of seven hours to test for gastric motility or retention, it being usual for the normal stomach to have emptied itself by that time. The general readings of the chemical tests in Riegel's meals after four hours are found to correspond more or less with those of the Ewald meal after one hour. As a matter of experience it might be said that careful physical examination will overcome the first objection, and that only occasionally, when the symptoms do not correspond with results of the Ewald test-meal, will the Riegel meal be necessary.

Many other test-meals have been proposed, but they do not seem to yield any better results than the two mentioned.[1]

After consideration of such points as the amount removed, the manner of its expulsion or withdrawal, and preferably after a preliminary settling, the upper liquid layer or the whole contents are filtered and the filtrate submitted to chemical examination.

Starch.—The effect of the salivary enzymes can be first and most simply ascertained. The conversion of starch to achroodextrin and maltose goes on in the stomach until the free acids of gastric secretion reach a certain point. The well-known iodine reaction, coloring starch violet, coloring erythrodextrin, the first product, mahogany brown, and having no characteristic color effect in the final stages, achroodextrin and maltose, allows us to estimate quickly and qualitatively the extent of salivary digestion. Both the filtrate and residue contain reacting substances, soluble and insoluble starch. Excess of unaltered starch gives at once with iodine solutions (Lugol's solution .1 Gm. iodine, .2 Gm. potass. iodide, 200 c.c. water) a deep violet color; achroodextrin and maltose show no color reaction, though the latter is readily detected by Fehling's solution. Iodine, however, must be added in excess, since achroodextrin has a greater affinity for it than has starch, and the violet starch reaction may only appear after all the achroodextrin has been satisfied. The same color effects can be readily seen under the low power of the microscope.

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[1] For a full discussion of this subject consult Clinical Diagnosis: Emerson; second edition, J. B. Lippincott Co., 1908.

The main relationship which starch tests, *i.e.*, salivary digestion, bear to stomach digestion is that hyperacid conditions of the stomach interfere with its progress, and that hypo-acid conditions may favor it.

The more important tests in ordinary clinical work are those for (1) acidity; (2) presence of free acids; (3) presence of free HCl, lactic, and butyric acids; (4) presence of HCl in combination (combined HCl). Tests for proteid digestion, pepsin and peptone reaction, are usually considered to be unnecessary when normal or increased free HCl is found. Milk-curdling ferment is rarely tested for. A fat-splitting ferment in small amounts has been occasionally demonstrated, but is not regarded as of great practical importance. The qualitative tests in common use can be first considered.

Qualitative Tests.—1. TEST FOR ACIDITY.—The products and agents of gastric digestion are normally acid, this reaction being due to free acids —HCl, lactic acid, butyric acid, and their combinations. Blue litmus paper is reddened by their presence.

2. TESTS FOR THE PRESENCE OF FREE ACIDS.—Congo red paper or solution is turned deep blue by free HCl. A less intense reaction is given by the organic acids.

3. TESTS FOR THE PRESENCE OF FREE HCl, LACTIC ACID, ETC.—*HCl.* —(a) *Methyl Violet Reaction.*—To a pale violet solution of methyl violet (one drop concentrated aqueous or alcoholic solution in a test-tube of water) add a few drops of the filtrate. A distinct blue change takes place if free HCl is present. A control tube should be on hand for comparison.

(b) *Tropæolin* 00 *Reaction.* — Two or more drops of fresh concentrated alcoholic solution of tropæolin (a deep orange-colored solution) are spread on a porcelain plate or dish. The same amount of filtrate is added to this surface and the porcelain gently heated. A distinct violet reaction turning to blue takes place.

(c) *Phloroglucin-vanillin* (*Günzburg's*) *Reaction.*—Two or three drops of the solution (phloroglucin 2 Gm., vanillin 1 Gm., alcohol 30 c.c.) are used with the same amount or more of the filtrate as in the tropæolin test and dried by gentle heat. The brown color of the phloroglucin-vanillin is changed to a distinct carmine red if free HCl is present.

(d) *Dimethylamidoazobenzol Reaction.* — A drop of a .5 per cent. alcoholic solution of this substance (a light red-brown solution) added to the filtrate or to the residue, quickly gives a bright red color if free HCl is present. In cases where but a few drops of gastric contents have been secured this test can be readily applied without waiting to filter.

The last two tests are by far the most reliable and are generally considered as absolute tests for the presence of free HCl. Lactic acid in excess may give suggestive results in the methyl violet and tropæolin reactions.

Lactic Acid.—*Uffelman's Test.*—One drop of a 10 per cent. solution of ferric chloride is added to 20 c.c. of a 1 per cent. solution of carbolic acid. The resulting deep blue mixture is diluted until it appears as a light amethyst. On the addition of a gastric filtrate containing lactic acid the amethyst changes to distinct yellow. Excess of free HCl, sugars, or peptones may decolorize the amethyst, and the yellow tint of many filtrates if added in excess gives suggestive but uncertain results. A comparison with a test solution of lactic acid is always useful for a beginner. Far better results are obtained by shaking a portion of the filtrate with ether, which extracts the lactic acid, and applying the test to the evaporated residue, which may be preferably diluted with 2 or 3 c.c. of water. Strauss' modification of this test is also serviceable. One may dispense with the separator if it is not obtainable. Five c.c. of gastric juice are placed in a test-tube; 20 c.c. of ether are then added, the tube corked, thoroughly shaken for a few minutes, and allowed to settle. The overlying ether and extract can now be carefully removed with a pipette and mixed with 5 c.c. of distilled water. To this mixture two drops of a 1 in 9 watery solution of ferric chloride are added and the mixture is again shaken. The watery layer, as it settles below, is of an intense greenish-yellow color if lactic acid be present.

Butyric Acid.—This acid is usually only tested for by its odor. This and others of the volatile fatty acids, acetic and valerianic, are only looked for in marked conditions of stagnation of the gastric contents. Very minute quantities of them all, however, occur in various food-stuffs.

Combined HCl.—A qualitative test for the proteid combinations of HCl is not in general use. The quantitative tests will be considered below.

Proteid Digestive Power.—For the qualitative tests of the power of the gastric juice to digest proteid, one or two simple devices have been used. Fibrin and coagulated albumin (egg albumin) are the common proteids used: the fibrin, well washed, hardened in alcohol and stained by neutral carmine, will digest in gastric juice containing free HCl and pepsin, imparting a red color to the liquid mixture as the carmine is set free by the digestion.

Small pieces, or disks 2 mm. in diameter, 1 mm. in thickness, of not too firmly coagulated egg albumin are placed in a few c.c. of gastric contents. According to the amount of pepsin and free HCl present digestion begins more or less rapidly, and softening of the edges of the disks can be seen in one to two hours. Many hours are required for the complete granulation of either fibrin or albumin. Gastric juices deficient in free HCl have less and less effect upon the proteids employed. Some slight digestion goes on even with complete absence of free HCl.

Fig. 85.—Separating apparatus suitable for testing for lactic acid.—Strauss.

Neither of the proteid digestive tests is very instructive, and one must remember that the pepsin present in the gastric filtrate has been already partly used in the proteid digestion of the test-meal. Sahli's desmoid-proteid digestive test will be considered under the absorption tests.

Tests for Rennin and Rennin Zymogen.—To 10 or 15 c.c. of neutral milk add 5 c.c. of gastric filtrate, and place the mixture in a thermostat or in any warm place. In 10 to 15 minutes coagulation begins. This is merely the familiar "junket making." Free HCl is not necessary for its performance.

Quantitative Tests. — For the differentiation of many of the disorders of gastric secretion an estimation of the amount of acids and acid combinations is often necessary. Free HCl is the only free acid regularly estimated. The estimation of the total acidity of the gastric contents, which is made up of free HCl, traces perhaps of other acids (lactic acid), and combinations of HCl and lactic acid (if present) with the proteids of the administered meal, is the next important step. Estimations of these combined products are also made.

QUANTITATIVE ESTIMATION OF THE AMOUNT OF FREE HCl. — The amount not used in the process of digestion at the time of the test-meal's removal. The general principle of all the tests for total acidity and free acids is the same, namely, to add to the filtrate a standard alkaline solution until the acid contents are neutralized. To aid in determining neutralization various coloring agents, some of which have been already described in the qualitative tests, are added to the gastric filtrate. These coloring agents all have the peculiarity of losing or even changing their color when the filtrate becomes neutral or faintly alkaline in reaction from the added alkali. The solution in general use is the "one-tenth normal" sodium hydrate. This one-tenth normal solution, written $\frac{N}{10}$ NaOH, is preferred on account of its dilution, which, when dealing with such small amounts and percentages as are found in the stomach contents, is very necessary. One c.c. of this $\frac{N}{10}$ NaOH corresponds to or exactly neutralizes .00365 gramme of free HCl.

In making the quantitative estimation, 10 c.c. of the gastric filtrate are taken in a beaker. By means of a graduated burette the $\frac{N}{10}$ NaOH is allowed to drop into the beaker until the so-called indicators or coloring agents show their characteristic changes, indicating complete neutralization. The number of c.c. of $\frac{N}{10}$ NaOH used, multiplied by the free HCl equivalent of 1 c.c. $\frac{N}{10}$ NaOH (.00365 gramme), gives the amount of HCl in the 10 c.c. of gastric filtrate, and one readily calculates the percentage amount therefrom, normally about .1825 gramme per 100 c.c.

At present it is more common to express the results in direct figures or per cent., indicating merely how many c.c. of $\frac{N}{10}$ NaOH are necessary to neutralize 100 c.c. of the gastric filtrate (*i.e.*, its contained free HCl), as, for instance, if 10 c.c. of gastric filtrate (containing free HCl) are neutralized by 5 c.c. of $\frac{N}{10}$ NaOH, the percentage of free HCl is said to be 50.

The most convenient indicator for free HCl is dimethylamidoazobenzol. In contact with free HCl in the filtrate a bright red color is shown. Neutralization by $\frac{N}{10}$ NaOH turns the red color to a turbid yellow. The beaker containing the filtrate and indicator should be well stirred or shaken while adding the alkaline solution from the burette.

Equally satisfactory for quantitative estimation is the phloroglucin-vanillin test. Ten c.c. of the filtrate are placed in a beaker, the $\frac{N}{10}$ NaOH is added slowly, and after every 10 to 15 drops one takes a drop of the filtrate and tests it for free HCl on a porcelain plate with the phloroglucin-vanillin. The non-appearance finally of any trace of the carmine-red color indicates the complete neutralization of the free HCl in the filtrate.

ESTIMATION OF TOTAL ACIDITY.—The same methods are used with merely different indicators: either phenolphthalein or rosolic acid. Two or three drops of a 1 per cent. alcoholic solution of the former give to the gastric filtrate a turbid appearance. Upon the gradual addition of the $\frac{N}{10}$ NaOH there appears a red-purple color where the drop strikes, quickly disappearing at first but becoming more and more persistent until shaking the filtrate no longer causes the color to disappear. A good rule to follow in this test is to consider the reaction complete when the color will remain for 40 or 60 seconds.

Upon the addition of 2 or 3 drops of a concentrated solution of rosolic acid to 10 c.c. of the filtrate the color is changed to light brown. Neutralization is shown by the appearance of a rosy red color.

Since the estimation of the total acidity requires the greater amount of alkaline solution, it is possible to make both tests in one beaker containing 10 c.c. of filtrate. Using dimethylamidoazobenzol as an indicator one can find first the amount of $\frac{N}{10}$ NaOH necessary to neutralize the free HCl present. By adding phenolphthalein or rosolic acid to the now light yellow mixture the determination of the total acidity can be made; the amount of $\frac{N}{10}$ NaOH dropped in after using the last indicator being merely added to the amount recorded in the estimation of the free HCl.

Frequently it is of interest to estimate how much of the secreted HCl has combined with the proteid of the meal forming the so-called combined HCl. Many cases showing no free HCl on tests will show that there has been free HCl secreted in the stomach as evidenced by the existence of its combined products.

The simplest tests require the finding of the total acidity in the beginning.

The total acidity represents free acid, acid combined with proteids, and acid salts (acid phosphate).

Alizarin as an indicator reacts acid to free acid and acid salts, but not to combinations of acids and proteids; hence the difference between two tests, the amount of $\frac{N}{10}$ NaOH used in one with phenolphthalein as an indicator, the other with alizarin, must represent the acids in combination. The reaction is complete when the yellow of the indicator turns to a distinct violet.

To summarize these tests with an example, the following normal figures may be used.

1. 10 c.c. of gastric filtrate with phenolphthalein as an indicator for neutralization require 4 c.c. $\frac{N}{10}$ NaOH: 100 c.c. would require 40 c.c. Free acids, acids in combination, acid salts = total acidity 40.

2. 10 c.c. of gastric filtrate with alizarin as indicator (free acids, acid salts) requires 3 c.c. $\frac{N}{10}$ NaOH: for neutralization 100 c.c. would require 30 c.c. Total acidity $40 - 30 = 10$. Combined acids 10.

3. 10 c.c. of gastric juice with dimethylamidoazobenzol as indicator (free hydrochloric acid only) require 2.5 c.c. $\frac{N}{10}$ NaOH for neutralization. Free HCl therefore = 25, in terms of 100.

A much more reliable method of estimating combined HCl is that of Cohnheim and Krieger. Calcium phosphotungstate separates HCl from its combination with albumin and albumoses, the calcium uniting with the HCl forming neutral calcium chloride. In the process a reduction of the total acidity takes place, corresponding to the amount of combined HCl, which has been changed to the neutral calcium chloride. The difference between titrations before and after the calcium phosphotungstate reaction must represent the amount of acid in proteid combination. The detail is more troublesome than the simple alizarin process, but gives far more accurate results than can be expected where two separate color changes are required.

Four per cent. phosphotungstic acid is neutralized by gently boiling with calcium carbonate. Calcium phosphotungstate is formed; the solution is filtered, tested for neutrality, and can be kept for any length of time. 30 c.c. of this calcium phosphotungstate is added to 10 c.c. of gastric juice. A heavy precipitate of proteid phosphotungstate results while the newly formed neutral calcium chloride remains in solution. This mixture is now filtered, the precipitate remaining on the filter paper being well washed by pouring on it distilled water (two or three separate additions of 5 or 10 c.c.) and adding the wash water to the original filtrate.

Using rosolic acid as an indicator the total acidity of 10 c.c. of gastric juice is first estimated, then the same test is repeated with the material obtained after the phosphotungstate reaction, usually about 50 c.c. of clear fluid. As an illustration:

1. Total acidity of 10 c.c. gastric juice, rosolic acid as indicator = 50.
2. Total acidity of mixture (10 c.c. gastric juice + 30 c.c. calcium phosphotungstate + wash water), rosolic acid as indicator = 35. $50 - 35 = 15$, difference due to conversion of HCl combined with proteids into neutral calcium chloride. Combined HCl therefore = 15.

Gastric juices in which the free HCl is absent are often examined for free HCl deficiency. This is necessary if the calcium phosphotungstate method of estimating combined acids is used. The process is simple and similar to the above tests. To 10 c.c. of filtrate, dimethylamidoazobenzol is added. With absence of HCl there is of course no reaction. $\frac{N}{10}$ HCl is now added until a reaction for free HCl takes place. If for instance 1 c.c. $\frac{N}{10}$ HCl must be added, the equation is 10 c.c. gastric filtrate, with dimethylamidoazobenzol as indicator, required 1 c.c. $\frac{N}{10}$ HCl to produce a reaction of HCl. In terms of 100, HCl deficit = 10.

Quantitative tests for lactic acid are not necessary. The chlorides as a general rule are not tested. Since, however, their increase in gastric carcinoma has been claimed, a quantitative estimation is at times called for. The procedure is lengthy, and for its methods the reader is referred to works on chemistry.

Test of Gastric Absorption.—The absorptive power of the stomach may be estimated by the administration, when the organ is empty, of a gelatin capsule containing 0.2 Gm. of potassium iodide. The saliva and urine are tested at intervals of several minutes by the addition of a small quantity of starch meal or a bit of starch paper and HCl. A positive reaction is shown by the familiar blue color which normally should appear in the saliva in six to fifteen minutes and in the urine in about fifteen minutes. This test is of no great value.

SAHLI'S DESMOID TEST.—More as a test of peptic activity than of gastric absorption, this deserves mention and description. Recognizing that peptic digestion as shown in the test-tube represents by no means the conditions inside the stomach, many investigators have endeavored to invent some capsule which would open and give out its absorbable contents as a result of gastric digestion alone. Great trouble was experienced for two reasons. First, osmosis between the contents of the capsule and gastric juice took place through the animal membranes (proteid substances), and, secondly, many of the substances used were disintegrated by the muscular action of the stomach. Sahli's invention consisted in enclosing absorbable substances (iodoform and methylene blue) in a small piece of rubber dam, tying them in with a strand of raw catgut. Osmosis cannot take place through the rubber; raw catgut can only be dissolved by the gastric contents resisting absolutely the pancreatic ferments. The appearance of iodine in the saliva and methylene blue in the urine is held to indicate that the raw catgut has been digested by the gastric juice and set the contents of the "pill" free; hence the main value of the test is the proof of the digestion of proteid and peptic activity. The details of the desmoid test are as follows:

Iodoform .1 gramme and methylene blue .05 gramme are enclosed in a square of rubber dam 2×2 cm. The rubber is stretched tightly to make a small pill and its loose ends tied with catgut which has been previously softened in water. All free hanging edges of rubber are trimmed off. The pill properly formed should sink in water and should show no diffusion of methylene blue when placed therein. Well made and tested in this way a pill given during a full general meal, preferably at mid-day, should sink to the bottom of the stomach and will not be carried off until the end of digestion. In from 5 to 7 hours the first blue tingeing of the urine from methylene blue takes place. Iodine can be determined in the saliva or urine by shaking a small quantity of the respective fluid with a few c.c. of chloroform and adding pure colorless nitric acid, a reddening of the chloroform being the indicator of the presence of iodine.

Tests of the Motor Power of the Stomach.—LAVAGE.—The most satisfactory test for gastric motility consists in the administration of a Riegel test-meal and the washing out of the stomach at the end of seven hours, when under normal conditions the organ will be found to have emptied itself. After an Ewald test-meal traces of food should have disappeared at the end of two hours. No remnants of an ordinary supper should be found upon washing out the stomach the following morning after rising at the usual hour. In atonic conditions and dilatation remnants of partially digested food may be washed out not only at the end of these periods but in extreme cases even at the end of two or three days.

THE SALOL TEST.—Less reliable is the administration of one gramme of salol in gelatin capsules directly after an ordinary meal. The urine is voided at subsequent intervals of half an hour, one, two, three, and twenty-seven hours, and the respective discharges preserved for examination in separate vials. Each portion is then separately tested for the presence of salicyluric acid by the addition of a small quantity of a solution of ferric chloride, which develops in the presence of the acid a violet color. The presence of salicyluric acid in the urine is the sign of the decomposition of the salol into phenol and salicylic acid, and, as this takes place only in an alkaline medium, it is the indication that the salol has passed from the stomach into the intestine, which with normal gastric motility takes place in about one hour. A retarded reaction indicates impairment of motility, a delay of twenty-four hours is suggestive of pyloric obstruction. This test is not accurate, as it is impossible to determine in different individuals the relative time consumed by the chemical changes in the intestine and the elimination by the kidneys. Moreover, the salol may go out of the stomach not with the first portion of the food but with the last. Normally all of the salicyluric acid should have been eliminated within twenty-seven hours.

(c) **Microscopical Examination of Gastric Contents.** — With the ordinary Ewald test-meal little is to be learned by microscopical examination. Starch granules, a few epithelial cells, and bacteria are usually seen. If there has been much trouble in passing the tube a few blood-cells may be found. With a mixed meal or in vomited material starch, potato starch, fat droplets, and meat fibres are readily recognized. Many and larger bacteria are seen, a few leucocytes are commonly met with, and in sediments deposited after standing, many large granular mononuclear cells. In cases of acute gastritis considerable blood and pus may be found among the stringy, transparent strands of mucus. In gastric ulcer, blood in the test-meal or vomitus is common. It may be recognizable if the hemorrhage has been recent. The hyperacid condition of the gastric juice in these cases, however, destroys the blood-cells rapidly and chemical tests for the blood are necessary. Tissue cells from the ulcerating area are often found. In gastric cancer with lessened acidity blood-cells are less quickly destroyed, but as a general rule the digestive juices rapidly alter the separate cells. Small clots which have partially resisted the gastric juice form the sediment in the characteristic "coffee-grounds" vomitus of cancer of the stomach. Occasionally small masses showing distinct adenocarcinomatous arrangement may be found and are conclusive proof of the existence of cancer. Small masses of tumor visible macroscopically are occasionally seen in the vomit or washings from a carcinomatous stomach.

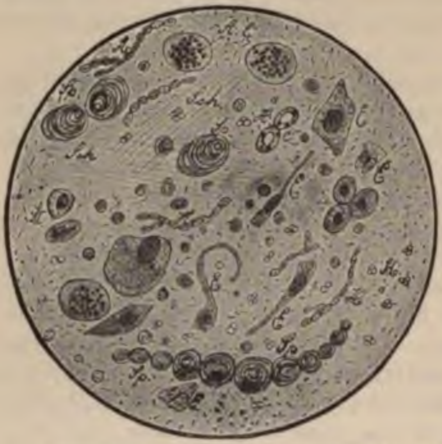

Fig. 86. — *K*, free nuclei; *Sp*, spirals; *Sch*, mucus; *H*, yeast-cells; *E*, epithelium; *AE*, alveolar epithelium.—Cohnheim.

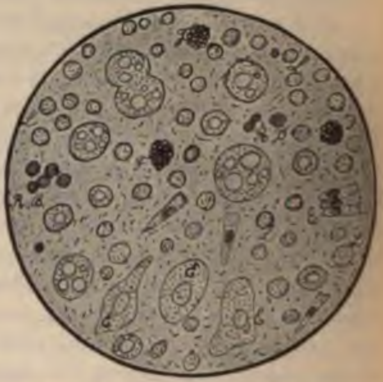

Fig. 87. — *E*, epithelium; *L*, leucocytes; *RB*, red blood-cells; *F*, fat-cells.—Cohnheim.

Of the bacilli present a majority are small, more or less motile, probably introduced with food. A few extra large organisms of the hay bacillus group are always to be found. One should be careful not to consider these large regular organisms as the form described by Oppler and Boas. The latter are large, irregular club-shaped and vacuolated bacilli, possibly the degenerate forms of the so-called gas bacillus, or of a special lactic acid forming bacillus. They are most commonly found when lactic acid is present, and under this condition have been considered as suggestive of cancer.

Einhorn's Bead-test of Digestive Activity. — Six small glass beads are connected with a silk thread; to each bead is tied some particular sort of food. Raw catgut and a soft long-bone of a pickled herring are the two substances used to test gastric digestion; raw meat, raw thymus gland, mutton fat, and a cube of cooked potato test the intestinal digestive power. The beads and thread can be placed together in a gelatin capsule and swallowed. Normally the beads should appear in the stool in one or two days; their elimination earlier than this indicates accelerated motility of the intestine; their appearance in the stool later than two days after administration is held to indicate retardation of the fecal excretion. All the beads should be empty, though traces of fat, thymus, and fish-bone may be left undigested. Excretion of the catgut and fish-bone undigested would indicate impaired gastric digestion. Excretion of undigested meat, thymus, or fat indicates deficient intestinal digestion. The silk thread is of course merely to facilitate the finding of the beads.

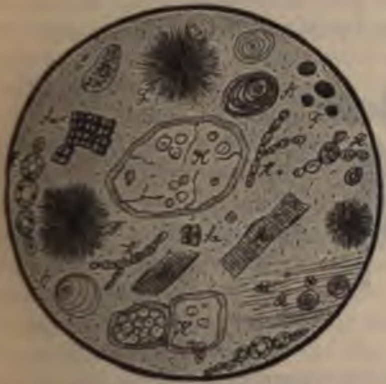

Fig. 88. — *St*, starch-cells; *H*, yeast-cells; *Sa*, sarcinæ; *M*, muscle-fibres; *F*, fat-balls and droplets; *K*, potato-starch cells.—Cohnheim.

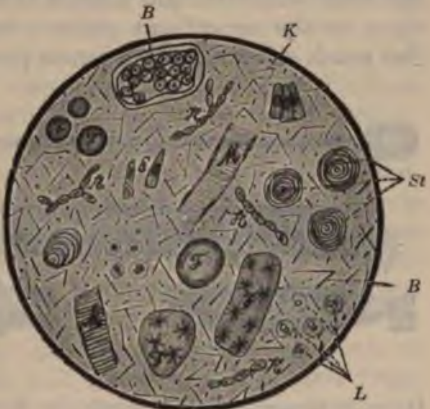

Fig. 89. — *H*, yeast-cells; *M*, muscle-fibres; *L*, leucocytes with shrunken nuclei; *B*, Oppler-Boas bacilli; *St*, starch-cells; *F*, fat; *E*, epithelium; *K*, potato-starch cells with yeast-cells.—Cohnheim.

EXAMINATION OF THE INTESTINES AND FÆCES.

Only the lower bowel is accessible for direct examination of its interior. Inspection with the aid of proctoscopes, digital examination of the rectum, and inflation of the colonic area enable us to investigate at least a part of the large bowel directly. The remainder as well as the small intestine can only be reckoned with through the abdominal wall by inspection, palpation, percussion, auscultation, and radioscopy. On the other hand, a careful examination of the fæces will tell us much as to function of the intestines and as to the presence of abnormal conditions.

Inspection as applied to the examination of the intestines may be, as we have said, direct when we are dealing with the large bowel; the proctoscope and the rectal tubes allowing inspection of the mucous membrane practically to the splenic flexure of the colon. The tubes or specula come in varying sizes, usually four in a set, ranging from 4 to 14 inches (14 to 35 cm.) in length and from 1 inch diameter in the short speculum to ½ inch diameter in the longer. They are provided with obturators. Their use is associated with considerable pain, though with persistence and gentleness most patients can go through the performance without an anæsthetic. Warming and oiling the instruments thoroughly will overcome some of the difficulties. After the sphincter muscle of the anus has been stretched and dilated the discomfort lessens. Either the knee-chest or the recumbent posture with the knees elevated may be used. Too much elevation of the lower part of the body will naturally by gravity send the bowel away from the examiner. The electric headlight with reflector facilitates examination. Very little trouble is experienced in straightening out the sigmoid flexure, nor do the valves of the rectum interfere with the progress of the speculum. It is common to find that the bowel dilates perceptibly with air when the speculum is in place, thus materially aiding the examination.

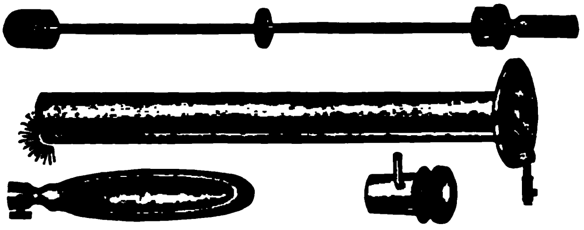

Fig. 90.—Rectoscope.

In the more modern rectoscopes and sigmoidoscopes the distal end is so arranged that air can be forced into the bowel in front of the advancing tube. A glass shield near the distal end allows the observer to look into the bowel and at the same time keeps in the injected air which balloons the bowel for several inches. A small electric light arranged in front of the tube gives a clear view of the mucous membrane.

Inspection with the proctoscope or rectoscope may show us first the presence of scybala, beyond reach of the palpating finger, revealing themselves often as hard, adherent, though detachable, masses of varying size, dark in color, or gray if covered by mucus, and usually easily indented or broken away with a probe, and readily differentiated from polypoid and other growths; second, ulcerating and bleeding points, dilated venules, fistulous communications, thickening and reddening of the mucous membrane of the bowel; general reddening of the whole surface in colitis, showing mucus, glairy or dense and white if the condition of mucous colitis is present, and often in large amounts; third, polypoid growths, malignant growths in the form of local thickening, strictures, foreign bodies, ulcerating tumor masses; fourth, obstructions outside the bowel, tumors of uterus, ovary, etc., preventing insertion of the examining tubes. It is usually considered permissible and advisable to remove small particles of suspicious growths for microscopical examination.

Examination of the bowel by means of bougies may at times help in diagnosing a stricture, particularly if the feeling of distinct resistance and the sensation of passing an obstruction can be appreciated repeatedly at the same point both during the introduction and withdrawal of the sound.

Palpation as a means of examination in regard to the intestine has but limited direct application, namely, the examination of the rectum by the finger, or if the sphincter can be dilated by two fingers or the whole hand. Examination is always facilitated by a previous good clearing out of the lower bowel. The forefinger of either hand may be used and various positions employed. The examiner, however, reaches a higher point in the bowel if the patient lies on his side with his knees drawn up to a moderate extent. In this posture more freedom is given the introduced finger, and the rotation of the hand and finger in examining the sides and front of the rectum much aided. The knee-chest position is also convenient.

To avoid the unpleasant fecal odor it is advised to fill the space between one's nail and finger with a little moist soap and to follow this by a free greasing of the finger with oil or vaseline. A finger cot can be used.

After inspecting the anus and neighborhood for fissures, fistulæ, hemorrhoids, exuding blood or pus, etc., the finger is gently inserted, overcoming gradually the spasm of the sphincter which always occurs and which must not be taken for a stricture. The tight grip of the muscle on the finger relaxes during the examination and gives considerably more freedom of movement. Examining as a routine the prostate and bladder, or the uterus, one may at once exclude or determine conditions affecting those organs; then, sweeping around to either side one feels for points of tenderness, irregularities on the smooth wall of the bowel, dilated veins, fistulous communications, polypi, etc. The finger is then turned toward the back of the rectum. The position and condition of the coccyx should always be determined during any rectal examination. Various obstetrical, genito-urinary, and gynæcological examinations may be made by way of the rectum. The fact that impacted ureteral calculi can at times be perceived by the examining finger if caught in the bladder wall or in the neighborhood of the pelvic brim must be mentioned. Too little consideration is given to a rectal examination in appendicitis and appendicular complications. An appendix abscess extending downward, though often painless, is frequently associated with an extreme tenderness when touched by the examining finger. This tenderness is usually too localized to be confused with any general abdominal distress, and its high position to the right is of diagnostic significance.

The examining finger has first 1–1½ inches of contracted sphincter area to overcome. A slow inserting movement dilates the muscles without pain, and allows the examiner and patient to appreciate localized tender areas, such as are occasioned by fistulæ, or ulcerations of hemorrhoidal veins. A forcible dilatation would readily, by the pain occasioned, prevent the disclosure of many of these minor conditions. Beyond the sphincters the finger has free play and at times one fails to touch any part of the bowel, the rectum being ballooned by flatus; by crooking the finger one touches readily the rectal wall. It is at times possible to distinguish emptied fallen coils of small intestines in the pelvis by rectal examination; such a condition may take place when a complete stricture has occurred high up in the small intestine, the empty tract below collapsing and descending.

Indirectly both the small and large bowel can be examined through the abdominal wall by inspection and palpation. Auscultation has but a doubtful bearing on abdominal conditions; even marked intestinal disorders may yield nothing to the most experienced. On the other hand, correct interpretations of simple existing phenomena may give most important results. Inspection is always preferably performed with the patient lying on his back, with his knees either flexed or extended. (See Methods of Physical Diagnosis.)

Examination of the large intestine and the cæcal region is helped by the easily applied method of inflation. Before this procedure a thorough purging is always advisable. The soft rubber rectal tubes, ¼ to ½ inch in diameter, with two or more lateral openings, and connected with a Davidson syringe or a double or single atomizer bulb, can be inserted to any distance desired. A slow twisting insertion will quickly put the openings above the anus and sphincters. Since the sigmoid flexure is the most commonly dilated part of the bowel, inflation should begin while the tube is entering the flexure, and the first examination directed to this part. Unless previously distended by gas or continued fecal accumulations the sigmoid flexure should not rise easily out of the pelvis during inflation. Usually when a point half way between the groin and the umbilicus is reached distinct discomfort is felt unless relieved by passage of the air upward. In many instances it will be found that the inflating air passes readily beyond the sigmoid and shows its presence in the descending and transverse colon. It is now generally conceded that the ileocæcal valve allows air to pass during the process of inflation and its passage can at times be heard with the stethoscope applied in the right iliac fossa.

To further inflate the colon the rectal tube may be passed upward its whole length; we cannot be sure, however, that it will pass beyond the splenic flexure nor could further passage be expected. Inflation of the transverse and ascending colon and of the cæcum take place quite readily with the tube in this locality. The pain of extreme distention will always warn the operator when to temporarily moderate the air-pressure, which, however, is usually relieved at this stage by the passage of air upward through the ileocæcal valve. Detaching the rectal tube from the inflating apparatus allows the bowel to return to its normal state, by expelling the contained air.

Carefully applied inflation in connection with inspection and palpation may give important results. Idiopathic dilatation of the sigmoid flexure can be readily differentiated from abdominal distention due to other causes, the sigmoid clearly outlining itself as it rises from and descends again into the left iliac fossa, often overlying the rest of the abdominal contents in its sweep upward and to the right. Tumors, malignant or other strictures of the bowel, fecal accumulations may be brought up from the pelvis into sight and touch. The position of the colon and cæcum can be readily outlined, visibly in thin subjects, by percussion and palpation in those stouter and more muscular, although the examiner can always see that inflation is going on by the puffing up of the various regions. One must know that at both the splenic and hepatic flexure the bowel will be less prominent than elsewhere. The same pathological conditions mentioned in connection with the sigmoid flexure may be shown in the colon. One would naturally expect that complete strictures from any cause would prevent passage of air upward or downward. In such cases the distention and condition (muscular hypertrophy, visible peristalsis) above the stricture may tell us as much or more than inflation from below; and further, in such conditions the diagnosis is rarely in doubt. Incomplete or partial strictures, whose symptoms may be very obscure, are at times clearly brought out by the rapid inflation from below, as a sudden narrowing above the dilated lower part.

Easily recognized is the displacement of the colon, particularly the transverse colon in enteroptosis. The transverse colon may lie below the umbilicus, or even in the pelvis, the common appearance on inflation being a shallow V-shaped protuberance, the arms of the V running up to the liver and spleen. The relation of the bowel to retroperitoneal and other tumors is more easily determined by inflation than by any other means. Inflation of the bowel above the ileocæcal valve doubtless may be of value. Its application has given but uncertain results. Inflation of the bowel as a test for perforation is now universally condemned. Many cases of flatulence supposedly due to gastric distention can be found to be due to distention of the colon. Inflation is a valuable aid in the differential diagnosis of these conditions.

Röntgen-ray Examination of the Intestines.—Large and small solid tumors, thickening and muscular hypertrophy can at times be made out by the fluoroscope or in skiagrams. The data obtained by this method are, however, usually confirmative of facts elicited by the anamnesis and the above described methods. Scybalous masses present no different shadow from that of organic disease. Lining the intestine by continuous doses of bismuth allows the coils to be readily photographed, and under this condition peristalsis can be readily observed by the fluoroscope and the rate of progress of fecal matter observed. More feasible and of distinct value in recognizing displacements of the colon is the injection of suspensions of bismuth in large quantities. Very serviceable photographs can be secured by this method. Localization of small metallic or other solid foreign bodies in the intestines is remarkably facilitated by the X-rays.

So-called "test lavage" is used at times to bring away secretion or material from the large bowel: mucus, blood, ulcerating fragments of new growths. The examination of the sediment of such washings at times gives distinct help. It can only be satisfactorily performed when the large bowel has been previously completely emptied.

It has been suggested that dilatation of the duodenum, with stricture beyond, can be diagnosed by filling the stomach and duodenum with water through a stomach-tube. Dulness and distention toward or in the right hypochondrium and the fact that the fluid may return as does the fluid from an hour-glass stomach, part at once and the rest a few minutes later, is considered suggestive. An inflation that outlines the stomach and produces an extra prominence in the right hypochondrium would be equally suggestive.

Fæces.—The accurate determination of many points with regard to the fæces is difficult, owing to the wide variations in their composition and to the fact that the establishment of a normal or standard bowel movement requires the continued administration of certain standard diets for several successive days.

Various standard diets are recommended, the simplest being milk, since it contains fat, proteids, and carbohydrates.

1. Milk, 8 oz. every two hours from 8 A.M. to 10 P.M., amounting to 4 pints in the twenty-four hours.

2. That of Schmidt is more complicated, but approaches more nearly a general diet:

7.30 A.M.	Milk, 17½ oz., and 6 biscuits.
9.00 A.M.	Gruel, 1½ oz. oatmeal, 1 egg, 2 biscuits, ⅓ oz. butter, 7 oz. milk, 10¼ oz. water.
1.00 P.M.	Minced beef, 4½ oz. raw weight, lightly fried in ⅓ oz. butter, leaving the interior raw, and potato purée—7 oz. mashed potatoes, 7 oz. milk, ⅓ oz. butter.
4.30 P.M.	Milk, 17½ oz.
7.30 P.M.	Same as at 9 A.M.

3. A "mixed diet" is more liberal and better borne, but the necessary cooking makes the eventual microscopical examination much less satisfactory than either of the preceding:

8.00 A.M.	10 oz. hot water.
9.00 A.M.	3 oz. fresh fish, 4 biscuits, ⅓ oz. butter, 10 oz. tea, 2 oz. milk.
12.00 M.	10 oz. hot water.
1.00 P.M.	3 oz. mutton, 3 oz. cabbage, 4 biscuits, ⅓ oz. butter, rice pudding (½ oz. rice in 10 oz. milk).
4.30 P.M.	10 oz. tea, 2 oz. milk, 2 biscuits.
6.00 P.M.	10 oz. hot water.
7.00 P.M.	3 oz. fresh fish, 3 oz. chicken, 3 oz. spinach, rice pudding (as before), 2 biscuits, ⅓ oz. butter.
10.00 P.M.	10 oz. milk.

4. A meat diet: ¼ lb. finely minced beef every three hours and 10 ounces hot water one hour before meal-time. It contains no carbohydrates.

The first dejecta usually appear in from twenty-four to forty hours after the standard meal has been given. Radioscopic examination of the intestine and the passage of its contents shows that in about seven hours the ileocæcal valve has been reached by part of the residue, which may now remain four hours in the colon, three hours in the sigmoid flexure and rectum before being expelled.

Attempts to describe bowel movements resulting from standard diets have been made.

1. FÆCES RESULTING FROM MILK DIET.

Amount.—

Quantity of milk in 24 hrs.	Fæces excreted, average weight in Gm.
4 pints	135.2 Gm.
5 pints	151 Gm.
6 pints	198 Gm.

Color.—Yellow-white, or white tinged with orange.

Consistency.—Not well formed, tending to be lumpy; rolls of fecal matter not homogeneous but composed of lumps welded together, or firm sausage-shaped masses plus soft paste.

Odor.—Not offensive; more like stale cheese than fæces.

If constipation exists, a tendency to isolated scybala of pale color is seen, often firm, hard, and dry enough to rattle in the vessel, and to break up like dry clay, with an earthy odor.

With diarrhœa a milk diet gives fæces resembling Devonshire cream—sticky, but capable of being poured from one vessel to another. Gas bubbles and froth are seen on shaking, and the odor is that of decomposed cheese or putrid proteid.

Caseous flocculi, the evidences of disturbed digestion, are readily recognized as bright white, small, fibrillary-looking, friable masses.

2. FÆCES RESULTING FROM THE SCHMIDT DIET.

Amount.—Smaller than that from the milk diet. Average 90 Gm.

Color.—Light brownish-yellow, darker on the outside than inside.

Consistency.—Well formed rolls or sausage-shaped masses, as a rule. These readily break up on drying.

Odor.—Distinctly fecal.

In constipation on a Schmidt diet lumps of fecal matter are massed together, or isolated scybala are seen.

In diarrhœa on this diet the fæces resemble closely those of a patient on a milk diet.

3. FÆCES RESULTING FROM A MIXED DIET.

Amount.—Average 102 Gm.

Color. — Nut-brown, olive-green (chlorophyll of vegetables), varies much from day to day.

Consistency and Form.—Usually large, firm, roll or sausage-like motions. On drying break up easily.

Odor.—Fecal.

In constipation the fæces of a mixed diet are usually dark brown or black scybala with pressure facets and mucus in the crevices. They may be of stony hardness and not offensive.

In diarrhœa the motions are dark brown or nearly black, of thick sticky or pasty consistence with small scybala. Soft movements in general from a mixed diet have most offensive odors. An increase of the quantity of milk in mixed diets makes the stools paler and less firm.

4. MEAT DIET.

Amount.—Average 54 Gm.

Color.—Dark brown to black.

Consistency and Form.—Firm rolls, 2 to 3 inches in length.

Odor.—Fecal but very offensive.

Variations in consistency and form, in odor, and in color naturally depend on local conditions and the time the fecal material is retained in the large bowel. The amount is important, but several days are required to get the proper average. The formation of scybala, according to these results, may take place in a very few days.

The fæces are composed of:

1. Food remains.
    (a) Indigestible remnants.
    (b) Digestible but not absorbed remains.
2. The remains of the digestive secretion.
3. Products resulting from the digestion of food in the intestinal canal.
4. Formed and unformed products of the intestinal mucosa.
5. Bacteria.
6. Various substances introduced accidentally from without; various concrements, gall-stones, intestinal stones. parasites, cotton, wool, or linen fibres.

The fæces are collected in a bed-pan or any large clean vessel.

In the study of any question of absorption or excretion the rule is to place the patient on one of the standard diets for at least four days before beginning any estimation. The administration of some coloring matter such as charcoal or carmine with the first meals of the standard diet will render easy the recognition of their first dejecta.

The fæces are to be examined macroscopically, microscopically, and chemically.

Fermentation.—Normal firm bowel movements will usually dry without appreciable gas formation, and even semisolid or pultaceous stools ordinarily produce only a small amount. A stool which on standing shows evidence of fermentation by the production of gas bubbles or a distinct frothy layer, or gas bubbles in such abundance as to give a pale appearance to a more or less solid stool, should be considered pathological and examined for fermentable products—carbohydrates.

Excess of neutral fat in the stools can be readily noted. The normal bowel movement leaves no greasy mark upon a vessel containing it. Neutral fat will show itself in the gross examination either as a very pale, white, distinctly greasy bowel movement, or if the stool be liquid the fat may rise to the top, forming the characteristic appearance of melted fat, and on cooling may partially or completely solidify. The soaps in ordinary amounts and the fatty acids are not macroscopically recognizable.

Excess of proteids in the fæces, when in the form of meat, can often be recognized by the appearance of numerous reddish points throughout the bowel movement. One must be certain that other coloring or colored matters have not been ingested. Casein shows itself as the familiar white flocculi, easily disintegrated, much denser white than mucus. Undissolved connective tissue has the appearance of fine cotton-wool fibres and can be removed for further examination. Other substances to be considered in the gross examination are mucus, blood, pus, foreign bodies, and parasites. Small amounts of mucus are always present, but require search to demonstrate their presence. A constipated stool often shows flakes of dense white mucus in the interstices of the firm masses, or mucus may follow the movement.

Brownish, gelatinous-looking mucus, colored by the bile pigments, usually comes from the small intestine; colorless mucus and that appearing as denser, whiter masses and flakes, from the colon. Tubular masses from the large intestine, sometimes many centimetres in length, are seen in membranous colitis. Floating or softening these masses or strands in water will usually determine their character. Unformed mucus, particularly in liquid stools, sometimes on standing accumulates in masses as large as a hen's egg.

Fresh blood can be easily recognized. Unless quickly voided, blood in the intestines becomes black and small amounts do not show in the stools. Large amounts appear as "tarry stools"—large black masses, clots too large to be broken up or absorbed.

Pus is usually quickly disintegrated. Fresh pus which retains its appearance is practically always from the sigmoid or rectum.

Parasites are described in another section. Many food remains are detected at a glance: fruit stones and seeds, skin of fruit, vegetables, food pulp of oranges, grape fruit, lemons, large masses of connective tissue, bones, etc.

For the more careful examination various simple plans are recommended. In examining the whole quantity of fæces an ordinary fine sieve on which running water can play enables one to collect the larger foreign bodies and solid material; or the fæces are placed in a large vessel with water and thoroughly broken up. Mucus, woody fibres, smaller seeds, and bacteria float and can be removed by pouring off after settling. By repeating the process several times a residue of solid matter, deodorized and decolorized, is obtained. Gall-stones, pancreatic calculi, muscle fibres, connective tissue, casein, parasites, are easily looked for in this way.

Spreading the fæces on a glass plate with a dark background facilitates the examination. Pieces of connective tissue, muscle fibres, casein, foreign bodies, or anything differing from the homogeneous fecal matter may be readily found in this way.

MICROSCOPICAL EXAMINATION.—MIXED DIET.—A small piece of fecal matter can be taken from the stool after it has been mixed in a mortar or a vessel, or several loopsful of a liquid stool can be smeared on a slide. A cover-glass is preferable if high power is used. For a low-power examination a glass 3 or 4 inches square on which a comparatively large amount of fæces has been thinly spread, can be placed on the stage of the microscope. A large area can be quickly gone over in this way.

Masses of mucus, blood, or pus, meat fibres, etc., should be picked off for separate examination before mixing the fecal material. Schmidt recommends taking three separate specimens of softened fæces. No. 1 is examined direct. In it we can note much fibre, colorless soaps, neutral fat if present, small and large yellow salts of calcium. No. 2 is stirred with a small drop of 30 per cent. acetic acid heated for a moment until it begins to boil, then covered with a cover-glass. After cooling, small flakes of fatty acids appear. The soap flakes and calcium salts will have disappeared. No. 3 is rubbed up with a drop of Lugol's solution. Under the microscope unaltered starch will assume a violet color.

Since 85 per cent. or more of the food is digested and absorbed, and since of the remainder a portion is in the shape of products—albumoses, fatty acid, soaps, dextrin, etc., little unaltered food is present in the specimen. Easily recognized are the bacteria which make up practically one-third of the dry substance of the stool. Acid-fast bacilli may be tubercle or smegma bacilli. Leptothrix threads are easily recognized. Epithelial cells in considerable numbers are always present. They are usually of the smaller round type, and show evidences of digestion or disintegration. No deduction can be drawn from their number or form as to conditions in the bowel. Squamous epithelia from the mouth or from the food are occasionally seen. Structureless or faintly striated mucus in small amounts, bile-stained if from parts high up, pale if from lower down, may, by the number of leucocytes or epithelial cells entangled in it, give evidence of catarrhal conditions of the bowels. Mucus is less dense, less sharp in outline than connective tissue; acetic acid causes it to show faint striations. A few leucocytes are always present.

Food Remains. — Undigestible remnants of any kind may appear. Many of them are recognizable macroscopically. The framework of vegetables gives most varied pictures. Many of the structures suggest parasites and have frequently been mistaken for them. Remnants of undigested starch may be suspected by their pallid color and their cellular envelope. It is well to stain suspicious specimens with iodine and look for the blue stained masses; to judge whether starch is being excreted in excess is not easy with the microscope; the fermentation test is the more accurate method.

Two or three small meat fibres in a field, showing very dim or no striation, and with no remnants of nuclei, may be considered normal in patients on a mixed diet. Retention of the striation, persistence of the nuclei in good condition, and presence of meat fibres in numbers suggest disturbance of intestinal digestion, particularly that part related to the pancreas. It is not likely that anacidity of the gastric juice will show the same condition. Specimens from fæces of patients with pancreatic derangements may show meat fibres in such numbers that counting them in one field may be difficult or impossible. Some cases show excess of meat fibres in the stools if over 60 grammes of meat are taken per day.

Schmidt's Nucleus Test for Pancreatic Disease or Impairment of Pancreatic Function.—The disintegration or non-disintegration of the meat fibre nuclei in the centre of small balls of meat of standard size—¼ to ½ inch in diameter—kept together by non-digestible netting and given in the food, cannot be said to be positive enough for any certain deductions to be made. We can only say that if all the nuclei, even those on the outside of the balls, are found unaffected by digestion, pancreatic insufficiency is suggested. Connective tissue and elastic tissue are constantly present on a mixed diet, though in very small amounts. They are readily recognized by their dense and fibrillated appearance. Gastric juice readily digests connective tissue, and its persistent presence in large quantity must be taken as pointing to impaired gastric digestion. A few fat drops may be found on a mixed or meat diet, but more than eight to ten fat drops in a single field should attract attention. This neutral fat is easily seen as yellowish, oily looking drops of varying size and shape. Constant presence of the flakes of the "higher melting point" fat, and the flaky needle-like crystals of the fatty acids, or of the flake or disk crystals of the soaps, is to be considered as abnormal. Gentle heating of the slide will dissolve the crystals and flakes of the fatty acid and soap. Triple phosphate crystals, colorless and of characteristic shape, neutral phosphate of lime crystals, colorless, or the yellow calcium salts (sometimes bile stained) are commonly found. Oxalate of lime crystals are usual in a mixed diet. Their presence in the fæces when no vegetables are being eaten is said to indicate some intestinal disorder. Cholesterin crystals, Charcot-Leyden crystals, especially if much mucin is present, are both found in the fæces. Very frequently present are the so-called "yellow bodies": large lumps of bright yellow material, structureless, often surrounded by mucus, and recognized macroscopically. They give a proteid reaction. They are considered to be albumin, bile stained, and, when in great amount together with much mucus, indicate some disturbance of proteid digestion.

Casein flocculi are seen microscopically as almost structureless masses, finely fibrillated and enclosing fat droplets in their meshes.

Hairs, cotton and linen fibres, are common in the stools, being taken in with the food in large numbers.

CHEMICAL EXAMINATION.—In health the **fæces have a** neutral or faintly alkaline reaction. On standing this becomes faintly acid. Stools with excess of carbohydrates ferment and give a strong acid reaction. Excess of fat, fatty acid, gives faintly acid stools. Decomposition of excess of proteid matter produces an alkaline reaction. A mixed diet in health causes neutral fæces; a pure proteid diet produces alkaline fæces; a pure carbohydrate diet produces acid fæces; a diet of fats produces acid fæces. Only freshly passed fæces can be used in testing. A markedly acid reaction in fresh fæces suggests fermentative changes from undigested carbohydrates. "Acid diarrhœas," so-called, may be associated with hyperacidity of the stomach and insufficiency of the biliary and pancreatic secretion.

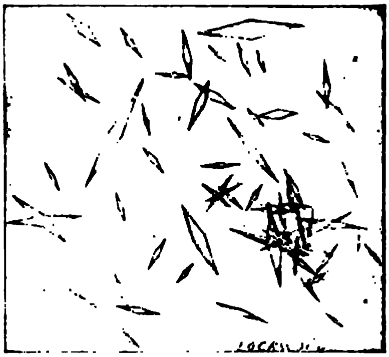

FIG. 91.—Charcot-Leyden crystals from the stools. × 400.—Emerson.

The test for hydrobilirubin or the bile products is important, since they may be present in colorless stools. The fæces are stirred up with a concentrated solution of mercuric chloride; normal fæces are colored red; fæces containing unchanged bilirubin become green. The pale stool of the leucohydrobilirubin gives the red reaction. Absence of the red or green coloring is seen in fatty stools with complete acholia.

COMPOSITION.—From 74 to 84 per cent. of the fæces is water; 16–26 per cent. is dry substance. Of the dry substance 10–20 per cent. can be extracted with ether, *i.e.*, are fats. Over 90 per cent. of fats taken in are absorbed.

Fats.—Qualitative tests only can be considered. The fats are readily detected macroscopically and microscopically. They are excreted as neutral fats, soaps, and fatty acids. These have been described. Crystals and flakes melt readily. Extracting a small mass of fæces with ether and pouring the ether through a piece of filter paper will, if fats are in excess, give the characteristic appearance of oil on the paper.

From 2 to 6 per cent. of the dry substance is carbohydrate, usually dextrin. Tincture of iodine or Lugol's solution will stain unaltered starch blue; dextrin remains red. There is no reaction for sugar. Fermentation is the simplest test for excess of carbohydrate or carbohydrate residue. Schmidt's fermentation tube may be employed or one may note carefully the presence of gas formation in the freshly passed stool.

Proteids.—More than 85 per cent. of proteids taken into the body are absorbed. The proteid residue in health is partly from the food, partly from the disintegration of proteids of the body—leucin, tyrosin, indol, skatol, mucin, nuclein. Albumin and globulins, and their transformative products, albumoses, peptones, are not found normally. Their presence in the stools means either insufficient proteid digestion and absorption, or that the "postdigestive putrefaction" in the large intestine has not had time to take place. The simple tests for albumin and albumose can be applied after dissolving, mixing a small amount of fecal material in water, and filtering. Any inflammatory condition of the lower bowel will yield albumin in the fæces. Serous exudation higher up may undergo the natural digestive processes. Persistent intense diarrhœa, choleraic diarrhœa, can hurry materials through before digestion of albumin or albumoses has progressed, and fæces from these conditions may give albumin reactions from food taken or from serous exudation into the bowel as in typhoid fever, cholera, or dysentery.

"Total nitrogen" estimations are necessary to determine the relation of proteid output and intake. As of the fats and carbohydrates one can say of the proteids—for clinical purposes macro- and microscopical examinations yield more useful information.

Digestive ferments are not found. The pigmentary remains of the bile have been spoken of. Mention has been made of the various salts and crystals, phosphates, oxalates, cholesterin, etc., visible microscopically, remains of food digested, or of digestive procedure. Chemical tests show presence of bile salts, bile acids, leucin, tyrosin, xanthin, carnin, and proteid derivatives.

Occult Blood.—The most important chemical examination for practical purposes. Teichman's acid-hæmin test may be used, but others are simpler and more certain. They all depend upon altered hæmoglobin reactions. No examination of fæces can be considered complete unless a blood test has been made; since occult bleeding may go on indefinitely with no gross signs of blood in the fæces and no blood-corpuscles to be seen microscopically. All bleeding from the nose, gums, pharynx, lungs, and vagina must be excluded. No meat can be taken during the days on which the fæces are tested. It is best to wait for forty-eight hours or to mark a food period by giving charcoal, lycopodium, or carmine.

To perform the test we must first remove gross fat by shaking with ether; otherwise the final ether extract may be clouded. This is poured away and the residue is used. 10 c.c. of fluid fæces or 5 c.c. of solid fæces, broken up in 5 c.c. water, are treated with 3 c.c. glacial acetic acid, thoroughly mixed and shaken. This dissolves red blood-cells and sets free hæmoglobin or makes acid hæmatin. After standing a few minutes excess of ether, 20–30 c.c., is added and the mixture vigorously shaken and then allowed to separate. The overlying ether is poured off and the tests made with it as follows:

Turpentine-Guaiac Test.—To a few cubic centimetres of the above ethereal extract previously treated with a little alcohol are added 10 drops of freshly made guaiac tincture and 30 drops of turpentine. In the presence of blood pigment a distinctly blue color occurs. Sources of error are the recent eating of potatoes or other starchy food, iron as a medicine, or the presence of bile, saliva, milk, pus in considerable quantities, and urobilin. The reaction may fail in the presence of minute traces of blood.

Aloin Test.—Klinge and Shaer.—This test is extremely delicate. Foods containing hæmoglobin and all vegetables and drugs must be avoided for several days. The diet period must be determined by charcoal or lycopodium, not carmine. From 1 to 1.5 c.c. of turpentine are superimposed and then 0.5 c.c. of freshly made 3 per cent. aloin solution. The reaction consists in the rapid development at the line of contact of a bright rose-red color. In a doubtful case both these tests may be used.

Benzidin Test.—Schlesinger and Holt's Modification.—1. Concentrated Benzidin Solution: as much benzidin (Merck's benzidin puriss.) as will go on tip of table knife in 2 c.c. of glacial acetic acid; shake lightly. 2. Piece of fæces the size of a pea (or several c.c. of a distilled-water extract) suspended in one-fifth of a test-tube of water; close with cotton and boil. 3. Ten to twelve drops of benzidin solution put in test-tube and 2–2.5 c.c. of hydrogen peroxide (3 per cent.) added. To this add 1–3 drops of boiled fæces after mixing the latter by slightly shaking. Green to blue color is positive and appears in two minutes in a blood mixture of 1–200,000 strength; hence this test is 5–10 times as delicate as other tests.

In ulcerating carcinoma ventriculi, occult blood is continually present in the stools; in ulcus ventriculi there are intervals in which no occult blood can be detected; in intestinal tuberculosis it is absent; in enteric fever it may occur in the absence of gross hemorrhage or may antedate the latter by twenty-four or forty-eight hours.

IV.

THE EXAMINATION OF THE UPPER AIR-PASSAGES AND THE EAR.

RHINOSCOPY. LARYNGOSCOPY. OTOSCOPY.

General Considerations.—Local affections of the nose, throat, or ears may give rise either to local or constitutional symptoms, while constitutional diseases frequently produce local manifestations. For this reason dexterity in the use of the mechanical means by which we are enabled to distinguish between the manifestations of local and constitutional diseases, as observed in these organs, is of no less importance to the general clinician than to the specialist.

The instruments employed in a simple examination of the nose, throat, larynx, or ear are a head mirror for reflection of light, tongue depressor, laryngeal mirror, and nasal and aural specula. They are of varied designs, but any instrument to which the physician has become accustomed will usually meet the requirements of ordinary cases. Of far greater importance than the instrument to be employed is its careful manipulation. Every instrument must be carefully cleansed in the presence of the patient, both before and after using. The speculum should be slightly warmed over a spirit flame or gas burner before introduction into the nose or ear.

Either natural or artificial light, if sufficiently strong, can be condensed and reflected by the mirror to the point or area to be examined, and the source of the light may be either to the right or left of the patient.

The Examination of the Nose.

Anterior Rhinoscopy.—Excoriations around the margin of the nares are usually produced by acrid secretions, excessively acid or alkaline, which occur in the course of various infectious diseases, colds, nasal hydrorrhœa, syphilis, etc. Rhinoscopy has to do with the examination of the interior of the nose, for which purpose it is necessary to dilate the nostrils, one at a time, with a bivalve speculum, using care to avoid injury to the mucous membrane or unnecessarily annoy the patient by overdistention.

Structures Observed.—Under normal conditions, the patient sitting erect before the operator, with the head tilted slightly backward, the distended alæ should present clearly to view the lower turbinates, the middle and lower meati on the outer walls, the area opposite to these on the septum, and the floor. This constitutes about the lower third or respiratory portion of the nares. The area just within the nares on the lower anterior margin of the septum should be especially examined as the most frequent location of the source of hemorrhage.

Tilting the patient's head backward brings into view the upper or olfactory portion of the nostrils, the middle turbinate and superior meatus, rarely a small portion of the superior turbinate—the close proximity of the septum and outer wall preventing an exposed view of the ethmoid and sphenoid area, superior turbinate and points of entrance to the frontal sinus. It is this space that we frequently find bathed in pus in the case of purulent sinusitis, ethmoiditis, or antrum disease. As a rule, an accumulation of pus above the middle turbinate is an indication of disease of the ethmoid or frontal sinus, while if pus collects beneath the middle turbinate its source is probably from the antrum of Highmore. In case the whole naris is bathed in the purulent secretion, first cleanse the nostril, then have the patient lean forward or turn the head well toward the side involved in order to favor the discharge of fresh pus and determine its origin more clearly. Nasal polypi most frequently originate in this part of the nares, at the marginal mucosa of a turbinate which has undergone mucoid degeneration from necrotic tissue in the ethmoid cells; less often from the sphenoid sinus, which lies slightly below and posterior to the ethmoid cells. Beneath the middle turbinate is the only natural opening into the antrum of Highmore—the *ostium maxillare*—which, however, in many cases is so obscure as to be found with difficulty even by experienced rhinologists. Occasionally two or more openings enter the antrum at variable points, even as high as the floor of the orbit. The inferior meatus is important for two reasons: first, it is beneath the lower turbinate that we find the nasal opening of the lachrymal duct, which may become occluded from either an acute or chronic enlargement of the turbinate; second, because of the thinness of the bony wall dividing the nares from the antrum of Highmore, through which a cannula may be easily introduced for diagnostic purposes in suspected purulent infection of the sinus.

If on first looking into the nose the view is obstructed by an intumescent condition of the membrane, which is found in nearly every local congestion, whether active or passive, the difficulty of obtaining a satisfactory view will be greatly obviated by the introduction of a small pledget of cotton dipped into a solution of cocaine and camphor, each two grains to the ounce of liquid albolene. The objection to the adrenalin preparations in examination is threefold: first, it frequently acts as an irritant, throwing the patient into a violent state of sneezing; second, by the intense bleaching of the membrane; and, thirdly, because of the aggravated congestion which follows its use. All the accessory cavities herein referred to are in direct communication with the nares; each sinus or cell is lined by mucous membrane, somewhat modified in character from that in the nasal chambers, and any inflammatory process in one cavity may cause more or less irritation in one or all of the others.

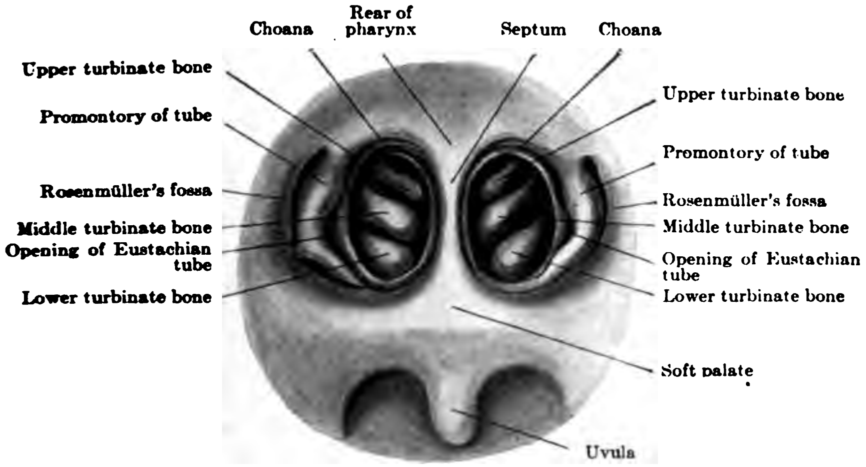

Fig. 92.—Normal posterior nares, view obtained by repeated change of the mirror.

Posterior Rhinoscopy.—To examine the nasopharynx there are needed a head mirror, tongue depressor, and rhinoscopic mirror. Some persons are able to depress their tongues by voluntary muscular effort, in which case the depressor is not needed. There is also a great difference in the ability of individuals to relax the soft palate at will, thus allowing an unobstructed vision in the mirror of the vault of the pharynx and the posterior nares. The process of such examinations will often require great patience if the pharynx be hypersensitive, since the slightest touch with the mirror may produce gagging.

Let the patient sit comfortably in the chair and assure him that there will be nothing connected with the examination to cause either pain or discomfort. There is a general tendency to hold the breath and strain on the pharyngeal muscles. To obviate these difficulties explain that it is important to allow the mouth to open widely and easily, without the slightest tension of the jaw, leaving the tongue at rest in its natural position, and to breathe quietly and freely through the mouth. The probability is that after this reassurance the soft palate will relax to its normal position. A common difficulty consists in the involuntary retraction of the soft palate tightly against the pharyngeal wall as soon as the mirror approaches the mouth, and its retention in that position until the mirror is withdrawn. This frequently can be obviated by having the patient close his eyes. Should this fail, the most satisfactory recourse left is cocainization to a degree sufficient to relieve the hypersensitiveness, when with a long applicator, bent at right angles, making a hook about three-fourths of an inch long on the end, the soft palate may be gently drawn forward, and the rhinoscope placed in position to reflect the image desired. It is always better to twist a small piece of cotton on the retractor, which being dipped into a bland oil will prevent injury to the mucous membrane.

Structures Observed.—With the rhinoscope just below and posterior to the margin of the soft palate, and with a strong light, the angle of reflection in the mirror may be so directed by manipulation as to show successively all the structures in the nasopharynx, viz., the Eustachian orifices on the extreme outer margins, and just above and slightly posterior to these the fossæ of Rosenmüller, which are occasionally obstructed by adhesive bands; in each naris are seen the middle and lower turbinates, the latter being indistinct except over its upper half; and directly posterior and below the posterior margin of the septum on the pharygneal wall is the usual position of the pharyngeal tonsil or adenoids. Since this lymphoid structure under normal conditions undergoes atrophy about the age of puberty, when observed in adults, or when sufficiently large in children to interfere with nasal respiration, it should be regarded as pathologic. Polypoid growths in the nasopharynx originate usually from mucoid degeneration of the posterior margins of the middle or superior turbinates or from the posterior ethmoid cells; fibromata, sufficiently large to fill the entire vault, suspended by a small pedicle and hanging low enough in the oropharynx for the lower margin to be seen by direct vision, are not infrequently observed. Posterior rhinoscopy is seldom accomplished in children with any degree of satisfaction, in which case ocular inspection must be supplanted by digital examination.

Laryngoscopy.

For the examination of the laryngopharynx, larynx, and trachea the same instruments are required as those used for posterior rhinoscopy, and the same precautions toward preventing nervousness on the part of the patient during examination are of even greater importance. The tongue depressor will not be needed in all cases, since in some a better view can be obtained by grasping the tip of the tongue with a towel or handkerchief and drawing it well out and downward, using care not to cause pain underneath the tongue by too forceful traction over the lower teeth. In still others the patient may be able voluntarily to depress the tongue.

The oropharynx is examined by direct inspection. The appearance and color of the mucous membrane of the posterior pharyngeal wall vary greatly according to the condition of the gastro-intestinal tract. The redness frequently observed along the anterior borders of the faucial tonsillar pillars in gouty or lithæmic individuals is a sign of diagnostic importance. This may vary in color from a dark pink blush to a purplish crimson, and may be regular in outline or occasionally present the appearance of petechial spots, particularly on the uvula. Another phenomenon often observed is indicative of either acute or chronic inflammatory Eustachian or middle-ear involvement. It consists of a prominence or bulging of the postpharyngeal wall, evidently an inflammatory infiltrate, just back of the posterior faucial pillar on the same side as that of the affected ear.

In the examination of the laryngopharynx the laryngoscope is used. Observe the base of the tongue carefully to detect the presence of an enlarged lingual tonsil, which gives rise to various annoying symptoms, most prominent of which is the constant accumulation of mucus about the glottis and the resulting pharyngeal tenesmus. Occasionally this mass of tonsillar tissue is sufficient to press the epiglottis downward and thus interfere with the examination of the larynx proper.

Foreign Bodies. — The most frequent locations of foreign bodies, such as broken bits of toothpicks or match-sticks, fish-bones, tooth-brush bristles, etc., in the laryngopharynx are the glosso-epiglottidean pouches at the base of the tongue, or else in the sinus pyriformis which lies partially posterior to and on either side of the glottis. The patient's sensation of locality of a foreign body in such a position is frequently misleading; for instance, a fish-bone or bristle sticking in the base of the tongue may give the sensation of being farther down in the larynx, or perhaps even in the nasopharynx.

Examination.—A strong, well focussed light is essential, and whether the patient be in the sitting or recumbent position, the head must be well extended and free breathing through the mouth insisted upon. The autoscope, an instrument devised some years ago for the purpose of making direct inspection of the larynx, is not generally employed at the present time. Proceeding with the usual method, the patient's tongue is depressed, or drawn outward, the laryngoscope is carefully introduced into the upper laryngopharynx in a manner that will push the uvula backward out of range of the reflected laryngeal image. The best angle of reflection can be determined according to the case in hand, the epiglottis, owing to its variability both in point of shape and position in different individuals, being the principal obstacle to a clear view of the underlying structures. This difficulty, however, can best be obviated by the influence which the effort on the part of the patient to produce certain vocal tones has upon the position of the larynx. Two vocal sounds are utilized; first, the classic "ah," during the intonation of which the larynx is in the most natural relation to the surrounding structures at rest, except for the fact that the cords are approximated or in the position of phonation. With the parts in this position there will be reflected in the laryngoscope the edge of the epiglottis and a narrow margin of its underlying surface, the arytenoids, and the posterior half of each vocal cord, which appears in the mirror as the inferior half.

The same relation will still be preserved if the patient now be instructed simply to breathe, allowing the arytenoids and hence the cords to swing freely open. But to obtain an image of the junction of the cords at the anterior ends, appearing superiorly in the mirror, the effort to produce the vowel tone "e" must be made. This will so elevate the larynx and change its position in relation to the epiglottis and other structures as to expose the whole length of the cords and the whole inferior surface of the epiglottis in one view, and in most cases, after holding the tone for a few seconds, the patient may breathe freely without the tongue falling back to its original position. A good plan is to have the patient hold the note for a moment, followed by free respiration, and repeat the process as often as required till a satisfactory view is obtained of all the intralaryngeal structures. During respiration the anterior wall of the trachea also may be seen, in some cases as far down as the bifurcation, though to be satisfactory an examination of the lower part of the trachea and bronchial tubes should be made with a bronchoscope. This instrument has been perfected in recent years to such an extent as to be of great value in the hands of a skilful operator for the removal of foreign bodies or for the inspection of any diseased condition of the lining membrane. If during the examination the patient has an inclination to gag, free and rapid respiration may overcome it; should the tendency persist, however, withdraw the mirror and allow the throat to be at rest for a short time; under no condition will anything be gained by forcing or attempting to prolong an examination when the patient coughs, gags, or the muscles of the throat become fatigued.

The larynx is subject to the same inflammatory changes which may take place in any other mucous membrane, and likewise to any local infection. The histologic structure of the submucous tissue seems to favor rapid and extensive œdema from local inflammations, due to traumata, scalds, and the inhalation of irritant vapors; from infectious processes involving adjacent structures, as diphtheria, follicular tonsillitis, and tuberculosis; and from circulatory disturbances such as may arise from cardiac or renal lesions.

Chronic hoarseness not amenable to treatment, particularly in individuals past forty years of age, must be regarded as suspiciously indicative of malignancy and be kept constantly under observation in order that should such a condition exist it may be detected at the earliest stage possible. Sluggishness in the movement of the vocal cord, or even an apparent paralysis of the cord on the affected side, has been observed not infrequently in laryngeal carcinoma long before any actual tumor was visible.

Otoscopy.

For convenience in description the organ of hearing is usually divided into the external, middle, and internal ear. The last embraces that part of the petrous portion of the temporal bone in which the terminal fila-

DESCRIPTION OF PLATE III.

1. **Laryngeal image during respiration.**
2. **Laryngeal image during phonation.**
3. **Laryngoscopic picture in a case of paralysis of the right recurrent laryngeal nerve.**
4. **Laryngoscopic picture in a case of bilateral paralysis of the recurrent laryngeal nerves.**
5. **Laryngoscopic picture in a case of paralysis of the interarytenoid muscle.**
6. **Position of the vocal cords in unilateral adductor paralysis.**
7. **Position of the vocal cords in bilateral adductor paralysis—during efforts at deep inspiration.**
8. **Position of the vocal cords in paralysis of the right internal tensor.**

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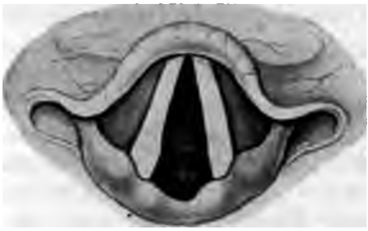

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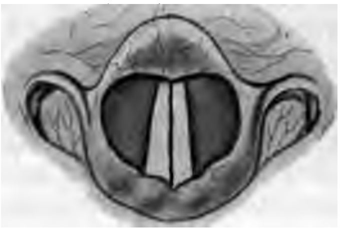

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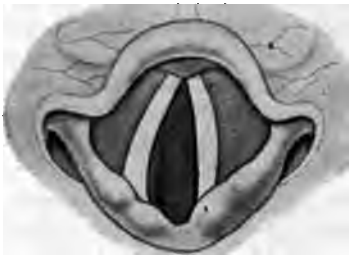

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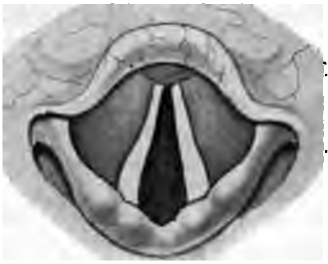

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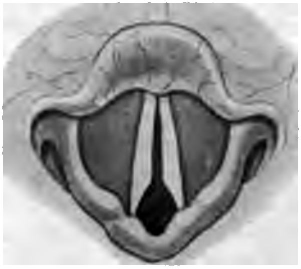

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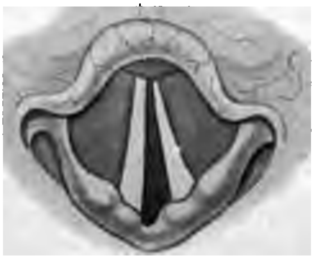

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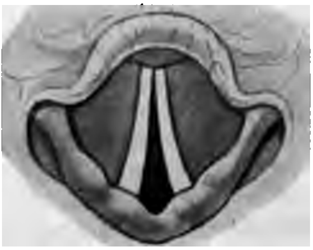

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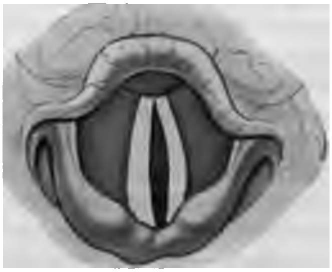

ments of the auditory nerve are distributed, and therefore is also designated as the sound-perceiving apparatus. The external and middle ear, since they serve the purpose of transmitting sound impressions to the nerve, are called the sound-conducting apparatus.

It is of importance to distinguish between diseased conditions of the sound-perceiving and the sound-conducting apparatus, or between disturbance of hearing caused by nerve lesions and that dependent upon diseased structures of the ear itself. For example, in any case of deafness the first thing to be ascertained is what part of the ear, if any, is at fault. Deafness, either partial or complete, may be caused by obstructions in the external auditory canal, such as foreign bodies, impacted cerumen, congenital atresia, exostosis, furunculosis, etc., and also by hemorrhage into the semicircular canals, or as the effect of certain drugs. The condition of the external canal and the tympanic membrane can easily be determined by direct ocular inspection, a strong, well focussed light being directed into the canal through a suitable speculum. If the canal be found clear, then the difficulty must lie either in the middle or the internal ear. To distinguish between these the tuning-fork test, devised by Weber, is usually employed.

External Auditory Canal.—The external auditory canal varies greatly in size and somewhat in direction in different individuals. The cartilaginous portion of the canal is usually directed more or less downward and forward, so that in order to bring this part of the canal and the bony meatus into the same axis for inspection of the walls of the canal and the drum membrane it is necessary to draw the auricle gently upward and backward. By holding the auricle in this position with one hand and manipulating the speculum with the other—a metallic conical speculum is the most desirable—every part of the canal wall and drum membrane may be clearly seen. Note the size of the canal and any acute inflammatory swelling or chronic induration. The cartilaginous portion of the canal comprises a little over one-third of the whole length of the meatus. Its junction with the bony meatus is the most frequent site of furunculosis. In young children the cartilaginous meatus comprises about two-thirds of the whole extent of the canal. When a furuncle is of deep origin pus may burrow beneath the periosteum inward toward the tympanic cavity, occluding the osseous meatus entirely and giving rise to most excruciating pain. The pain within the ear and swelling extending even back of the auricle may be confused with acute mastoiditis. In furunculosis the most acute pain is apt to be elicited by pressing upon the tragus, or, if there be postauricular tenderness, it will likely be superficial; in mastoiditis, however, the pain may be slight superficially and intensified by deep pressure over the mastoid, and pain is not apt to be elicited on pressure over the tragus.

When the cartilaginous portion of the canal is occluded by swelling, gently insert a tightly rolled pledget of cotton dipped in a solution composed of camphor and carbolic acid, equal parts, and allow it to remain a few minutes. The swelling is thus sufficiently reduced to allow the introduction of a small speculum for the examination of the deeper canal and tympanic membrane. This solution also produces partial anæsthesia of the membrane, thus allowing a more thorough examination.

The tympanic membrane separating the external canal from the tympanum, irregularly oval in shape and slightly concave in its normal state, is affected to some degree by every inflammatory disease of the middle ear, both acute and chronic, and should therefore receive most careful attention in every aural examination. A strong, well focussed light is a necessity and the largest speculum which the canal will admit should be used. The external layer of the drum membrane is modified skin, clear and almost translucent in its normal condition, and through it can be seen the impression of the malleus with which it lies in direct contact. Acute inflammations of the middle ear produce a pink or reddish hue along the margins of the malleus and in some cases over the entire membrane.

Exudates, serous or purulent, in the tympanic cavity, even though small in quantity, produce bulging of the membrane and frequently terminate in spontaneous rupture into the external canal. In acute and chronic inflammations causing occlusion of the Eustachian tube the tympanic membrane will be found retracted. Retraction may also be brought about by adhesions within the tympanic cavity following marked inflammatory involvement. In a case of retracted membrane we can ascertain whether the tube is patulous by one of the usual methods of inflation. Valsalva's consists of a vigorous expiratory effort while the nose and mouth are kept closed. Politzer inflates the tympanum through one nostril by compression of a rubber air-bag while the patient is in the act of swallowing. The opposite nostril and the mouth are closed. Eustachian catheterization is the most satisfactory method in difficult cases. With Siegel's otoscope the air within the external auditory canal can be exhausted and adhesions involving the tympanic membrane observed. Aural polypi originate most frequently within the middle ear from granular or necrotic tissue and protrude into the external canal through perforations in the tympanic membrane, though they occasionally may be found in any part of the canal, particularly at the cartilaginous and osseous junction. Exostoses occur in any portion of the osseous canal, particularly from the posterior wall and from the osseous and cartilaginous junction. In chronic non-suppurative processes involving the **middle ear,** the drum membrane becomes opaque and thickened, and usually distorted in shape. In cases of otosclerosis white chalky spots are observed in the membrane which may otherwise appear normal. A sign of diagnostic importance in mastoiditis complicating chronic suppurations of the middle ear is an infiltration of the membrane covering the superior posterior osseous wall of the external auditory canal, presenting the appearance of a circumscribed drooping or bulging.

Pharynx and Eustachian Tube. — No examination of the ear can be considered complete without a careful inspection of the nasopharynx at the entrance of the Eustachian tube slightly below and anterior to the fossa of Rosenmüller. The technic of this procedure is described under posterior rhinoscopy. Catheterization of the tube for diagnostic purposes can be accomplished either through the nose or by way of the oropharynx.

V.

THE EXAMINATION OF THE BLOOD.

General Considerations. — Information derived from blood examinations, while not essential in the establishment of a diagnosis in most instances, is frequently a useful aid. Negative blood reports are often important in diagnosis, as in the exclusion of malaria from a group of diseases which have similar clinical features, such as malignant endocarditis, septicæmia, and certain types of tuberculosis. Diseases associated with marked splenic or glandular enlargement present so close a resemblance to leukæmia that only a study of the blood can exclude the latter condition. Many blood examinations elicit results which assist in arriving at or completing a diagnosis. Evidence of a pathognomonic character gained from hæmatological studies is available in only a limited number of diseases, notably in myelogenous leukæmia, malaria, relapsing fever, trypanosomiasis, and filariasis. The condition of the blood as to hæmoglobin value, the number of erythrocytes and leucocytes, may serve as an index of body nutrition. Blood counts often yield information which bears upon prognosis,—*e.g.*, in chlorosis a steady hæmoglobin rise is an evidence of favorable progress of the patient, while an erythrocytic gain in progressive pernicious anæmia or leucocytic decrease in leukæmia likewise points to improvement. Counts of the white corpuscles also aid in establishing the leucocytic standard of the patient. The opsonic index and the agglutination phenomenon are recognized adjuncts in the field of diagnosis.

Methods of Blood Examination.

Obtaining Blood. — For most clinical examinations a few drops of blood, obtained from a puncture in the lobe of the ear or the finger-tip, will suffice. The lobe of the ear is sometimes selected for making the puncture on account of its lessened sensibility and because the operation can be performed without the patient seeing it, but the finger-tip is generally chosen as this site is more convenient for the examiner. The puncture should be made with a lancet-shaped or triangular surgical needle, an instrument especially devised for this purpose, or a steel pen with one of the nibs broken off. The part selected should be cleansed with alcohol or with soap and water followed by alcohol, and dried with a towel or handkerchief. If not warm, the skin is warmed by gentle friction, but forcible rubbing should be avoided, since it excites active hyperæmia. If the individual is a bleeder, the precaution of making a superficial puncture and of having measures at hand to control hemorrhage should be observed. It is obvious that areas of œdema and of inflammation must be avoided. If the former be present about the hands or ear, an area free or nearly so of œdema is chosen. The puncture is made with a quick thrust of the instrument, which has previously been cleansed with alcohol or passed through a flame. The first drop or two of blood should be wiped away.

Forcible squeezing of the tissues in the immediate vicinity of the wound must be avoided, as this may alter the composition of the blood by the addition of lymph fluids. As the blood flows from the wound, its gross appearance as to color and fluidity is noted.

Preparation of Fresh Blood for Immediate Examination. — A cover-glass is applied to a droplet of blood and then placed upon a clean slide. The blood usually spreads into a thin layer. Warming the slide by friction with a piece of gauze, a handkerchief, or tissue paper before applying the cover-glass facilitates spreading. In a well-prepared preparation the corpuscles are arranged in a single layer separated from each other over an area sufficiently large for the desired study. If it be necessary to delay the examination, drying of the specimen can be prevented by ringing the margins of the cover-glass with vaseline or cedar oil.

Preparation of Blood for Staining.—Cover-glasses which have clean polished surfaces are placed upon a sheet of paper, or preferably upon a folded towel, from which they can be picked up easily. A cover-glass, held with the fingers, or with forceps, is applied to the summit of the droplet of blood, being careful to avoid touching the skin, and allowed to fall upon another cover. As soon as the blood has ceased spreading, the covers are slid apart. In performing this operation, care should be exercised not to lift the glasses apart: the sliding motion must be performed rapidly, avoiding a jerky uneven stroke. The smear may be made upon a slide by placing a drop of blood upon it and spreading with another slide or with a glass rod especially designed for this purpose.

Methods of Fixation.—HEAT FIXATION.—The covers are placed in an oven, the ordinary dry-heat sterilizer being convenient for this purpose, and heated gradually until the temperature reaches to 120° C. or up to 155° C. and subjected to this temperature for from ten to twenty minutes. A convenient plan for fixing the specimens with heat consists in placing the spreads upon a heated copper plate. The plate, about 20 centimetres in length, 8 centimetres in width, and from ½ to 1 centimetre in thickness, supported by a suitable stand, is heated at one end with the flame from a Bunsen burner or an alcohol lamp. When the plate is thoroughly heated, the covers are placed upon it at a point where the temperature is sufficient to boil water (which is previously determined by dropping water upon its surface, beginning at the end farthest away from the flame) and exposed to this heat for about 30 minutes. A method less suitable than the ones mentioned consists in passing the film rapidly through a Bunsen flame forty or fifty times.

FIXATION BY WET METHODS.—Fixation may be obtained by submerging films in a mixture of equal parts of absolute alcohol and ether for 20 or 30 minutes, or in absolute alcohol for five minutes. The Futcher-Lazaer method subjects the films to .25 per cent. formalin in 95 per cent. alcohol for one minute; they are then rinsed in water and dried with filter-paper.

Blood Staining.—Many methods for staining blood are available. To Ehrlich belongs the credit of devising a mixture by which all known varieties of blood-cells except those which contain basophilic granules are completely colored. At the present time certain panoptic fluids containing eosin-methylene-blue compounds are employed extensively in routine work, having largely supplanted Ehrlich's triple stain.

Ehrlich's triple stain is prepared by mixing saturated aqueous solutions of acid fuchsin of orange G. and of methyl green $\overline{00}$ with glycerin, ethyl alcohol, and water. The following formula is recommended by Emerson:

Acid fuchsin solution	6–7 c.c.
Orange G. solution	13–14 c.c.
Distilled water	15 c.c.
Absolute alcohol	15 c.c.

Add drop by drop, shaking after each addition:

Methyl green $\overline{00}$ solution	12.5 c.c.

Then add:

Absolute alcohol	10 c.c.
Glycerin	10 c.c.

Allow the mixture to stand for twenty-four hours, and if, as is sometimes the case, a precipitate is present, care should be exercised not to disturb it when removing some of the stain. The combination of dyes in this mixture contains a neutral staining ingredient in addition to its acid and basic principles.

Staining Technic.—The stain is applied to the blood-film previously fixed by heat, for five minutes, after which the excess of stain is drained off and the cover washed with water, dried, and mounted in xylol balsam or cedar oil. Normal erythrocytes are colored orange, eosinophilic granules dull red, neutrophilic granules violet or lilac, nuclear structures various shades of green, blue, or black, malarial parasites and bacteria green or blue, while basophilic granules are unstained.

Jenner's stain is prepared as follows: Mix equal parts of a 1 per cent. aqueous methylene-blue solution with a 1.25 per cent. aqueous eosin (water soluble) solution. After shaking thoroughly, the solution is allowed to stand for twenty-four hours and then filtered. The precipitate is dried. One part of precipitate is dissolved in two hundred parts of methyl alcohol. Films are treated with this solution without previous fixation for from three to five minutes, washed with water, dried, and mounted in xylol balsam or cedar oil. The following tinctorial reaction is secured: Normal erythrocytes stain terra-cotta; nuclei, various shades of blue or green; basophilic granules, dark blue; neutrophilic granules, pink; eosinophilic granules, bright red; the cytoplasm of lymphocytes and malarial parasites, a deep blue. A deposit of dark granules upon the film which is often observed is an objectionable feature and interferes with the usefulness of this method.

Leishman's stain, an improvement on Jenner's, is based on the Romanowsky method. It is prepared as follows: (1) A one per cent. aqueous solution of methylene blue (Gruber's medicinal), containing 5 per cent. of sodium carbonate, is heated at 65° C. for twelve hours and then allowed to stand for ten days. (2) An equal volume of a 1 per cent. solution of eosin in distilled water is added to the methylene-blue solution in an open vessel and the mixture stirred from time to time. After twelve hours the resultant sediment is collected on filter-paper and washed with water until the washings are almost colorless. One and a half parts of dried powdered precipitate are added to one hundred parts of pure methyl alcohol. Previous fixation is not required with Leishman's reagent, since it possesses the double property of fixing and of staining. Three or four drops of this stain are placed upon the blood-film and allowed to act for thirty seconds, when double the amount of water (six or eight drops) is poured upon the cover and mixed with the stain. After five minutes the spread is washed gently with water and a few drops allowed to remain upon the specimen for about a minute. The smear is now dried, first between filter-paper and then in the air, and mounted in balsam or cedar oil. The nuclei of leucocytes and of erythroblasts, and blood-platelets are stained various shades of purple, the protoplasm of lymphocytes and certain polychromatophilic erythrocytes various tints of blue, basophilic granules dark violet or royal purple, normal erythrocytes and eosinophile granules pink, and neutrophile granules a dull red. Malarial parasites and trypanosomes are distinctly stained by this method. Wright's stain, which is also a modification of the Romanowsky method, contains an eosin-methylene-blue combination held in solution by methyl alcohol. Wright's method is extensively employed in this country. Hasting's mixture, another eosin-methylene-blue stain, is much used by some workers. For general routine work Leishman's stain can be highly recommended.

DOUBLE STAINING. — The films, after suitable fixation obtained by immersion in absolute alcohol, alcohol and ether, or by heating as previously described, are treated first with an acid stain followed by a basic dye, rinsed in water, dried, and mounted. A staining fluid containing acid and basic coloring principles may be employed for this purpose. Most of the methods of double staining are not suitable for differentiating all forms of blood-cells, as certain histological elements remain unstained. Neutrophilic granules are as a rule not colored, and therefore neutrophilic myelocytes cannot be distinguished from large mononuclear leucocytes. If only the nuclear structures are to be studied, double staining is of service, but is nevertheless inferior to Leishman's stain and similar methods.

Plehn's stain has the following formula:

Saturated aqueous solution of methylene blue	60 c.c.
One-half per cent. eosin solution (in 75 per cent. alcohol)	20 c.c.
Distilled water	40 c.c.
Twenty per cent. solution of caustic potash	.5–1 c.c.

Specimens are fixed in absolute alcohol for from three to five minutes, stained with Plehn's solution, washed in water, dried, and mounted. This mixture stains malarial parasites blue and eosinophilic granules red.

Eosin and Methylene Blue.—A convenient plan consists in treating the fixed smear with a solution consisting of eosin .5 part in 70 per cent. alcohol 100 parts, for a minute or two; wash the cover in water, and then counterstain with a half-saturated solution of methylene blue or Delafield's hæmatoxylin solution for a half to one minute. The specimen is then rinsed in water, dried between bibulous paper or in the air, and mounted.

Chenzinsky recommends a stain composed of 40 cubic centimetres of saturated methylene blue, 20 cubic centimetres of a .5 per cent. eosin solution in 70 per cent. alcohol and 40 cubic centimetres of distilled water. Films fixed in absolute alcohol are subjected to Chenzinsky's solution for from three to six hours, the staining being done at 37° C. in an incubator. *Ehrlich* suggested a mixture consisting of hæmatoxylin 2 grammes, eosin 0.5 gramme, absolute alcohol 100 grammes, distilled water 100 grammes, glycerin 100 grammes, acetic acid 10 grammes, and an excess of alum. The stain is not ready for use until several weeks have elapsed, since this time is required for the ripening of the stain.

Basophilic granules may be demonstrated by a stain recommended by Ehrlich which has the following formula:

Saturated alcoholic solution of dahlia	50 c.c.
Acetic acid	10–20 c.c.
Distilled water	100 c.c.

Differential Counting.—This method consists of determining the relative number of the different forms of blood-cells, generally expressed in percentage figures and sometimes as the number per cubic millimetre. The leucocyte differential estimation is important in the diagnosis of a number of conditions. An approximate differential count can be made by an examination of fresh, unstained blood by the experienced worker, but for accurate determinations stained films are essential. A mechanical stage is necessary for this method of counting.

Technic.—The specimen is brought into focus, and the slide is shifted with the mechanical stage so as to bring successive fields into view, being careful not to pass over any portion more than once. The different forms of leucocytes are noted and their number recorded until at least five hundred cells have been studied. From these figures the relative percentages are calculated. When nucleated erythrocytes are encountered, their number should also be noted, and the total number of these cells per cubic millimetre can be determined by the following formula:

$$\frac{\text{Number of leucocytes per cu. mm.} \times \text{number of nucleated red cells counted in the stained film}}{\text{Number of leucocytes counted in the stained film}} = \text{Number of nucleated erythrocytes per cubic millimetre.}$$

It is sometimes important to estimate separately the different varieties of abnormal red cells, especially the varieties of nucleated cells.

Enumeration of the Erythrocytes, Leucocytes, and Blood-platelets.—For clinical purposes, the red cells are counted in a small amount of blood of known quantity; from which an estimate of the number per cubic millimetre is made, this figure being the standard upon which the variations in health and disease are based. A number of methods are available for this purpose. The one recommended by Thoma is generally selected, as it gives fairly accurate results.

The Thoma-Zeiss Hæmocytometer.—This apparatus consists of two graduated pipettes (the red and white counters) for measuring, diluting, and mixing the blood, and a glass chamber in which the corpuscles are counted.

The erythrocytometer consists of a graduated capillary tube, upon which the figures .5 and 1 appear. The tube expands into a bulb, above which the figure 101 is inscribed. A rubber tube with a mouth-piece attached is fastened to the short end of the pipette. Filling the pipette with blood to the point marked .5 and then drawing a diluting solution into it until the fluid reaches to the point marked 101, insures a blood dilution of 1:200, while a dilution of 1:100 is obtained when the pipette is filled to the point marked 1, and then with a diluent to the mark 101. The white pipette, or leucocytometer, is similar in construction to the red pipette, but differs in that the capillary bore is larger and the bulb smaller so that dilutions of 1:20 and 1:10 may be secured.

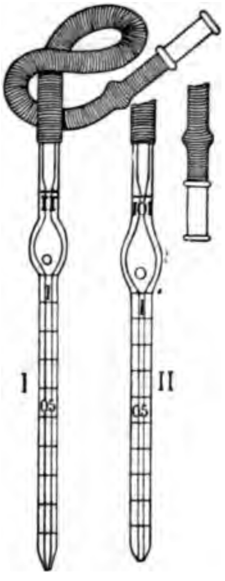

Fig. 93.—I, leucocytometer; II, erythrocytometer of Thoma-Zeiss hæmocytometer.

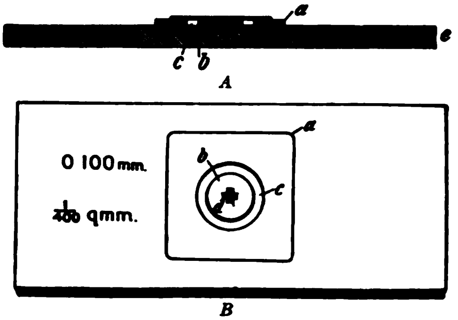

Fig. 94.—Counting chamber of the Thoma-Zeiss hæmocytometer. *A*, profile view; *B*, face view; *a*, wall of cell; *b*, central disk; *c*, groove about disk; *d*, ruled surface.

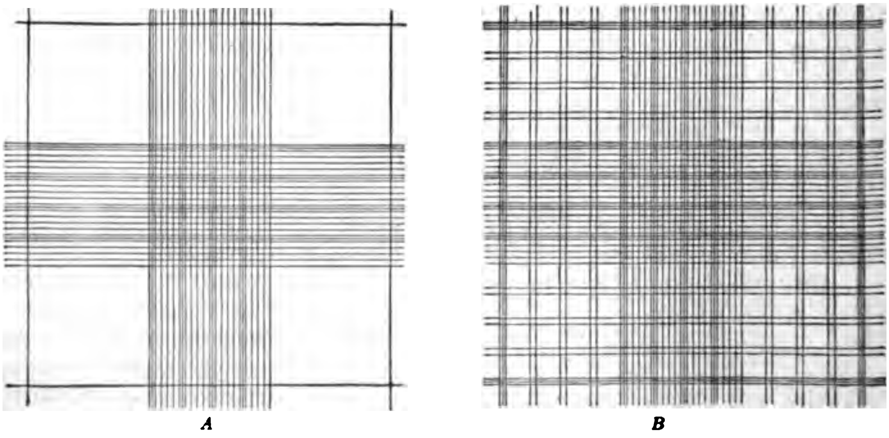

Fig. 95.—*A*, Zappert ruling; *B*, Türk's ruling.—Emerson.

The counting chamber consists of a heavy glass slide upon which is cemented a glass plate having a circular opening; a disk is cemented to the slide so that it occupies a central position in the circular opening of the plate. The disk is slightly thinner (by one-tenth of a mm.) than the plate which surrounds it. When the cover-glass, a part of this instrument, is placed upon the plate, the distance between the disk and the cover is one-tenth mm. The surface of the disk is ruled by vertical and horizontal lines one-twentieth of a mm. apart. These lines form four hundred squares, the dimensions of each being one-twentieth by one-twentieth mm. Groups of 16 squares are indicated by a double ruling. The space overlying each square between the surface of the disk and the cover-glass measures $\frac{1}{4000}$ cu. mm. ($\frac{1}{20}$ mm. $\times$ $\frac{1}{20}$ mm. $\times$ $\frac{1}{10} = \frac{1}{4000}$ cubic millimetre). Zappert's modified ruling of the Thoma-Zeiss counting chamber divides the surface into eight large squares, immediately surrounding the 400 small squares; each large square is equal to the surface ruling of the 400 central squares. The total ruling represents an area of 3600 small squares.

TECHNIC OF COUNTING THE ERYTHROCYTES. — Special fluids are employed for diluting the blood. *Toisson's solution* stains nuclei a pale blue, therefore rendering differentiation between non-nucleated erythrocytes and white corpuscles easy. Its composition is as follows:

Methyl violet, 5B	0.025	part
Sodium chloride	1.0	part
Sodium sulphate	8.0	parts
Neutral glycerin	30.0	parts
Distilled water	160.0	parts

Hayem's solution:

Mercuric chloride	0.25	part
Sodium chloride	0.5	part
Sodium sulphate	2.5	parts
Distilled water	100.0	parts

Other diluting fluids recommended for clinical work are a 2.5 per cent. aqueous solution of potassium bichromate, a .5 per cent. aqueous solution of sodium sulphate, and a .7 per cent. aqueous solution of sodium chloride. These solutions should be filtered before using.

The blood obtained in the usual manner is drawn into the erythrocytometer to the point .5, unless decided oligocythæmia is suspected, when it is desirable to fill to the mark 1, after which the tip of the pipette is wiped. Toisson's or some other diluting solution is drawn into the pipette until the fluid reaches to the point 101. The pipette should be rotated gently, as the diluting fluid enters the bulb, in order to secure a mixture. After filling the pipette, the thumb and finger are immediately placed over its ends and the instrument shaken for about a half minute, in order to obtain a thorough mixture. The unmixed fluid in the capillary portion is then blown out. The counting chamber is now placed upon a perfectly level surface and a droplet of the mixture is deposited in the central portion of the ruled disk. The pipette should be shaken just before adjusting the diluted blood, and the fluid in the capillary portion should always be expelled after mixing in this manner, since corpuscles in the capillary tube may gravitate on standing, thus creating an uneven mixture. The cover-glass is then quickly adjusted in its position. If the fluid flows into the depression surrounding the disk, the operation must be repeated. After the corpuscles have settled, the counting chamber is placed upon the stage of the microscope and a field of 16 squares is brought into focus. In general routine work, the calculation of determining the number of erythrocytes per cubic millimetre is usually based on the number of cells found within 64 squares, provided a uniform distribution of the cells exists. The following plan may be adopted in counting the corpuscles: The cells within the upper left-hand corner square of a group of 16 squares are first counted, then the cells in each of the remaining three squares in that line, going from left to right, after which the corpuscles in the next row of squares are enumerated, proceeding from right to left, next those in the third row and finally in the last line of squares, as shown in the diagram (Fig. 96). The counting chamber is now moved, so as to bring into focus another area of 16 squares, and the number of cells in this group is estimated. This process is repeated until the desired number of squares (not less than 64) has been covered. In order to avoid confusion in counting, the corpuscles which touch the right and lower lines are included in the count of the square in question. The formula for calculating the number per cubic millimetre is as follows:

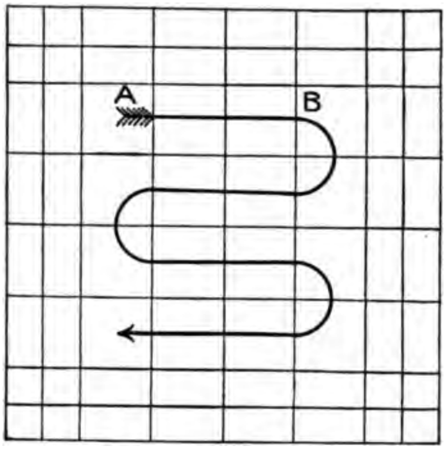

Fig. 96.—Scheme for counting cells overlying ruled surface.

$$\frac{\text{Number of cells counted} \times 4000 \times \text{number of dilutions}}{\text{Number of squares}} = \text{Number of cells per cubic mm.}$$

The greater the number of cells counted, especially with low dilutions, assuming that the mixture is thorough, the more accurate will be the results.

Technic of Counting the Leucocytes.—In determining the number of leucocytes, the red pipette may be used, but more accurate results are obtained with the white pipette, as lower dilutions are secured. A ½ or ⅓ per cent. aqueous solution of acetic acid is employed when using the white counter in order to dissolve the red cells. Except in the case of leukæmic blood, a dilution of 1:20 or 1:10 is most convenient for the majority of leucocytic counts. When the number of white cells is estimated with the red counter, with a 1:100 or 1:200 dilution, Toisson's solution is very useful, since with it the leucocytes are tinted blue and therefore readily distinguished from erythrocytes, which have a yellowish or greenish color. With Zappert's modified ruling the cells overlying a larger area can be counted. The formula for estimating the leucocytes per cubic millimetre is the same as that used for determining the number of erythrocytes. In routine clinical work the corpuscles overlying the entire ruled area of at least 400 squares should be counted when employing dilutions of one in ten or twenty.

Cleansing the Instrument.—After removing the fluid from the pipette, it is rinsed with water, then with alcohol, and finally with ether, and dried thoroughly. An atomizer bulb is useful for expelling the fluid from the tube and for drying. A simple method of removing the fluid from the pipette consists in pressing the end of the rubber tube between the fingers so as to occlude its lumen, and then by twisting the tube the fluid is expelled from the pipette. The counting chamber should be cleaned with water and dried with a soft handkerchief or tissue paper. Alcohol, ether, and xylol should not be used for cleaning the counting chamber, since these substances may dissolve the cement which holds the parts together.

Gowers's Hæmocytometer.—The principle of determining the number of corpuscles with this instrument is similar to that of the Thoma-Zeiss method. A mixing jar is used instead of a mixing pipette. The instrument consists of a capillary tube for measuring 5 cubic millimetres of blood, a pipette having a capacity of 995 cubic millimetres for measuring the diluting fluid, a mixing jar, a glass stirring rod, and a ruled counting chamber. The counting chamber is so constructed that the space overlying each square represents $\frac{1}{5000}$ of a cubic millimetre.

Oliver's Hæmocytometer.—With this method the number of corpuscles is approximately estimated by an optical effect and not by actually counting the cells in a known area. As the number of leucocytes cannot be determined with this method, it is not suitable for the majority of blood examinations and therefore has not been adopted for routine clinical work.

.Enumeration of Blood-platelets.—The blood-platelets are rarely seen in fresh unstained specimens, as they disappear almost immediately after the blood is exposed to the air. They are colorless, spherical, oval, or irregular, varying considerably in size, usually from one to three microns. In fresh blood, platelets are demonstrated by placing a cover-glass upon a slide and bringing their edges in contact with the blood as it flows from the puncture. Their number may be approximately estimated by Determan's method as follows: Place a drop of a 9 per cent. aqueous solution of sodium chloride upon the skin and make the puncture through the drop of fluid. As the blood flows from the wound, it is mixed with the reagent by stirring with a cover-glass or slide, and then a part of this mixture is placed upon the Thoma-Zeiss counting chamber and the cover-glass adjusted. The ratio of blood-platelets to erythrocytes is next determined in a given area. The number of red corpuscles per cubic millimetre is found by the Thoma-Zeiss method, and from this figure the actual number of blood-platelets per cubic millimetre can be calculated by the ratio the red cells bear to platelets.

Hæmoglobin Estimation.—The principle involved in the estimation of hæmoglobin with most of the instruments used in clinical work is based upon a comparison of the color of undiluted or diluted blood with a standard color scale.

Dare's Method.—The principle of this method is based on matching the tint of a film of undiluted blood of definite thickness with a graduated color scale. The essential parts of this hæmoglobinometer are a wedge-shaped semicircle of glass stained with Cassius's "golden purple" so that the various depths of the color displayed by the scale represent hæmoglobin values ranging from 10 per cent. to 120 per cent. (this wedge is contained within a hard-rubber case so that it can be revolved by operating a thumbscrew); a telescoping camera tube supplied with a magnifying lens through which the color of the blood and that of a part of the wedge is viewed; a pipette composed of two plates of glass, one being transparent and the other opaque (white glass); a part of the surface of the latter is slightly bevelled, so that a thin compartment is formed between the plates when their surfaces are opposed; and a candle holder.

Technic.— The pipette is brought in contact with a large drop of blood. It fills by capillarity. The pipette is then placed in its compartment on the side of the case. The light of a candle is used in making the color comparison, the instrument being held in a position so as to avoid direct sunlight. The rapidity with which an accurate hæmoglobin estimation can be made is the greatest advantage of this method. The matching of the colors should be done immediately after filling the pipette, since coagulation may begin within three or four minutes. The tint of the colored wedge of Dare's hæmoglobinometer does not in every instance correspond exactly with the color curve of certain anæmic bloods.

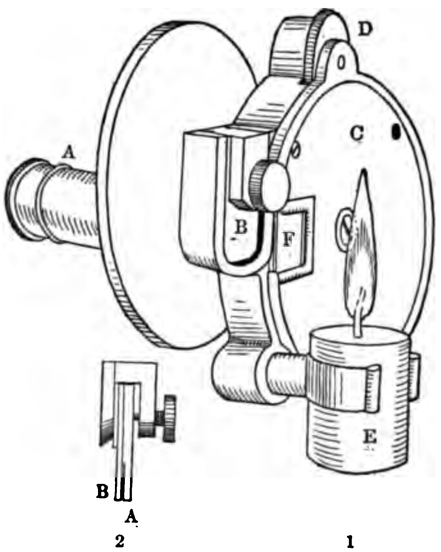

FIG. 97.—1. Dare's hæmoglobinometer. *A*, telescope; *B*, pipette in place; *C*, case enclosing color-prism; *D*, milled head moving prism; *E*, candle; *F*, window admitting light to color-prism. 2. Pipette. *A*, the white glass; *B*, clear glass disk.—Emerson.

TALLQVIST'S HÆMOGLOBINOMETER.—With this method the color of a drop of blood soaked into filter-paper is compared with a color scale lithographed upon paper. The apparatus consists of a book containing sheets of white filter-paper and a lithographed color scale of ten tints representing hæmoglobin values between 10 and 100 per cent.

Technic.—A piece of the white filter-paper is applied to the drop of blood, and, as soon as the moist gloss has disappeared from the surface of the blood-soaked paper, its color is compared with the scale. Accurate results are not claimed for this simple method. An error of at least ten per cent. is unavoidable.

VON FLEISCHL HÆMOMETER. — This instrument is composed of the following parts: A metallic stage having a circular opening in its centre, supported by a stand. To the frame of this stand is attached a plaster-of-Paris reflector. A glass wedge, tinted with Cassius's "golden purple," fixed within a metal frame. The depths of the color of the wedge correspond to a scale of hæmoglobin percentages stamped upon the frame, which range from 1 to 120. A cylindrical metallic mixing cell, divided into equal parts by a vertical partition, and provided with a glass bottom. A capillary measuring pipette attached to a metal handle. As the capacity of the

s varies in different instruments, a figure is stamped upon the handle
ipette and a similar marking on the stage of the instrument for which
ited. A finely pointed glass dropper, for filling the metallic cell.

chnic.—When one end of the pipette is brought in contact with
od, secured in the usual manner, it fills automatically by capil-
Blood adhering to the external surface of the pipette must be
away before emptying its contents. After partially filling one
compartments of the cell with water, the blood is washed out
pipette with water. The blood and the water are then thoroughly
by stirring with the handle of the pipette. The fluid adhering
handle must then be washed off with water, which is allowed to
into the mixing compartment. The other division of the cell is
ith water. Avoid moistening the top of the vertical septum, as
ay cause the fluids of the compartments to commingle. The filled
now adjusted in its proper position on the stage, and a comparison
color of the diluted blood with that of the scale is made in a dark-
om, or with a light-proof box. A candle flame placed about 15
entimetres in front of the plaster-of-Paris reflector is used for illu-
n. The operator, standing to one side of the instrument, matches
ors by turning the thumb-screw. The glass wedge should be moved
. Never view the colors for more than a few seconds, since the
easily fatigued by prolonged inspection. After two readings have
ade, the mean of these is taken as the result. An attempt should
be made to compare only the median portion of the color fields,
nay be readily accomplished
ing under the glass bottom of
a diaphragm of thin metal
r, having a narrow slit about
metres in width, the long
which is at right angles
partition of the mixing cell.
hen the hæmoglobin percent-
ow (below 30), two or three
s full of blood should be used,
e result divided by the
r of pipettes employed.
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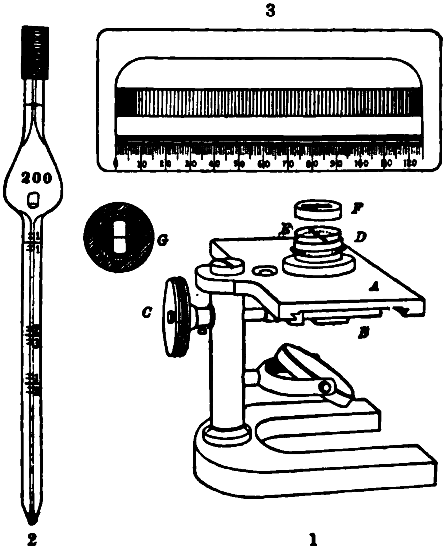


FIG. 98.—1. Meischer's modification of Fleischl's hæmoglobinometer. *A*, stage; *B*, color-prism rack; *C*, milled head; *D*, cell; *E*, cover-glass; *F*, cap; *G*, cell seen from above. 2. Mixing pipette. 3. Color-prism.—Emerson.

**E MEISCHER'S HÆMOGLO-
TER.**—This modification of
n Fleischl instrument pos-
certain advantages over the
hereby the degree of error is
rably lessened. The prin-
f Meischer's method is the
s that of von Fleischl. A
pipette is employed with which accurate dilutions of 1:200, 1:300, or
an be secured. For normal blood or nearly so, dilutions of 1:400 are

most convenient, but with low hæmoglobin values dilutions of 1:200 or 1:300 are more serviceable. Two metallic chambers are employed, each of which is divided by a vertical partition and supplied with a glass bottom. One compartment receives the diluted blood, the other water. One chamber is shallower than the other. The partition dividing the cells is slightly raised so that the glass cover, provided with a groove, may be slid over the top of the cylinder, thereby preventing the fluids from commingling. A lid having a narrow oblong opening is used to cover the chamber so that the width of the field exposed when making the color comparison does not correspond to more than three degrees of the percentage scale. The tinted wedge of this instrument is more accurate than that of the von Fleischl. After securing the desired dilution and mixture in the pipette, one of the compartments in each of the cells is filled with the blood solution, the other compartment with water; the glass cover is then slid into position and the metal top adjusted. The reading of each cell is then made with artificial illumination, using the same technic as with the original von Fleischl method. The result of the reading of the shallower cell is multiplied by $\frac{5}{4}$; this figure should correspond closely with the reading of the other chamber, one result controlling the other. The mean of the two readings represents the hæmoglobin percentage.

Oliver's Hæmoglobinometer.—With this method the color of a definite quantity of diluted blood is compared with a standard color scale, consisting of a series of tinted glass plates. The instrument is composed of the following parts: A standard blood scale composed of 12 colored disks, mounted upon a perfectly white surface in two metal frames. Their tints correspond with the color of various dilutions of blood. These primary disks correspond to hæmoglobin percentages ranging from 10 to 120; two pieces of tinted glass, called riders, are supplied with the instrument for ordinary clinical purposes. When a rider is superimposed upon a primary color, its shade deepens and therefore determines intermediate percentages between those indicated by the disks. An error of 2½ per cent. is unavoidable. A capillary tube having a capacity of 5 cubic millimetres for measuring blood. A standard mixing cell provided with a glass lid. A camera tube through which the colors are viewed, and a pipette for washing the blood out of the measuring pipette.

Technic. — The blood measured in the pipette is washed into the mixing cell with water and mixed with the handle of the pipette. The fluid which adheres to the handle is rinsed with the water and the cell filled. The glass lid of the mixing cell is then adjusted in a manner so that a small air bubble is present under the cover. The color of the diluted blood is matched with one of the disks of the color scale in a darkened room, illuminated with the light of a small wax candle placed about 10 centimetres in front of the mixing cell and the color disk. One or both riders may be required to intensify the tint of the primary disk.

Gowers's Hæmoglobinometer.—With this method a definite quantity of blood is diluted, until the color of the mixture corresponds with a standard color contained in a tube. This instrument consists of: A standard color tube which contains glycerin jelly colored with picrocarmine, so that its tint corresponds with that of a solution containing one

›f normal blood in a hundred parts of water; a mixing test-tube ȥ a graduated scale ranging from 5 to 120; a pipette for measuring ›ic millimetres of blood.

'*echnic.*—The measuring pipette, to which is attached a small rubber is filled by suction up to the point marked 20. A few drops of are placed into the mixing tube, then the blood in the pipette is into the tube. Water is added in small amounts, shaking after ıddition in order to secure a mixture, until the color of the solution ponds with that of the standard tube. The height of the fluid ›d indicates the hæmoglobin percentage. The color comparison is with daylight by holding the tube against a white background.

AHLI'S HÆMOMETER.—The principle of this method is based on ıring the tint of a standard fluid ›sed of a definite amount of normal and of a decinormal solution of chloric acid with the tint of a solu- f blood to be tested treated with a ›rmal hydrochloric acid solution and in sufficient quantity to exactly ı the colors. The height of the n of fluid in the mixing tube indi- the hæmoglobin percentage. Sahli ı that with this method the color standard solutions and that of the properly diluted corresponds quite ıtely, thereby insuring uniform s. The apparatus is similar in con- ion to Gowers's hæmometer. It ts of a sealed tube containing the ard color solution of decinormal chloric acid holding one per cent. of ; a graduated test-tube for mixing lood with a decinormal hydrochloric acid solution and water; a ›e for measuring 20 cubic millimetres of blood; a perforated stand a white glass back for holding the tubes; a bottle for carrying the solution; and a finely pointed pipette. The standard color fluid brownish-yellow color, due to hæmatin hydrochlorate held in sus- ›n. Since precipitation of this substance will occur on standing, ›aled tube is provided with a glass ball which serves to mix the parti- hen the tube is agitated.

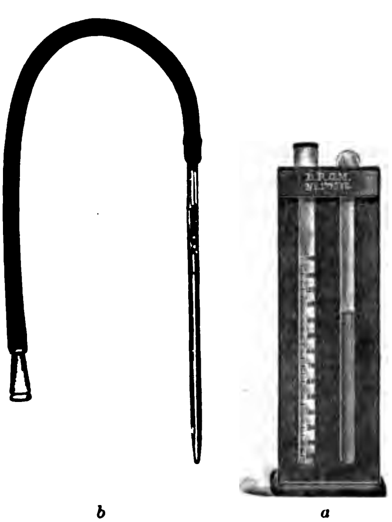


FIG. 99.—*a*, Sahli's hæmometer; *b*, pipette.

'*echnic.*—The graduated tube is filled with decinormal hydrochloric ›o the mark 10. Twenty c.mm. of blood measured in the pipette ›en blown into the acid solution and mixed. The measuring pipette ›n filled with water and discharged into the mixing tube. The ated tube is now placed in its compartment in the stand alongside › standard tube and water is added in small amounts to the blood ›n, mixing after each addition, until the color matches the standard The height of the column of fluid in the tube, as indicated by the

6

graduated scale, represents the hæmoglobin percentage. The test is conducted with natural or artificial light. More accurate readings are possible when the test is made with artificial light in a darkened room.

Color Index.—The terms color index, blood decimal, or blood quotient are used to express the average hæmoglobin richness of the erythrocytes. This factor is determined by dividing the hæmoglobin percentage by the percentage of colored corpuscles per cubic millimetre. The normal color index is expressed by the figure 1, *i.e.*, 100 per cent. of hæmoglobin divided by 100 per cent. of red cells. In anæmic states the same result is obtained when the hæmoglobin and red cells are proportionately reduced. In chlorosis the color index is generally decidedly diminished, while in most symptomatic anæmias it is slightly and in some cases markedly lowered. In pernicious anæmia, except during periods of improvement, it is generally increased.

Estimation of the Relative Volume of Plasma and of Corpuscles.—This determination is made by applying centrifugal force to blood contained in a tube, which separates the corpuscles from the plasma. By estimating the volume of corpuscles, an approximate idea may be formed of the number of cells per cubic millimetre.

DALAND'S HÆMATOCRIT.—This instrument consists of a set of gears operating a metal frame into which are fastened two capillary tubes. A hand lever is connected with the gears. The tubes for measuring the blood, graduated into 100 equal divisions, are 50 millimetres in length, with a lumen of ½ millimetre diameter.

Technic.—A piece of rubber tubing with a mouth-piece is attached to one end of the graduated tube. Blood is sucked into the pipette until completely filled. After removing the rubber tubing, the pipettes containing blood are fastened into the metal frame and immediately the handle of the instrument is turned for 3 minutes, at the rate of about 77 revolutions per minute, which produces the speed desired. The centrifugal force separates the blood into three layers; the one most distant, of dark red color, is composed of erythrocytes, the middle one, of milky color, is formed of leucocytes, while the inner clear layer consists of plasma. With normal blood the column of erythrocytes reaches to the graduation marked 50 or 51; each division of the scale approximately represents 100,000 corpuscles per cubic millimetre. Accurate estimations of the number of cells per cubic millimetre is impossible, since the size of the erythrocytes varies in pathological conditions and because a uniform speed is almost impossible to obtain. Variations in the centrifugal force will produce differences in the degree of compactness of the cells. The number of leucocytes can only be roughly estimated when there is a marked increase, as in leukæmia, but under normal conditions or pathological states with slight or moderate variations the leucocytic layer is too indistinct to warrant an opinion as to their number. The pipettes of this instrument should be cleaned immediately after using by passing a fine wire through the lumen, then washing with water, followed by alcohol and ether.

Volume Index.—Volume index, the term applied to represent the average volume of the erythrocyte, is determined by dividing the pertage volume, as estimated with the hæmatocrit, by the percentage of erythrocytes per cubic millimetre, obtained with the hæmocytometer.

Estimation of Specific Gravity.—An accurate estimation of the cific gravity of the blood can be obtained by Schmaltz's method, which sists of weighing a dry pipette upon a sensitive balance. The pipette then filled with water and the weight determined. After cleaning and ying, the pipette is filled with blood and again weighed. From these ures the specific gravity is calculated.

Hammerschlag's Method.—Hammerschlag's modification of Roy's thod is based upon the principle of suspending a drop of blood in a uid having the same specific gravity. The specific gravity of the sussion fluid is then determined with a hydrometer, which corresponds that of the blood.

Technic.—Pour benzol and chloroform into an hydrometer jar, in h proportions as to secure a mixture having a specific gravity of out 1.060. Partially fill a pipette, or medicine dropper, with blood d insert it into the benzol-chloroform solution: expel a droplet into fluid. If the blood is lighter than the mixture, it will rise to the . Benzol should then be added and the fluid carefully stirred with a ss rod until the blood is suspended in the mixture. The specific grav- of the benzol-chloroform solution is next determined, which corrends to that of the blood. If the specific gravity of the blood is greater n that of the benzol-chloroform mixture, causing the blood to sink, addition of chloroform is necessary to cause suspension. This method determining the specific gravity is seldom employed in clinical work, it is tedious and as errors of technic are readily made. The specific vity ranges of the blood correspond quite closely to definite hæmoglobin rcentages; notable exceptions to this rule are found in progressive perious anæmia, where the hæmoglobin percentage is slightly higher than specific gravity indicates, while in leukæmia the reverse is observed. mmerschlag's scale of specific gravity ranges with equivalent hæmoglobin rcentages is as follows:

Spec. Gravity.	Hæmoglobin.
1.033–1.035	25–30 per cent.
1.035–1.038	30–35 per cent.
1.038–1.040	35–40 per cent.
1.040–1.045	40–45 per cent.
1.045–1.048	45–55 per cent.
1.048–1.050	55–65 per cent.
1.050–1.053	65–70 per cent.
1.053–1.055	70–75 per cent.
1.055–1.057	75–85 per cent.
1.057–1.060	85–95 per cent.

Estimation of the Time of Coagulation.—As a number of condins influence the rapidity with which coagulation of the blood occurs er it is withdrawn from the blood-vessels, such as the amount of blood d the temperature, the results obtained by different methods of deterning the clotting time are not available for comparative studies. In s connection it should also be borne in mind that the factors which ntrol intra- and extravascular coagulation are in all likelihood dissimilar.

Method of Russell and Brodie.—The coagulation time is determined by microscopical study of the blood. The apparatus needed for this method is provided with a moist chamber having a glass bottom. A removable glass cone (the lower surface of which is 4 mm. in diameter) forms the upper portion of the chamber. A current of air is introduced into the chamber by means of a small tube one end of which projects into the cell, while to the other end is attached a rubber tube supplied with a bulb. Boggs's coagulometer, a modification of the instrument just described, is equipped with an improved glass cone and a metal tube.

Technic.—A drop of blood is placed upon the lower surface of the cone which is then immediately fitted into the chamber. The instrument is then put upon the stage of the microscope and with a low-power objective the blood is brought into focus. At successive intervals the blood is agitated by means of the current of air sent into the cell from the bulb. It will be noted that at first the stream of air causes the corpuscles to move freely. A little later clumps form in the peripheral zone of the blood and these can be advanced by the air current. Then as clotting progresses masses of blood-cells cease to move freely, the drop alters its shape, and the corpuscles exhibit a concentric motion. Lastly, a radial movement appears, clumps of cells being displaced by the air current towards the centre and these quickly return to their original position. Clotting is now considered complete. The normal coagulation time as determined by this method varies from three to eight minutes, the average time being about five minutes.

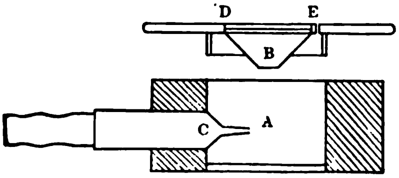


Fig. 100.—Coagulometer of Russell and Brodie as modified by Boggs. *A*, moist chamber; *B*, cone of glass, the lower surface of which holds the drop of blood; *C*, side tube; *D* and *E*, cover-glass; at *E*, a pinhole.—Emerson.

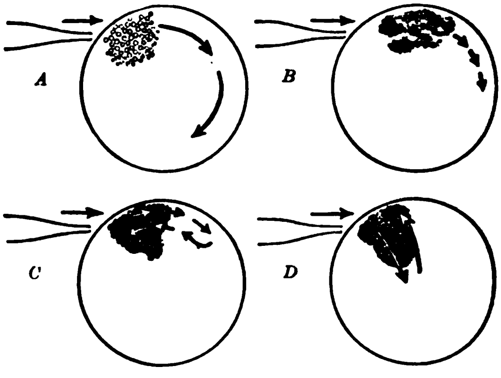


Fig. 101.—Diagram to illustrate the movement of the cells during coagulation.—Emerson.

Wright's Method.—The coagulometer devised by Wright consists of a cylindrical tin vessel provided with a perforated partition, the openings of which are so arranged as to support twelve graduated tubes and a thermometer. The tubes are graduated for 5 c.c. of blood, and are numbered from one to twelve.

Technic.—Water having a temperature of 18.5° C. is poured into the metal container. The blunt end of six or eight of the tubes is then covered with a rubber cap. The tubes are then placed, closed end downward, into the water. After having acquired the temperature of the water, they are removed separately at once or one-half minute intervals. the cap taken off, filled with 5 c.c. of blood and immediately replaced into the water without reapplying the caps. Attempts at short intervals are made to dislodge the blood from the tubes by blowing. When the blood cannot be removed from one of the tubes, coagulation may be considered complete. The clotting time is the difference of time between the filling the tube and the unsuccessful attempt to expel its contents. With this instrument the coagulation time of normal blood in most instances is from three to six minutes, although the period may be as long as fifteen minutes.

Bacteriological Examination.—There are two methods of demonstrating bacteria in the blood—one by an immediate miscroscopical examination of stained films, the other by blood culturing. The former plan is applicable only in a limited number of diseases and cannot be employed as a routine procedure because the blood obtained by puncturing the skin frequently becomes contaminated with bacteria which are normally present in the skin, especially the staphylococcus epidermidis albus. However, this method has been found convenient in the diagnosis of very severe cases of bubonic plague. Films can be prepared by placing several large drops of blood upon a slide so as to secure a thick spread. Before staining, much of the hemoglobin may be removed from the dried film by soaking the specimen in distilled water. Far more reliable results are obtained by removing 2–5 c.c. of blood from a vein and diluting it with 100 c.c. of bouillon. From this mixture the bacteriological investigations are then made. In the late stages of anthrax infection the blood may show a large number of bacilli so that the immediate microscopical method may give positive results. In this disease the cultural method gives more certain results than the study of films prepared from blood obtained by puncture of the skin.

Technic.—The skin of the flexor surface of the elbow is cleansed as for a surgical operation, by scrubbing thoroughly with soap and water, washing with sterilized water, alcohol, and ether, after which an antiseptic dressing is applied and allowed to remain for six or eight hours. The operator, having prepared his hands, should, after removing the antiseptic dressing, wash the skin with sterilized water. A syringe (of moderate size like the instrument used for exploratory puncture) or a special "blood aspirator" is required to remove the blood from the vein. A most useful instrument employed by many workers consists of a graduated glass tube having a capacity of about 10 cubic centimetres, one end of which is fitted to a No. 42 hypodermic needle, and into the other end a small plug of cotton is inserted. In order to sterilize the instrument, it is placed in a large glass tube, the ends of which are then plugged with cotton. After sterilization a piece of rubber tubing is fastened to the end of the aspirator containing the cotton. A bandage is wound around the arm of the patient so as to obstruct the venous circulation, and when the superficial veins at the elbow become distended, the needle of the syringe or blood aspirator is inserted into the most prominent vessel. The piston is withdrawn slowly until the desired quantity of blood is obtained. As a rule, the blood flows freely into the aspirator previously described, but, should this not be the case, a sufficient amount can be secured by making suction through the rubber tube.

The blood is placed in a suitable culture medium. Fluid media such as bouillon and litmus milk are generally selected for the primary inoculations when certain types of bacteria are suspected, while agar may be chosen when the medium is to be plated. One or two cubic centimetres of blood are added to 50 or 100 c.c. of fluid medium so that dilutions of one in fifty or one in one hundred are secured. For details of bacteriological technic, which do not fall within the scope of this work, special treatises on bacteriology should be consulted.

Agglutination Reaction.—The blood in certain stages of typhoid fever, and often after the attack, possesses the property of checking the motility of typhoid bacilli and causing these organisms to form into clumps. This agglutination phenomenon is so pronounced that high dilutions of blood, as one in fifty or one hundred, or even higher, give positive results. The blood in similar dilutions in other diseases and in health does not act in this manner with typhoid bacilli. With low dilutions, however, a positive agglutination reaction is often present with normal or abnormal blood. In a number of diseases—as pneumococcal and streptococcal infections, paratyphoid fever, Malta fever, tuberculosis, cholera, plague, relapsing fever, glanders, and others—specific agglutination reactions have been obtained. The agglutination test is chiefly employed in the diagnosis of typhoid and paratyphoid fevers and is generally spoken of as the Widal, Gruber-Widal, or Pfeiffer-Widal reaction. For this test two methods are available, (1) the microscopic and (2) the macroscopic. In typhoid fever the agglutination reaction is positive in about 97 per cent. of the cases during the course of the disease, manifesting itself in a majority of them about the end of the first or during the second week, in a few instances as early as the third or fourth day, while in others it is not obtained until the attack is far advanced, and it often persists long after convalescence. The intensity of the reaction varies in different cases. Positive reactions are obtained with dilutions as high as 1:200. In some instances the reactions occur almost instantly, while in others the stoppage of motility and clumping take place slowly.

1. Microscopical Serum Test. — This test may be performed with fluid blood, blood-serum, or dried blood.

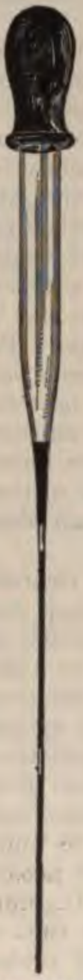


Fig. 102.—Capillary pipette.

Technic.—*Preparing Cultures.*—From a slant agar growth of typhoid bacilli, preferably not older than one month, subcultures are made in sterile bouillon and incubated at blood heat for 8 to 12 hours, when they are ready for use. The stock culture should be kept in a cool place. Some workers prefer a suspension of typhoid bacilli in salt solution made by placing a loopful of a twenty-four hour agar growth in saline solution. The tube containing the fluid is agitated until a uniform suspension of the germs is obtained.

Collecting and Diluting Serum. — A capillary pipette, suitable for measuring the blood, is made from a piece of glass tubing about 30 cm.

in length and 5 or 6 mm. in diameter. The middle portion of this tube is heated in the Bunsen flame, rotating continuously in its long axis until the glass is thoroughly softened over 3 to 6 centimetres of its length; remove from the flame and draw the two ends apart with a steady uniform pull so that the heated portion tapers into a long capillary tube. By melting the middle of the capillary tube in the flame, two pipettes with the capillary end sealed off are made. A Wright's blood capsule, shown in Fig. 103, will be found convenient for collecting the blood.

Preparing the Serum.—The patient's finger-tip is cleansed and rubbed briskly so as to produce hyperæmia. A puncture is then made of sufficient size so as to insure a good flow of blood. The sealed tips of a Wright's capsule are broken off, and the end of the short curved portion of the capsule is placed into the blood as it issues from the small wound, the body of the tube slanting downward so as to allow the blood to enter by gravity. The capsule is partly filled (½ or ⅔ full). The tip of the longer arm is sealed off by heating in a flame. When properly cooled, the blood is shaken down. The other end may then be closed to prevent evaporation, if the test is not made immediately. The capsule is now hooked upon the rim of a centrifuge tube and centrifugalized until clear serum separates. Slight turbidity of the serum does not interfere with the test. The capsule containing the centrifugalized blood is opened by filing a groove into the glass tube above the level of the serum and breaking off the end. The fine end of a capillary pipette (having previously been broken off the sealed tip) is inserted into the capsule and the serum drawn into the tube. The blood-serum may be diluted and mixed with the culture in watch crystals or in a porcelain plate having a number of cup-shaped depressions as shown in Fig. 104. One drop of serum is now placed into one of the depressions of the porcelain plate. Dilutions of the serum with sterilized normal salt solution are then made. The capillary tube, having been cleaned with salt solution or water, is partly filled with saline fluid. Into the depressions containing a drop of serum, 24 drops of salt solution are allowed to fall from the pipette and mixed, thus making a dilution of 1–25, since the drops from the pipette are practically of the same size. Into a second depression are placed 5 drops of salt solution and 5 drops of the diluted serum of 1–25, securing a 1–50 dilution. More accurate results are obtained by measuring and diluting the serum in a Thoma-Zeiss hæmocytometer pipette. Two hanging drop preparations are prepared—one from each dilution—by mixing upon a cover-glass a platinum loopful of bouillon culture of typhoid bacilli with a loopful of diluted serum. Since each dilution of serum is again diluted to ½, the proportions now stand 1–50 and 1–100. The cover-glasses are adjusted upon the slides and the edges of the slips surrounded by petrolatum to prevent evaporation. The

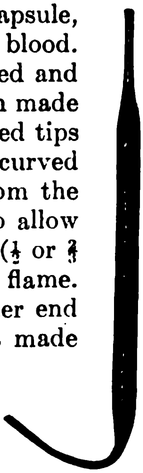


FIG. 103.—Tube for serum work.—Emerson.

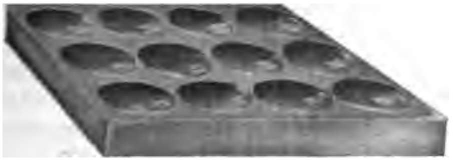


FIG. 104.—Porcelain disk for mixing serum, salt solution, and bacterial cultures.

preparations are allowed to stand at room temperature for exactly one hour. In order to secure correct results, it is essential that the motility of the bacilli should be active, and the density of the culture be uniform and not show clump-like gatherings.

Recording Results.—At the end of one hour, the hanging drop slides are examined microscopically. When motion of the bacilli is found absent and clumping good in both slides, the reaction is termed "positive," but when the free motion without clumping of bacteria is noted, the test is negative. Variations between these two extremes may be recorded according to the judgment of the examiner. Thus, if 1–50 shows no motion and good clumping, but 1–100 exhibits slight motion and only fair clumping, the reaction may be called "very suggestive;" or, again, if 1–50 shows slight motion and poor clumping, while 1–100 free motion and no clumping, the reaction may be called "slightly suggestive." It is best to indicate definitely the results of each dilution, as for example:

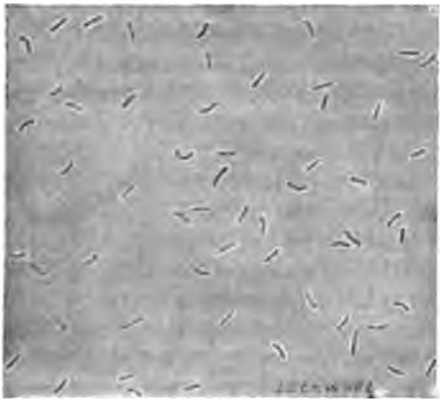


FIG. 105.—Widal test. Field of motile organisms.—Emerson.

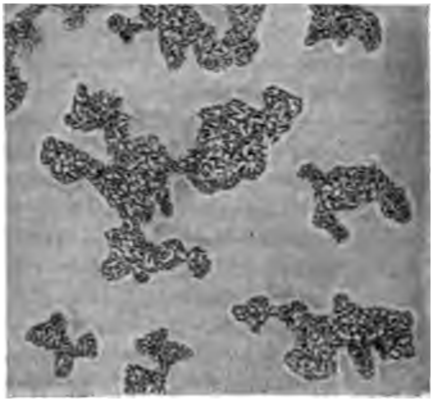


FIG. 106.—Widal test. Field of agglutinated organisms.—Emerson.

1–50 Good clumping.	No motion.
1–100 Fair clumping.	Slight motion.

This allows the diagnostician to form his own conclusions and does away with dogmatic assertions, such as "Widal positive" or "Widal negative," which are so often a matter of personal equation upon the part of the laboratory worker.

The liquid serum method, unfortunately, cannot always be employed by physicians in active practice. The microscopical test may be carried out with blood collected upon a piece of paper, or upon a slide and allowed to dry, after which the test may be made at any time. The blood secured in this manner is moistened and dissolved in sterilized water, and then diluted and mixed with the culture in the desired proportions. It is obvious that accurate dilutions are impossible, an objection to this method.

2. **Macroscopical Serum Test.** — By aspirating a vein, a sufficient amount of blood is collected in a sterile test-tube and allowed to clot, so as to separate the serum, or the blood may be centrifugated. The

serum is mixed with salt solution and bacterial culture in the desired proportions (1–50 or 1–100). In the case of a positive reaction a flaky precipitate will separate with a clear supernatant fluid, while a negative reaction shows uniform turbidity of the fluid. The macroscopical test may also be performed by mixing bouillon and serum in proper dilutions and inoculating the mixture with a loopful of a broth culture. The presence of a precipitate in the tube at the end of twenty-four hours' incubation signifies a positive result. The chief objection to the macroscopic method is the relatively large amount of blood required. The microscopic test is generally employed in clinical work. The macroscopical test may also be performed with dead cultures of the bacilli. The principle of the Ficker "Typhus Diagnosticum" is based on mixing a dead culture with diluted blood-serum. Bacilli in liquid media killed with carbolic acid or formalin are also employed for this test.

Opsonic Index of the Blood and Its Determination.— Leishman in 1902 devised a method for estimating the phagocytic activity of the leucocytes. Extensive researches upon this subject have recently been made by Wright and Douglas, and many other investigators. Opsonins are substances within the blood which prepare bacteria for ingestion by the white cells. The power of the leucocytes alone to ingest bacteria, the so-called "spontaneous" phagocytosis, has been shown to be very slight, and the rôle played by them in fighting diseases is merely as scavenger, collecting bacteria acted upon by the opsonins. Opsonins do not stimulate or otherwise affect the leucocytes. These substances are destroyed by a temperature of 65° C. for ten minutes.

TECHNIC.—The special technic used for the determination of the opsonic power of the blood may be briefly set forth as follows: There must be on hand for the test (1) an emulsion of the bacteria in salt solution, (2) washed white blood-cells, taken from any source, (3) serum from the patient's blood, and (4) serum of normal blood or from a mixture of healthy bloods taken as a standard control.

Preparing the Bacterial Emulsion.—The micro-organisms for the test are inoculated upon culture medium. For some forms of bacteria, as the Staphylococcus aureus, an agar medium is selected. After twenty-four hours of incubation at 37° C. a fair-sized colony, found on the culture medium, is removed and mixed with a sterile .85 per cent. salt solution. The resulting bacterial emulsion is drawn up and down in a small pipette by means of rubber teat. The emulsion is set aside for a few minutes so as to allow the bacterial clumps to settle. The supernatant liquid is then removed and diluted to the desired density. Centrifugalizing may be necessary to separate bacterial clumps. With bacteria not readily emulsified, such as tubercle bacilli, grinding between glass plates or in an agate mortar is required to disintegrate the masses. The emulsion in case of tubercle bacilli may be made from fresh cultures or from dry, dead germs, such as are obtained in the production of tuberculin. Tubercle bacilli are best emulsified with a 1.5 per cent. salt solution. The proper density is one which on mixture with a normal serum and with the leucocytes in equal proportions will show that after incubation an average of 5 or 6 germs have been phagocytozed by each leucocyte. The density of the standard bacterial mixture may be fixed by McFarland's nephelometer, or by counting the bacteria in a given amount of emulsion in a Thoma-Zeiss counting chamber.

Obtaining the Washed Leucocytes.—A test-tube is filled two-thirds full with an aqueous solution containing 1.5 per cent. sodium citrate and .85 per cent. sodium chloride. The finger is pricked and 8 to 12 drops of blood are allowed to fall into the tube. The solution is shaken and then the tube is placed in an electric centrifuge and centrifugalized for 5 minutes at a speed of 1500 to 2000 revolutions per minute. The citrate defibrinates the blood and prevents clotting, while the sodium chloride solution washes the cells free of serum. Upon removal from the centrifuge, the tube is found to contain a compact sediment of blood-cells with a clear or very slightly cloudy supernatant fluid which consists of serum and salt solution. Overlying the surface of the red sediment will be found a white coating termed the "creamy layer," which is formed principally of white cells. The clear supernatant fluid is now drawn off with a capillary pipette by means of a rubber bulb. The layer of leucocytes, which contains some red cells, is now carefully removed from the compact layer of erythrocytes with the capillary pipette and placed in a small glass tube having a sealed end. The leucocytes may be washed with saline solution several times in order to remove the sodium citrate. This is accomplished by placing the leucocytes in a centrifuge tube or small test-tube and partly filling it with .85 per cent. salt solution. The tube is then centrifugalized, after which the supernatant fluid is removed with a pipette. This operation may be repeated.

Obtaining the Serum.—A Wright's capsule is filled two-thirds full of blood, obtained from the patient, and centrifugalized until the serum is clear. Serum must also be obtained from normal blood. Having on hand the bacterial emulsion, washed corpuscles, and serum, the main part of the test may be carried out. By means of a capillary pipette, equal amounts of bacterial emulsion, white blood-cells, and serum are measured, and the fluid mixed on a slide or watch crystal by drawing the material up and forcing it down the capillary pipette. The fluids are measured in the following manner: The rubber teat attached to the pipette is compressed, and, by gently relaxing the pressure, white cells, bacterial emulsion, and serum, in the order named, are drawn into the capillary bore up to the mark indicated by the pencil mark, each column being separated, by a small air bubble. Two pipettes are necessary for one test, one for the patient's serum, the other for the control serum. A special pipette, supplied with a rubber teat, is often used to measure and mix the bacterial emulsion serum and washed leucocytes, and is constructed in such a manner as to allow the worker a means of controlling accurately the amount of fluids drawn up in the long arm of the pipette and of mixing the contents afterwards. The sealed tip of a capillary pipette having been broken off squarely, a pencil mark is made 2 or 3 cm. above its extremity. After the ingredients are thoroughly mixed, the fluid is drawn into the pipette and its end sealed in the flame. The tube is then placed in the incubator at 37° C. for 15 minutes. In a like manner, the control test is prepared with equal amounts of white corpuscles, bacterial emulsion, and normal serum, which are also incubated. After incubation the end of the pipette is broken off and the contents are run up and down so as to mix thoroughly. Smear preparations are made of the material from each pipette upon slides or cover-glasses. After fixation the smears are treated with any reliable stain, such as Leishman's, which brings out distinctly the leucocytes and bacteria. For tubercle bacilli, carbol fuchsin and Gabbett's or Pappenheim's stain may be employed. The specimens are now examined with an oil-immersion lens. The number of bacteria in 100 typical polymorphonuclear neutrophiles is determined in both specimens. The average number of bacteria per leucocyte is then calculated for each specimen, which constitutes the phagocytic index. The phagocytic index of the patient's serum divided by the phagocytic index of the normal or control serum gives the opsonic index. The test can only be carried out properly in a well-equipped laboratory by one who has mastered opsonic technic. The strength of the bacterial emulsion, the length of incubation, the age of the ingredients employed, and the personal equation are some of the factors which influence the results. Opsonic index is employed in the diagnosis and prognosis of certain infectious diseases and in gauging the dose and the frequency of administration of bacterial vaccines. After the injection of therapeutic doses of bacterial vaccines, the index is seen primarily to fall and soon afterwards to rise above the normal. The initial fall constitutes the "negative phase" and the rise the "positive phase." The vaccine should not be repeated until the negative phase has passed into the positive, and this can only be gauged by repeated observations of the index. The value of the opsonic index for therapeutic or diagnostic purposes has not been definitely settled.

Test for the Detection of Diabetes Mellitus.—Two clinical blood-tests have been devised for this purpose, both of which are based upon similar principles.

WILLIAMSON'S TEST.—Twenty cubic millimetres of blood measured with the pipette of a Gowers's hæmocytometer are mixed with 40 cubic millimetres of distilled water in a test-tube. One cubic centimetre of a solution of methylene blue, of a strength of 1:6000, and 40 cubic centimetres of a 6 per cent. aqueous solution of potassium hydrate are then added to the diluted blood. A control test with normal blood should always be made. The test-tubes are then placed in boiling water for about five minutes, after which the solution containing the diabetic blood will have a yellowish color, while the color of the control mixture remains unaltered. This reaction may be positive in cases of diabetes mellitus after sugar disappears from the urine.

BREMER'S TEST.—Cover-glass films of the suspected blood and of normal blood to be used as a control test are made, which are fixed by heat at a temperature of 135° C. The smears are treated with a freshly prepared aqueous solution of Congo-red. The stain is washed off with water, and the preparations dried. The diabetic blood-film is colored a greenish-yellow, while the control film is stained red. Other aniline dyes, as methylene blue, methyl green, may be employed for this test. This test is at times positive with the blood of individuals suffering from other diseases, as leukæmia, Hodgkin's disease, and Graves's disease.

General Results of Blood Examinations.

Volume. — The blood, which forms from 4 per cent. to 7 per cent. of the total body weight, is a highly specialized tissue, consisting of erythrocytes, leucocytes, blood-plaques, and hæmokonia, suspended in a liquid matrix, the plasma. In health the total volume of blood varies within narrow limits. The view formerly entertained that an increase in the total amount is constantly present in some individuals—*plethora*—is not sustained by recent researches. *Oligœmia,* or a decrease in the total quantity,—e.g., due to a copious hemorrhage,—persists only for a short time after the bleeding, as the volume is rapidly brought up to its normal standard by the absorption of fluid from other tissues, which dilutes the remaining blood, producing a condition termed *hydrœmia* or *serous plethora.* Rapid abstraction of watery elements from the blood by sweating, diarrhœa, or vomiting causes a transitory increase in its density, known as *anhydrœmia.*

Color.—The color of the arterial blood is bright red, due to the presence of a large amount of oxyhæmoglobin, while that of venous blood, which contains less oxyhæmoglobin and much carbon dioxide, is dark red or purple. In some pathological states, as in diabetes mellitus and in leukæmia, the blood often has a milky tint; a peculiar chocolate color is sometimes imparted to the blood by poisoning with potassium chlorate, nitrobenzol, and hydrocyanic acid. Imperfect aëration, encountered in some diseases of the respiratory organs and heart and in chronic polycythæmia with splenic enlargement (Osler's disease), causes dark red blood similar to the color of venous blood. In carbon monoxide poisoning it is bright scarlet.

Reaction.—The reaction of normal blood is alkaline. The degree of alkalescence varies considerably both in health and in disease. None of the methods of determining the intensity of this reaction has been generally adopted for routine clinical purposes, and, as the results of various methods are not uniform, comparative studies by different observers are in the main inaccurate. The adoption of some standard technic may establish definite results, but up to the present time the data bearing upon this subject are insufficient to warrant positive opinions. Statistics indicate that the alkalinity is lowered in many pathological conditions, notably in diabetic coma, in many of the infectious diseases, especially in Asiatic cholera, in organic hepatic disease, in uræmia, in cachectic states, in a considerable group of skin affections, in poisoning by mineral acids, and in a number of other conditions. In chlorosis and rheumatic fever it has been found increased.

Specific Gravity.—The specific gravity of normal blood is about 1.060. It fluctuates slightly in health, while in disease there are wide oscillations. The specific gravity range is decidedly influenced by the amount of hæmoglobin, and so close is the relation between the two that an approximate hæmoglobin estimation can be made by determining its specific gravity. Exceptions to this rule are found in the case of leukæmia, in which the range of specific gravity would indicate a higher hæmoglobin value than actually exists, while in pernicious anæmia the reverse is true.

Coagulation of the Blood. — Within a short time after blood has been withdrawn from the circulation of a healthy individual, it undergoes coagulation, a process which determines the formation of fibrin and the separation of a clear, straw-colored fluid, the blood-serum. In a number of diseases considerable importance is attached to the determination of the time required for clotting. Delayed coagulation is encountered in persons suffering from obstructive jaundice, purpura, scurvy, and hæmophilia. In pernicious anæmia, in some cases of leukæmia, and at times in Hodgkin's disease, the rate of coagulation is prolonged. In some of the infectious fevers, and in acute inflammation attended with abscess formation, clotting is retarded, while in chlorosis, pneumonia, and scarlet fever it is rapid.

The Plasma.—The plasma, a complex albuminous body, which holds in suspension the solid elements and in solution many organic and inorganic compounds, is the vehicle through which substances are transported to the tissues and waste products carried to the excretory organs. The plasma also holds certain bodies possessing antitoxic, bactericidal, agglutinative, and opsonic properties. Agglutinins are of importance in the diagnosis of many infectious diseases, as in enteric fever, paratyphoid infections, Malta fever, cholera, relapsing fever, and dysentery. Some observers have noted this reaction in tuberculosis, pneumococcus and streptococcus infections, plague, and leprosy.

Erythrocytes. — The red blood-corpuscles in a preparation of fresh blood, taken from the peripheral circulation of a healthy person, appear as pale yellowish-green, non-nucleated, flattened, biconcave cells of a circular outline. They are pliable, somewhat elastic, non-amœboid, transparent, and show a tendency to form into groups or rolls when withdrawn from the circulation, and consist of a fine stroma which holds an albuminous iron compound, the hæmoglobin. Structural alterations of these cells occur when blood is removed from the circulation. They occasionally exhibit amœboid activity and may undergo disintegration, fragmentation, vacuolation, and crenation. A crenated corpuscle is a shrunken cell from which knob-like processes project. Structural alterations similar to those caused by withdrawing the blood from the vessels occur within the circulation as a result of pathological factors.

Staining Reaction of the Erythrocyte. — The normal red blood-cell, when properly fixed, has a monochromatophilic reaction, showing a selective affinity for acid dyes, while the living cell does not absorb stains (achromatophilic). On account of its biconcavity, the central part of the cell stains less intensely than the peripheral zone. The long diameter of the majority of the healthy cells measures about 7.5 microns, while its variations are between 6 and 9 microns.

Hæmogenesis and Hæmolysis.—It appears to be definitely established that in the adult the red bone-marrow is the chief, if not the only seat of erythrocytic formation. The colored cells develop from nucleated elements, erythroblasts, situated along the walls of capillary spaces of the marrow. Some authorities contend that erythroblasts and certain forms of immature leucocytes are derived from a common ancestral cell. The spleen and lymphatic glands are regarded by some as sources of erythrocytic formation, a view which is not entertained by many writers. The fairly uniform number maintained in the circulating blood of the healthy individual depends upon the existence of a parallelism between the rate of formation and the rate of destruction. Pathological erythrocytic destruction, unless excessive or prolonged, excites augmentation in the activity of erythroblastic multiplication and is followed by an increase in the output of red cells from the marrow. Most authorities maintain that the liver and in a less degree the spleen and the gastro-intestinal capillary area are concerned in destroying weakened, degenerated, or necrotic cells, while some hold that the bone-marrow also has a hæmolytic function. The coloring material derived from the disintegrated cells is in part transformed, in the liver, into bile pigment and eliminated through the biliary channels, in part discharged by the kidneys, and probably, to a considerable extent, stored up in many of the tissues where it is available for future needs of the body.

Number of Red Blood-cells. — The normal number of erythrocytes, which is 5,000,000 per cubic millimetre for an adult male and 4,500,000 for an adult female, is subject to slight variations under certain physiological conditions and to pronounced alterations in many morbid states. A decrease in the number is termed *oligocythæmia,* while an increase is designated *polycythæmia.* High counts are at times an indication of a decrease in the volume of plasma causing a relative polycythæmia. After blood transfusion, and after active blood regeneration, a temporary rise may be noted. Polycythæmia is encountered in the new-born, where it exists for some days after birth—probably not exceeding ten—in individuals residing in high altitudes, and in robust and well-developed persons. Massage, electricity, and cold bathing may also induce an increase in the erythrocytes in the peripheral blood. A slight reduction in the erythrocytes is brought about by pregnancy, menstruation, and lactation; it is also met with in poorly nourished individuals, in those who are fatigued, and during the period of digestion. Oligocythæmia, due to physiological causes, may sometimes be accounted for by temporary dilution of the blood, while in other instances an absolute decrease in the number of cells offers the best explanation.

In pathological states a relative transitory increase arises when the output of fluid from the body is decidedly in excess of the intake, and is therefore conspicuous in diseases associated with marked polyuria, as diabetes, with copious sweating, as from night sweats of pulmonary tuberculosis, with frequent vomiting, with profuse diarrhœa, as in Asiatic cholera, and after the withdrawal of a large quantity of fluid from a serous cavity, which rapidly reaccumulates, thereby draining the blood of much fluid. The pathological factors responsible for oligocythæmia are numerous, and in the vast majority of diseases associated with lowered counts the reduction depends upon increased blood destruction, in some it may be due to defective blood formation, or to a combination of both of these factors, while in others a slight transitory decrease is brought about by blood dilution—whenever the amount of fluid taken into the body is above the output of liquid. Lowered erythrocytic standards are noted in the primary anæmias, notably pernicious anæmia, in which the figure often falls as low as one million, and in occasional instances below half a million. Secondary anæmias arise from a great variety of causes, as from infections due to bacteria and animal parasites, metallic poisoning, organic visceral disease, hemorrhage, and many others.

The Hæmoglobin. — Hæmoglobin, a complex albuminous compound containing iron which is a component of the red cells, normally exists in two chemical states, in combination with oxygen (oxyhæmoglobin) and as reduced or plain hæmoglobin. Normal blood contains about 14 per cent. of hæmoglobin. A reduction of hæmoglobin, termed *oligochromæmia*, is generally associated with a fall in the number of erythrocytes. This deficiency of corpuscles, as a rule, is not so marked as the hæmoglobin loss, although sometimes the number of colored elements remains normal; rarely there is a proportionate reduction of the coloring matter and of the number of cells, or the latter may even show a greater percentage reduction than the hæmoglobin. An insufficient amount of hæmoglobin in the corpuscles is brought about by an effort on the part of the bone-marrow to rapidly regenerate cells at the expense of perfect formation (*i.e.*, after or during rapid hæmolysis). A high color index occurs in pernicious anæmia. Investigations, both experimental and clinical, have demonstrated that the hæmoglobin rises in certain anæmic states by the administration of iron compounds.

Hæmoglobinæmia, the term which implies the presence of hæmoglobin in solution in the plasma, is due to a number of causes, and is at times followed by the excretion of hæmoglobin by the kidneys (hæmoglobinuria). The disease known as paroxysmal hæmoglobinuria is a striking example of the latter condition. Among the conditions capable of producing hæmoglobinæmia are poisoning by sulphuric acid, nitrobenzol, phenacetin, acetanilid, phenol, hydrochloric acid, potassium chlorate, mushrooms, and snake venoms. Hæmoglobinæmia is excited by some of the infectious diseases, as malarial fever, enteric fever, yellow fever, typhus fever, variola, septicæmia, diphtheria, and syphilis; also by malignant jaundice, scurvy, sunstroke, burns, and from exposure to intense cold.

Methæmoglobin, another combination of oxygen and hæmoglobin, is a component of some pathological bloods and is present in poisoning with such substances as potassium chlorate, aniline, amyl nitrite, potassium permanganate, antifebrin, nitro-benzol, hydrochinone, potassium ferrocyanide, and snake venom. Carbon monoxide hæmoglobine, which occurs in coal-gas poisoning, gives the blood a bright scarlet appearance.

Abnormal Erythrocytes. — Cells exhibiting abnormal variations in size are common in anæmic states, particularly small erythrocytes known as *microcytes*, which have a diameter of less than 6 microns, and when these forms predominate in the blood the condition is termed *microcytosis* or *microcythæmia*. This change is conspicuous in chlorosis and in some secondary anæmias of the chlorotic type. Minute erythrocytes, spherical in form, with a deeply colored protoplasm, are often described as Eichhorst's corpuscles. Cells measuring more than 9 microns are designated *macrocytes*, and when these abnormally large forms outnumber other colored elements *macrocytosis* or *macrocythæmia* exists. Typical cases of pernicious anæmia show an average increase in the size of the red cells.

Rapid or defective blood formation has been advanced as the factor responsible for microcytosis, although structural alteration in the cells after they have entered the circulation, such as fragmentation and loss of hæmoglobin, may account for some of these dwarfed elements. Macrocytosis appears to depend upon the development of large cells in the marrow, or perhaps it is due to swelling of the protoplasm of the erythrocytes while in the general circulation.

Poikilocytes are cells having an irregular or distorted outline and often appear as pear-shaped, elongated, oval, and "hour-glass" forms. These pathological cells show wide variations in size and in staining peculiarities. The degree of cell deformity and the extent of the variation in size are generally proportionate to the severity of the anæmia. Pernicious anæmia and grave secondary anæmias reveal poikilocytosis in its most typical form. Cells so deficient in hæmoglobin that a mere colorless shell remains are termed phantom or shadow corpuscles or achromacytes. Erythrocytes which react indifferently and irregularly to acid and basic dyes, staining diffusely with both,—termed *polychromatophilic cells,*— are observed in profound anæmias, particularly in progressive pernicious anæmia. They appear in specimens stained with eosin-methylene-blue mixtures, as purple, brownish, or bluish cells, their color, as a rule, being unevenly distributed, and in some instances only a part of the protoplasm exhibits this altered tinctorial reaction. The cytoplasm of nucleated red cells, especially of the megaloblast, often shows this change. Some authorities maintain that this abnormal staining quality is an indication of immature cell development, while others regard it as a sign of stroma degeneration. Oval or "ring-like" bodies reacting to basic dyes are occasionally observed in the red cells which some students assert are the remains of a nuclear structure. Red cells which contain granular areas having a basic stain affinity scattered through the cytoplasm, appearing in some corpuscles as a fine stippling and in others as coarse irregular granules, are described as cells showing *granular basophilia.* This condition is observed in severe anæmias, especially of the pernicious type, in leukæmia, and constantly in chronic lead poisoning. Some investigators regard it as an indication of degeneration of the cells, while others are inclined to view this feature as an evidence of nuclear fragmentation.

NUCLEATED RED BLOOD-CELLS. — Nucleated red blood-corpuscles are normally found in the blood during the early months of fetal life and in the blood-marrow of all individuals. Two principal types are found, normoblasts and megaloblasts.

Normoblasts. — This cellular element, a normal constituent of the bone-marrow of the healthy adult, is about the size of the normal erythrocyte, and consists of a single oval or round nucleus (rarely two or three), which reacts intensely to basic stains, while the cytoplasm has an acidophilic affinity like a normal erythrocyte. At times this cell contains an irregularly shaped nucleus or several may be noted in a single cell. The nucleus is often eccentrically placed, and sometimes extrudes from the cell or may be found free in the plasma. The occurrence of normoblasts in the circulation of the adult is generally regarded as a sign of rapid blood regeneration, well illustrated after a profuse traumatic hemorrhage, where arge numbers of these cells often are present in the blood, a condition which Von Noorden has termed "blood crisis." Cells having the diameter ranging from 4 to 6 microns, with a round or oval nucleus reacting sharply to basic dyes, and a shrunken irregular protoplasm, called microblasts, probably represent normoblasts having a degenerated cytoplasm. *Megaloblasts* vary in size between 11 and 20 microns in diameter, and consist of a large nucleus of loose texture staining feebly, surrounded by a comparatively small amount of cytoplasm. A clear hyaline space or ring sometimes separates the nucleus from the protoplasm, which not infrequently has a polychromatophilic reaction. Fetal bone-marrow normally contains megaloblasts. Most writers regard the presence of megaloblasts in the circulating blood of the adult an indication of a reversion of the marrow activity to an earlier type similar to that found in the fœtus. These cells are found in grave forms of anæmia, as typified in pernicious anæmia.

Blood-platelets.—Blood-platelets, or blood-plaques, are small, spherical, oval, or irregular bodies, having a pale yellowish color, and measure from 1 to 4 microns in diameter. They are not endowed with amœboid activity, and stain with both acid and basic dyes. Blood-plaques disappear rapidly after the blood is exposed to the air. Some writers consider these elements as being derived from fragmented red blood-cells.

Technic of Counting Blood-platelets (Method of Wright and Kinnicutt).—Two parts of cresyl blue (1 : 300) are mixed with three parts of potassium cyanide (1 : 14000) and rapidly filtered, then immediately used to dilute the blood 1 : 100. The blood must be from a free-flowing drop and the whole procedure must be carried out as quickly as possible. Variations under physiological and pathological influences are common. In many severe secondary anæmias, in leukæmia, in chlorosis, and in rheumatoid arthritis an increase is encountered, while in pernicious anæmia their number is generally reduced. In some of the specific infectious diseases, particuarly in pneumonia and in bubonic plague, an increase is found, while n others, notably in erysipelas, in malaria, and in typhus fever, there s a decrease. The average per cubic millimetre at sea level is 302,000, and 340,000 at an altitude of 6000 feet—a percentage increase of 12.2. (Webb and Gilbert.) They are consistently increased in tuberculosis of man and guinea-pigs. A marked reduction is frequently seen in purpura and hæmophilia.

Hæmokonia.—In fresh unstained blood there are found, in the plasma, small, transparent, highly refractile bodies, not exceeding one micron in diameter, of spherical, oval, or dumb-bell shape, possessing active molecular motion, which are termed hæmokonia, or blood dust. These bodies are insoluble in ether or alcohol and do not stain with osmic acid. Their significance is as yet unknown; it has been suggested that they represent fragments of cells or free cell granules, as eosinophilic or neutrophilic granules.

Leucocytes.—The leucocytes, or white blood-corpuscles, in a wet preparation of fresh blood taken from a normal individual, appear as pale, colorless, nucleated cells, the greater number of which are granular and endowed with amœboid activity. Ehrlich's classification is generally adopted for clinical work. The following table includes the main varieties of leucocytes with their relative percentages present in the blood of the normal adult:

Polynuclear neutrophiles	60–70 per cent.
Eosinophiles	.5– 4 per cent.
Basophiles or mast-cells	.025– .5 per cent.
Small lymphocytes	20–30 per cent.
Large lymphocytes, hyaline cells, and transitional forms.	4– 8 per cent.

In infancy the percentage of lymphocytes is greater than in adult life, while eosinophiles may reach as high as 14 per cent. in childhood.

Polynuclear Neutrophiles.—These cells, the diameter of which ranges between 7½ and 11 microns, have an irregular nucleus, appearing in various shapes, as in the form of the letters U, Z, S, and a finely granular protoplasm. The irregularly shaped nucleus, which is composed of enlargements or lobes connected by bands, reacts to basic dyes with marked affinity. The granules are fine, of an irregular outline, and absorb acid dyes (finely granular oxyphile cells). According to Ehrlich, the granules have a neutral staining property. The polynuclear neutrophiles possess amœboid and phagocytic properties.

Eosinophiles or polynuclear eosinophiles (coarsely granular oxyphiles) are about the size of or slightly larger than the normal erythrocyte, their diameter ranging from 7 to 10 microns. They possess a nucleus similar in structure and tinctorial qualities to that of the polynuclear neutrophile; their protoplasm contains coarse, highly refractile, oval or spherical granules, staining deeply with acid dyes. They are endowed with active amœboid qualities.

Basophiles or Mast-cells.—Under this term are classified leucocytes which have a lobed or twisted nucleus like that of the neutrophiles and a cytoplasm beset with very irregularly shaped basophilic granules of varying size. The granules are not colored with Ehrlich's triple stain, but may be plainly seen when treated with Leishman's or Ehrlich's dahlia mixture.

Small Lymphocytes. — These are essentially non-granular cells, the majority being about the size of the normal erythrocyte. They consist of a large circular or oval nucleus, which has a decided basic property and a relatively small amount of protoplasm, reacting feebly to basic and occasionally to acid stains. With Ehrlich's triple stain, the cytoplasm is colored a pale pink or gray, while with Leishman's eosin-methylene-blue mixture, a light blue, showing less basic affinity than the nucleus. Lymphocytes treated with Leishman's stain occasionally show a few fine pink granules in their cytoplasm. These cells are neither amœboid nor phagocytic.

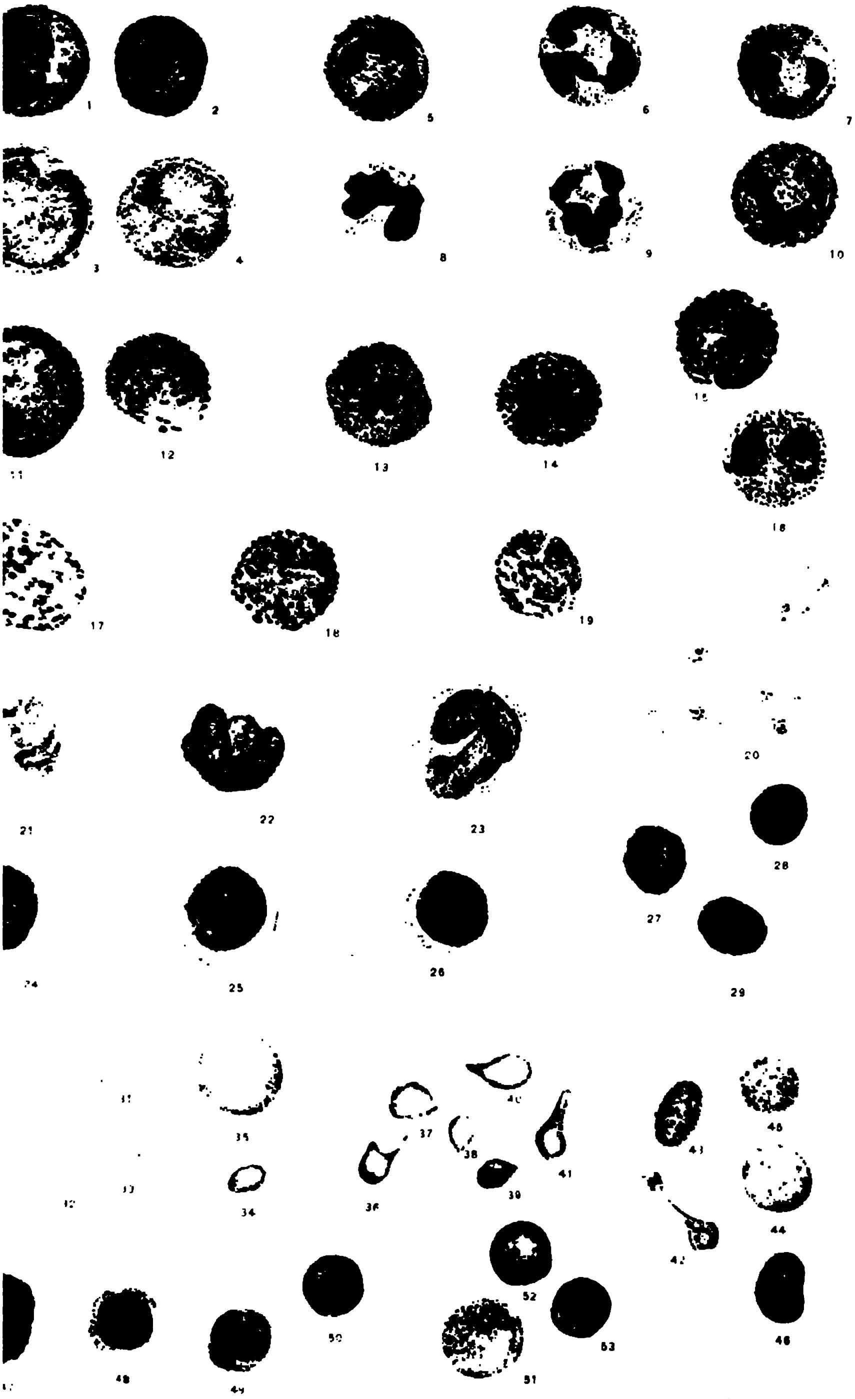
Large Lymphocytes. — Several varieties of leucocytes are embraced under this heading—*lymphocytes proper of large size,* generally regarded as the product of lymphatic tissue, and *large mononuclear or hyaline cells,* probably of bone-marrow origin. A distinction cannot always be made between large lymphocytes and hyaline cells, since they resemble each other as to structure and tinctorial reactions. The nucleus of the latter cell is round or oval. The protoplasm of the lymphocyte has a slightly stronger basic affinity than that of the large mononuclear. From a clinical standpoint this differentiation does not appear important. The large lymphocytes have a relatively smaller nucleus than the small forms, and stain less intensely. *Transitional forms* closely resemble large lymphocytes and hyaline cells in size and staining qualities, but differ from

DESCRIPTION OF PLATE IV.

1. Neutrophile myelocyte.
2. Neutrophile myelocyte showing indentation of its nucleus.
3, 4. Neutrophile myelocytes.
5, 6, 7, 8, 9, 10. Polynuclear neutrophiles.
11, 12. Eosinophile myelocytes.
13, 14, 15, 16. Polynuclear eosinophiles.
17. Basophile myelocyte.
18, 19. Polynuclear basophiles.
20. Blood platelets.
21. Large mononuclear form.
22, 23. Transitional forms.
24, 25. Large lymphocytes.
26. Lymphocyte showing acidophilic granules in its protoplasm:
27, 28, 29. Small lymphocytes.
30, 31, 32. Normal erythrocytes.
33, 34. Microcytes.
35. Macrocyte.
36, 37, 38, 39, 40, 41. Poikilocytes.
42, 43, 44, 45. Erythrocytes containing basophilic granules.
46. Erythrocyte exhibiting polychromatophilia and granular basophilia.
47. Megaloblast.
48, 49, 50. Normoblasts.
51, 52, 53. Erythrocytes with polychromatophilic protoplasm.

(Leishman's Stain.)

PLATE IV



L. Schmidt

(LEISHMAN'S STAIN)

these leucocytes in having an indented or distorted nucleus, resembling the form of the nucleus of some of the polynuclear neutrophiles.

Hyaline cells are said to possess active phagocytic and amœboid properties.

Myelocytes.—These cells, which are normal constituents of the bone-marrow and only present in the blood in pathological states, are in the main large cells supplied with a large circular, oval, or slightly indented nucleus, staining feebly with basic principles, and surrounded by a granular protoplasm.

These cells are classified into three groups, depending on the microchemical reaction of the cell-granules, namely into neutrophilic, eosinophilic, and basophilic varieties. The neutrophilic myelocyte is the commonest form. Neutrophilic granules of myelocytes do not stain as distinctly as those found in the polynuclear cells. Types intermediate between typical polynuclear neutrophiles and typical neutrophilic myelocytes are observed in some pathological conditions, especially myelogenous leukæmia and not infrequently in leucocytosis. Myelocytes often show indistinct granulations embedded in a feebly basic protoplasm; these forms are considered by some to be closely related to cells farther back in their ancestral development.

Atypical lymphocytes are not infrequently encountered in severe anæmias, particularly in lymphatic leukæmia. Some of these cells are almost devoid of protoplasm, while others contain a distorted nucleus. The term neutrophilic pseudolymphocyte has been suggested for those cells which have a round nucleus, rich in chromatin, surrounded by a narrow rim of protoplasm, filled with neutrophilic granules. These leucocytes may represent small myelocytes, as their color characteristics, excluding the strong basic nucleus, suggest neutrophile myelocytes. Türk employs the name "stimulation form" for moderately large leucocytes having a single round weakly basophilic nucleus, and a non-granular cytoplasm, which stains a brown color with the triple mixture.

Development of Leucocytes.—Most authorities regard the bone-marrow and the lymphatic tissues as the seats of leucocyte formation, the former being concerned in the development of polynuclear neutrophiles, eosinophiles, basophilic and hyaline cells, while the latter appear responsible for the production of lymphocytes. In the bone-marrow are found groups of cells, "leucoblastic areas," consisting of myelocytes surrounded by polynuclear elements, while erythroblastic zones are present principally along the margins of vascular spaces. It is conceded by most authorities that the polynuclear neutrophile is developed from the neutrophile myelocyte, the polynuclear eosinophile from the eosinophile myelocyte, and a basophilic cell from its parent marrow cell. In the transformation of the myelocyte into the polynuclear leucocyte, the nucleus undergoes condensation and lobulation, the size of the cell decreases, and the staining reaction of the nucleus and of the granules intensifies. The large mononuclear or hyaline and transitional leucocytes are probably formed in the marrow. The lymphocytes are derived from lymphatic tissue, the small form being the progeny of the large cell.

Number of Leucocytes. — Many circumstances affect the number of leucocytes in the circulation. The colorless corpuscles range between 6000 and 8000 per cubic millimetre in a healthy person; this standard is, however, subject to slight variations, beyond these limits, in certain physiological, and often to pronounced alterations in pathological states. The number is influenced by the condition of body nutrition, *e.g.*, in prolonged starvation low counts are conspicuous, by unequal distribution of the cells, by blood dilution and by blood inspissation. The most acceptable theory, advanced to explain the occurrence of an increase in the number of colorless elements (leucocytosis) in disease, points out that irritants acting in the tissues produce chemical substances which attract certain leucocytes towards the seat of mischief, and cause the leucocyte forming organs to pour out an increased number of cells. This attraction force has been termed "*positive chemotaxis*," in contradistinction to a repelling action set up by some irritants called "*negative chemotaxis.*" The degree of leucocytosis depends mainly upon the intensity of the chemotactic force and the responding powers of the individual.

Leucocytosis.—The term leucocytosis or hyperleucocytosis designates an increase in the number of leucocytes in the peripheral blood over the normal standard. This increase, as a rule, involves a marked percentage gain in the polynuclear neutrophile elements with a fall in the percentage of other forms, but sometimes comprises a proportionate rise in all the varieties, or a percentage gain in the lymphocytes, eosinophiles, or basophiles. Leucocytosis is classified into special forms, depending on physiological or pathological disturbances; these types being further subdivided into special varieties, dependent upon certain etiological factors,—*e.g.*, digestive, inflammatory, toxic, malignant, and post-hemorrhagic leucocytosis. A rise in the number of polynuclear neutrophile cells is called polynuclear neutrophile leucocytosis; an augmentation in lymphocytes is termed lymphocytosis; an increase of eosinophiles is known as eosinophilia; the latter forms also being sub-grouped into (a) an absolute and (b) a relative increase; *e.g.*, absolute lymphocytosis is shown by a gain in the total count with a rise in the percentage of lymphocytes, while relative lymphocytosis implies a percentage gain without an increase in the total number of these cells. The terms absolute and relative as applied to leucocytosis are often misleading to the student.

Physiological Leucocytosis.—The leucocytosis which depends upon physiological factors is, in the main, slight, of short duration, and commonly involves a proportionate increase in all of the forms of leucocytes, less often an absolute and a relative gain in the polynuclear neutrophile elements.

LEUCOCYTOSIS OF DIGESTION. — In nearly all healthy individuals, during the period of digestion, from one to four hours after taking food, a slight rise in the number of leucocytes is present, which generally consists of an absolute increase. Some claim that the gain principally involves the neutrophiles, while others assert the lymphocytes are responsible. In the new-born, leucocytosis of digestion is pronounced. In starvation and frequently in the morbid states associated with faulty nutrition, the number of white corpuscles decreases. The rapidity with which digestion leucocytosis manifests itself after taking food is regarded by some writers as bearing a direct relation to the activity of the digestive function. In persons suffering from gastric ulcer, leucocytosis sometimes comes on very soon after taking food, pointing to rapid digestion; while in gastric cancer, it may be delayed or absent. This rule is not constant and little importance should be attached to digestion leucocytosis in diagnosis.

LEUCOCYTOSIS OCCURRING DURING PREGNANCY AND AFTER PARTURITION.—A moderate rise in the number of leucocytes occurs during the later months of pregnancy, and persists for about two weeks after parturition.

LEUCOCYTOSIS OF THE NEW-BORN.—The high counts observed for about ten days after birth are attributed to blood inspissation and to the establishment of digestion leucocytosis. Higher counts are present in early childhood than in the advanced periods of life. Leucocytic oscillations of a very moderate character occur after exercise, massage, after cold or hot bathing, and after the use of electricity.

Pathological Leucocytosis. — INFLAMMATORY AND INFECTIOUS LEUCOCYTOSIS.—The presence or the absence of a leucocytosis in many of the infectious and inflammatory diseases is a sign of considerable importance in diagnosis. Its clinical value is comparable in a measure with other signs, such as temperature range and pulse, certain physical signs, etc. This pathological increase, which is essentially a polynuclear neutrophile leucocytosis, is as a general rule encountered in acute local inflammations, as in furuncles, cellulitis, abscesses, in general sepsis, and in nearly all of the specific infectious diseases, except in uncomplicated cases of enteric fever, paratyphoid infections, tuberculosis, malaria, measles, German measles, influenza, leprosy, and Malta fever. High leucocytic ranges are often attributable to suppurative inflammation. Duration and height of fever have no direct relation to the leucocyte curve. Counts between 15,000 and 20,000 are common, while ranges above 30,000 are uncommon and above 50,000 very rare.

PREAGONISTIC OR TERMINAL LEUCOCYTOSIS.—The exact nature of the leucocytosis which so often precedes death is still a mooted question; many investigators attribute this rise to terminal infections.

MALIGNANT LEUCOCYTOSIS.—Different opinions have been advanced to explain the gain of colorless elements so frequently noted in individuals suffering from carcinoma and sarcoma. Some writers contend that associated inflammatory disturbance about the growth or septic absorption from the tumor induces the leucocytic gain, while others hold that the direct effect of the tumor is the responsible factor. In rapidly spreading malignant growths, especially when metastasis has occurred, counts are generally high and much above those noted in slowly growing tumors; in sarcomata the gain is usually more pronounced than in carcinomata. The cellular rise in the majority involves mainly the neutrophiles, although lymphocytosis, especially in sarcomata, has been recorded. In cases of carcinoma of the stomach, leucocytosis is often absent.

POST-HEMORRHAGIC LEUCOCYTOSIS.—The gain in the white corpuscles which follows and persists for some days after a profuse hemorrhage is generally accounted for by an increased production and output of polynuclear neutrophiles. Some observers hold that the lymph (rich in cells) which passes into the blood after blood loss is the responsible factor.

A leucopenia, lasting for a few hours, precedes the leucocytosis due to hemorrhage. Leucocytosis is present in many cases of secondary anæmia.

Leucocytosis due to Toxic and Therapeutic Agents. — Among the substances capable of producing a rise in the leucocytic standard may be mentioned quinine, ether, chloroform, potassium chlorate, illuminating gas, salicylates, uric acid, and various organic extracts.

Lymphocytosis. — This condition, an increase of lymphocytes, is normal in infants and in young children. A relative lymphocytosis due to diminution of the polynuclear elements has been recorded in the following conditions: chlorosis, pernicious anæmia, severe secondary anæmia, and in some of the infectious diseases, as tuberculosis, enteric fever, malaria, and influenza. A lymphocytosis is sometimes observed in Hodgkin's disease, and often in children suffering from syphilis, anæmia, pseudoleukæmia infantum, rickets, whooping-cough, and gastro-intestinal diseases. Diseases of the lymphatic glands and spleen are sometimes attended by an increase in these cells. Lymphocytosis in most instances is a relative condition. Absolute lymphocytosis of high grade is a constant feature of lymphatic leukæmia. In myelogenous leukæmia the percentage of lymphocytes is reduced, although the total number is increased.

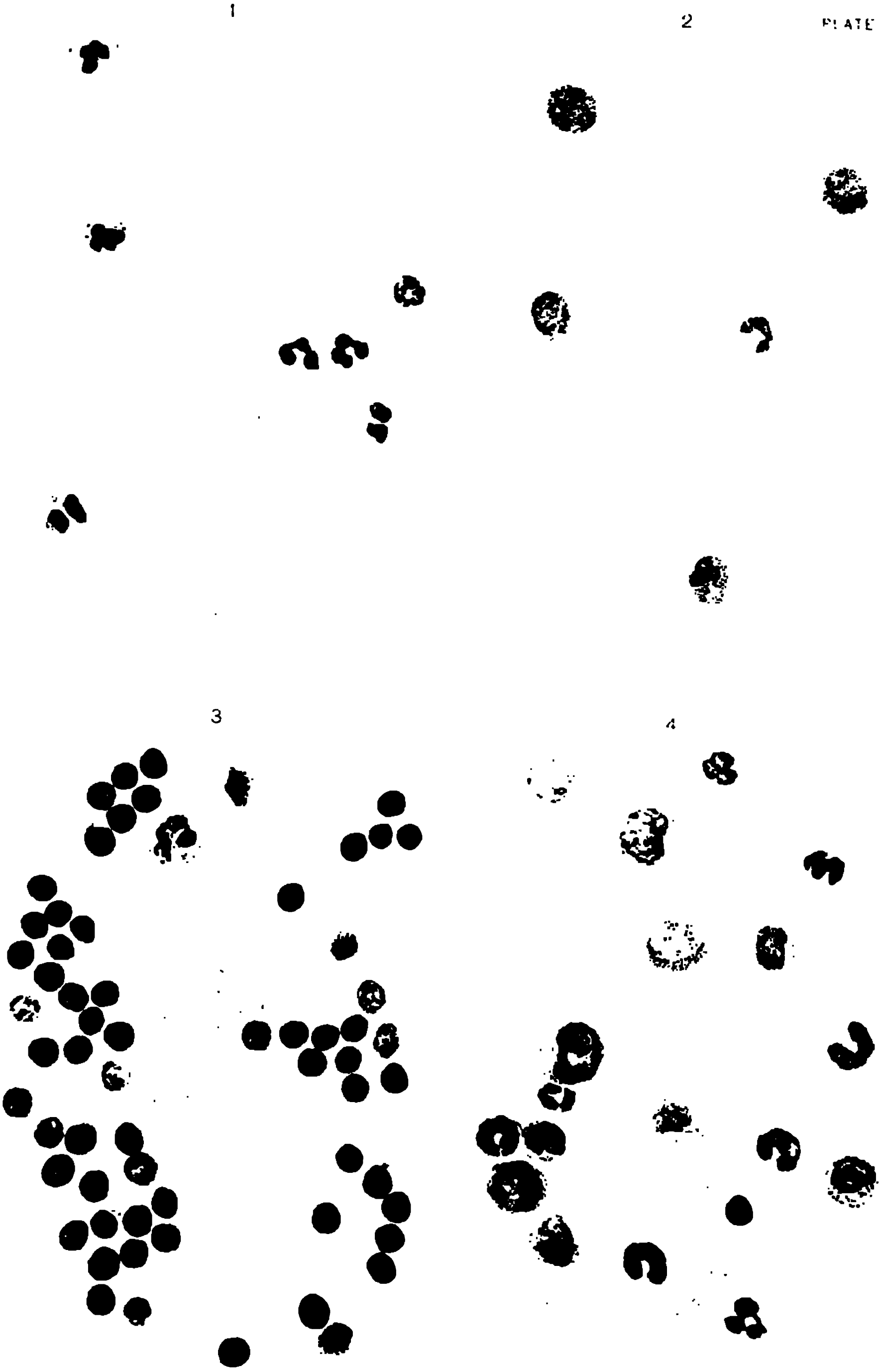
Eosinophilia.—An increase in the number of eosinophiles has been observed in the blood of normal infants, in anæmia pseudoleukæmia infantum, in splenomedullary leukæmia, in bronchial asthma, after hemorrhage, in diseases of the skin, as urticaria, lupus, eczema, leprosy, and pemphigus, after coitus, during convalescence from many of the acute infectious diseases, in bone diseases and in morbid states caused by intestinal worms, particularly in trichiniasis. Definite conclusions regarding the clinical significance of fluctuations in these cells in morbid states cannot be drawn from our present knowledge, although some diagnostic importance should be attached to the almost constant eosinophilia in trichiniasis, in ankylostomiasis, and in bilharzial infection.

Basophilia.—This term is used to express an increase in the number of basophiles in the circulating blood, which is frequently noted in splenomedullary leukæmia. Some writers have reported basophilia in splenic anæmia, in certain skin diseases, in acute bone inflammation, and in gonorrhœa. The clinical significance of basophilia remains unsettled.

Leucopenia.—A decrease in the number of leucocytes is termed leucopenia, or hypoleucocytosis, and may be brought about by physiological and pathological factors. A deficiency in the number of colorless cells is seen in starvation and in malnutrition, and almost constantly in the infectious diseases not associated with leucocytosis, as tuberculosis, malaria, measles, influenza, enteric fever, Malta fever, and German measles. Low leucocyte counts are common in pernicious anæmia, in chlorosis, in splenic anæmia, and in profound symptomatic anæmias.

Myelæmia.—The appearance of myelocytes in the blood, spoken of as myelæmia, points to rapid leucocyte proliferation in the bone-marrow. Myelocytes are often found in the circulation when leucocytosis is present. In splenomedullary leukæmia this condition is most striking, in pernicious anæmia, cholorsis, lymphatic leukæmia, Hodgkin's disease, and in profound secondary anæmias, a small number of myelocytes is not infrequent.

PLATE V



J. Seamat

1. NEUTROPHILE LEUCOCYTOSIS.
3. LYMPHOCYTOSIS.

2. EOSINOPHILIA.
4. MYELÆMIA.

(LEISHMAN'S STAIN)

Parasites.—Bacteræmia.—Bacteria frequently gain access to the circulation, either from an infected area within the body or from without. Bacteria are seldom found by a microscopical examination of the fresh unstained blood; culturing methods being required to reveal their presence. From a clinical view-point the demonstration of bacteria in the circulation may be regarded as evidence of disease. The detection of micro-organisms in the blood is often essential in establishing a diagnosis of septicæmia, malignant endocarditis, puerperal sepsis, and pyæmia. In some of the specific infectious diseases the exciting principle has been isolated from the blood. In over 80 per cent. of the cases of enteric fever, Eberth's bacillus can be recovered from the blood by culture methods. The specific micro-organisms of paratyphoid fever, croupous pneumonia, anthrax, glanders, leprosy, influenza, plague, tuberculosis, and Malta fever have been isolated from the blood. In septicæmia or septicopyæmia staphylococci, streptococci, and gonococci have been separated from the blood.

Animal Parasites. — From our present knowledge it is impossible to fix a sharp dividing line separating some of the lower vegetable and animal parasites. Investigators seem to favor placing the Treponema pallidum and the spirochæta of relapsing fever among the animal parasites. For a description of the animal parasites found in the blood, the reader is referred to the section which deals with diseases caused by animal parasites.

Method of Examination for Malarial Parasites.—Fresh, unstained blood is more suitable for study than stained blood, because it enables the examiner to observe the activity of the parasites. The most desirable time for conducting the examination is during the period when pigmented forms are present. The intracellular pigmented bodies are usually most abundant about 6 or 8 hours before a paroxysm. Considerable experience is necessary before the various types can be differentiated; especially is this true of hyaline unpigmented forms. The skilled microscopist must often make a prolonged search before parasites are detected in malarial blood. The fresh blood is prepared in the usual manner, between a cover-glass and slide, and the examination is made with an oil-immersion lens with moderate illumination. When the examination of fresh blood is impracticable, dried specimens treated by one of the different forms of the Romanowsky stain as variously modified by Leishman and others (Wright, Hastings) constitute a satisfactory means of direct diagnosis. The effectual administration of quinine reduces the number of parasites and causes their disappearance from the peripheral circulation. In a suspected case the examination of the blood should precede the institution of treatment.

VI.

THE EXAMINATION OF THE URINE.

The essential diagnostic principles of urinalysis, coupled with certain methods of examination required for general clinical work, are detailed in the following section. Tests suitable for the general practitioner must in the main be simple and easily applied. The importance of the findings obtained by careful and systematic studies of the urine, when correctly interpreted and given their proper place in a symptom-complex, can scarcely be overestimated in diagnosis. In a considerable group of morbid states the urinary picture is absolutely necessary in establishing a final diagnosis, while in a large number of cases the results of the examination form a link in the chain of symptoms completing the diagnosis. Negative reports in some instances are essential for the solution of certain clinical problems. For an exhaustive account of the urine, which does not fall within the scope of this book, the reader is referred to special treatises on this subject.

PHYSICAL EXAMINATION.

Amount.—The daily standard for a healthy adult, as estimated by different authorities, varies between 900 and 2000 c.c. Most observers fix the amount between 1000 and 1500 c.c., although temporary oscillations beyond these limits, of slight, moderate, or even of an excessive degree, are often physiological. The urinary secretion is greater during the day than through the night, more abundant in cold than in hot weather, and relatively more active in children and infants than in adults. Polyuria depends in the main on, (a) increased ingestion of water, (b) heightened blood-pressure, and (c) on increased activity of the renal epithelium, while oliguria results from (a) lessened consumption of water, (b) lowered blood-pressure, and (c) impaired function of kidney epithelium.

Specific Gravity.—In health the specific gravity ranges between 1.015 and 1.025 while the daily output of urine is within normal limits. In general terms the specific gravity is a fair index of the bulk of solids eliminated. An approximate estimate of the weight of urinary solids expressed in grammes for a thousand cubic centimetres of urine may be determined by multiplying the second and third decimal figures of the specific gravity by two. Pathologically, wide variations, as low as 1.002 and as high as 1.040, are frequent. Ranges above 1.050 are extremely uncommon. The volume of urine and the specific gravity in the main fluctuate in an inverse manner; therefore, without a knowledge of the daily output the density has practically no clinical significance. A correct specific gravity determination can only be made from a mixed twenty-four hours' sample. In diseases associated with polyuria lowered ranges are the rule. A notable exception is found in the case of diabetes mellitus, in which the increased density is caused by the presence of glucose. A diminished volume of urine of low specific gravity is seen in a number of chronic diseases, and often in cases of œdema. Large urinometers are preferable to small instruments for measuring the specific gravity, since the divisions of the scale can be read with greater accuracy. The large amount of urine necessary to float these hydrometers is, however, an objectionable feature. This may be overcome by estimating the density with a set of two or three pycnometers of moderate size, each of which represents a portion of the scale. In measuring the specific gravity the following precautions are essential for accurate results: The hydrometer should be placed into cool urine, the surface of which is free from foam; the instrument should not touch the sides of the hydrometer jar.

Color.—Normal urine owes its color to urochrome and urobilin, chiefly to the former, while the tints of many pathological urines depend upon such substances as biliary pigment, hæmoglobin, methæmoglobin, hæmatin, hæmatoporphyrin, melanin, indican and alkaptone. After the ingestion of iodine, phenol, salol, senna, santonin, and methylene blue, the urine often has an abnormal hue. The shade of the color depends chiefly upon the amount of urinary water, so that concentrated specimens are usually dark, while those of low specific gravity are generally pale. An exception to this rule is noted in diabetic urine, which is light yellow or pale greenish-yellow, of increased density and of excessive quantity. In acute febrile diseases and pernicious anæmia the urine is high colored, while in chlorosis, diabetes insipidus, and contracted kidney it is pale. Bile pigment is responsible for dark yellow, yellowish-green, dark brown, and rarely, black urine, which on shaking develops a yellowish foam; blood pigment stains the urine bright red, dark red, reddish-brown, or rarely, black. Melanotic pigment imparts a brown or black appearance which generally develops some time after the urine is voided, but occasionally is noted in the fresh specimen. A similar color occurs in alkaptonuria, which condition can be differentiated from melanuria by testing with Fehling's solution; the alkaptone bodies reduce the copper salt, while the latter gives a negative reaction. The ingestion of phenol and its allied compounds may cause a greenish-black discoloration, of methylene blue a green or blue urine, of santonin a yellow, and of rhubarb an orange-colored urine. A milky appearance is noted in chyluria and at times in phosphaturia and pyuria. The presence of hæmatoporphyrin may impart a dark red color.

Transparency. — Normal urine immediately after being voided is generally clear; on cooling it occasionally becomes turbid, due to precipitation of urates or phosphates. Turbidity, associated with a sediment, is one of the characteristic features of many abnormal urines, and may depend upon an excess of urates or phosphates, or on the presence of epithelial elements, pus-cells, red blood-corpuscles, chyle, or bacteria.

Odor.—This property of the urine, although of little clinical significance, except in a few instances, occasionally attracts the attention of the patient who suspects that an abnormal state of the kidneys exists. The odor of normal urine is sufficiently familiar to require no special description. Urine decomposed by bacterial growth within the bladder, or after it has been voided, emits an ammoniacal stench. Acetone, when present in large amount, may give the urine a sweetish, fruit-like odor. The ingestion of turpentine, terebene, asparagus, and onions imparts peculiar odors.

Reaction.—A normal, mixed twenty-four hour sample in nearly every instance is acid, while individual specimens passed during the day vary considerably in reaction. For clinical purposes the reaction may be determined with litmus paper. An alkaline reaction after a heavy meal is attributed to the increased alkalinity of the blood during gastric digestion. The ingestion of food rich in vegetables, or the administration of tartaric, citric, or acetic acid lessens acidity, or produces alkalinity, while a diet rich in meat or the taking of mineral acids intensifies the acidity. The reaction of normal urine is held to be due to diacid phosphate; Folin, however, contends that free organic acids are in part responsible. The total acidity of a twenty-four hour collection of healthy urine is equal to from 1.5 to 2.3 grammes of hydrochloric acid. Alkalinity is caused by the presence of alkalies in excess of acids. An amphoteric reaction depends on a balance existing between the acid and basic equivalent of the urinary salts. Urine that has been exposed to the air for some time becomes alkaline from ammoniacal decomposition. Alkaline urine is frequently seen in cystitis, but in a number of cases of bladder inflammation, both acute and chronic, the urine is acid. A strongly acid urine occurs in gout, diabetes, rheumatic fever, in some varieties of nephrolithiasis, in leukæmia, in intestinal and stomach diseases associated with diminished or abolished gastric secretion, in scurvy, in chronic nephritis, and often in febrile states. Lowered acidity, and occasionally alkalinity, is seen in anæmia, notably pernicious anæmia and chlorosis, following the crisis of pneumonia, after blood transfusion with saline solution, in hæmaturia, and when transudates are rapidly absorbed.

Sediments. — Occasionally deposits of uric acid, amorphous urates, carbonates and phosphates, and invariably those consisting of pus, epithelial and red blood-cells, are significant of morbid states. The presence of a sediment of uric acid crystals, amorphous urates, or phosphates need not, and as a rule does not, imply an increased output of these salts, but may depend on changes in the reaction of the urine.

MICROSCOPICAL EXAMINATION.

Microscopical examination of sediments is of cardinal importance in urinalysis. A sediment best suited for this method of study should be secured from a fresh specimen of urine by centrifugalization. When the examination cannot be made immediately after the urine is voided, it is advisable to add an antiseptic to it, such as a little powdered camphor, a few drops of formaldehyde solution, or thymol, in order to prevent decomposition.

Crystalline and Amorphous Substances Present in Acid Urine.—CALCIUM OXALATE.—Crystals of calcium oxalate are found in many sediments and appear in various sizes, mostly in the form of colorless octahedra, generally designated "envelope crystals," sometimes as dumb-bell figures and rarely as oval disks. These crystals are soluble in hydrochloric acid, but not in acetic acid or sodium hydrate. Normal urine may contain these crystals, after the eating of tomatoes, asparagus, garlic, rhubarb, or oranges. After the ingestion of bicarbonate of soda, in certain forms of gastro-intestinal neuroses, in jaundice, in phthisis, and in diabetes mellitus, oxalate crystals are sometimes noted. Renal calculi composed of this substance are not uncommon. Oxaluria can only be regarded as pathological when a chemical examination shows an increased quantity of oxalic acid, provided the factors responsible for its occurrence in health can be excluded.

Uric Acid.—This substance, as a rule, crystallizes in the form of whetstone shaped crystals, arranged singly or in clusters, and occasionally in the form of dumb-bell figures, or as rhombic plates. These crystals vary considerably in size, and their color ranges from a pale yellow to a dark brown. Uric acid crystals dissolve in a sodium hydrate solution, and when this test is followed by the addition of hydrochloric acid to the alkaline solution, rhombic plates appear. Uric acid crystals are frequently seen in the urine when the uric acid output is normal or even decreased, since this substance is more readily soluble in warm than in cold solution. Urinary inspissation is another factor which determines precipitation. In leukæmia and during or immediately following acute gout, the urine contains an excess of uric acid, and may reveal an abundant precipitate of these crystals (brick-dust sediment). Calculi formed of uric acid are among the most common renal concretions. When amorphous granules of sodium and potassium urate occur in the urine in abundance, they impart a turbidity to it, which is often associated with a light yellow or pink sediment. This precipitate disappears upon heating the urine to a temperature of 50° C.; on the addition of hydrochloric acid to the urine, amorphous urates are converted into uric acid crystals. Amorphous sediments of urates are frequently present in scanty, concentrated urines, such as occur in fevers.

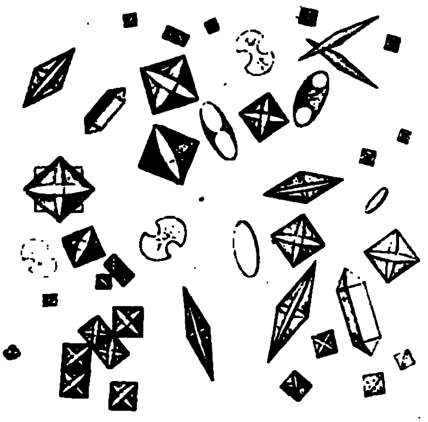


Fig. 107.—Calcium oxalate crystals.

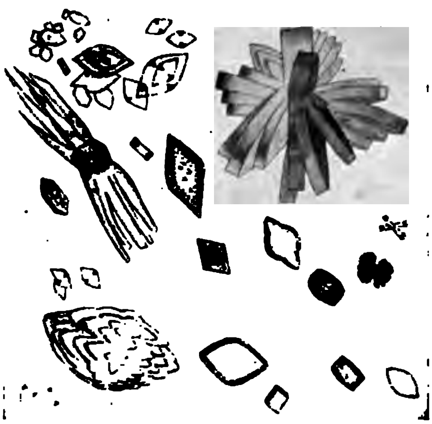


Fig. 108.—Uric acid crystals.

Calcium sulphate is seen in the form of long, transparent colorless needles, or elongated platelets, arranged singly or in crystalline masses. They are insoluble in ammonia, acetic acid, and alcohol. Von Jaksch found these crystals in association with triple phosphates and calcium carbonate in the urine of a patient who showed a tendency to calculus formation. No special clinical significance has as yet been attached to the presence of these crystals.

Hippuric acid occurs as rhombic prisms or slender needles arranged separately or in clusters. These crystals are soluble in ammonia and insoluble in hydrochloric acid. They have been noted, though very infrequently, in febrile diseases, in diabetes, and after the ingestion of benzoic acid, salicylic acid, cranberries, mulberries, blueberries, and prunes.

Bilirubin is found as fine needles arranged in clusters or rhombic plates having a yellow or ruby color, or as an amorphous substance. This sediment is soluble in sodium hydrate and chloroform; on treating the crystals with nitric acid a green color appears about them. Von Jaksch contends that the presence of crystals of bilirubin in the urine, as a rule, points to antecedent hemorrhage into the urinary tract or to the rupture of an abscess. Their presence, either free or imbedded in cells or tube-casts, has been recorded in acute nephritis, chronic interstitial nephritis, amyloid kidney, jaundice, acute yellow atrophy of the liver, hepatic cirrhosis, phosphorous poisoning, carcinoma of the bladder, and after the rupture of a suppurating hydatid cyst into the urinary tract.

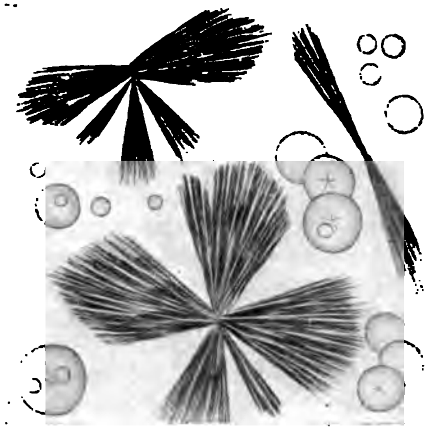


Fig. 109.—Leucin spheres and tyrosin crystals.

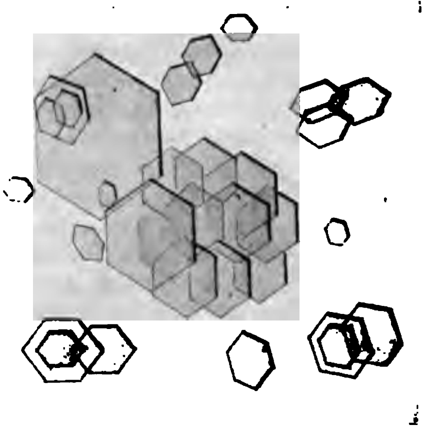


Fig. 110.—Cystin crystals.

Leucin and Tyrosin.—These substances are never found in normal urine. They are generally held in solution unless present in considerable quantities, when they separate in a crystalline form. Their precipitation can be brought about by treating the urine with an excess of basic plumbic acetate; then filtering; and to filtrate hydrogen sulphide is added to remove the excess of lead acetate. The filtrate is then evaporated to a small volume. Absolute alcohol is used to remove traces of urea. The insoluble residue is finally extracted with alcohol containing a little ammonia. Leucin and tyrosin will precipitate in this concentrated solution. Leucin presents the appearance of spheres of varying sizes, sometimes termed "leucin balls." These spheres have a brown color and show delicate lines radiating from their centre to the periphery. Leucin spheres are insoluble in ether. Tyrosin crystals are noted in the form of slender needles, frequently grouped in bundles. They dissolve in ammonia and hydrochloric acid, but are insoluble in acetic acid. Leucin and tyrosin occurring mostly together have been observed almost constantly in acute yellow atrophy of the liver, phosphorous poisoning, and Weil's disease, occasionally in catarrhal jaundice, cholelithiasis, cirrhosis and cancer of the liver, enteric fever, gout, and diabetes, and rarely in a limited number of other conditions.

XANTHIN is found in the form of colorless crystals resembling those of uric acid in outline. They are soluble in ammonia. These crystals are rare ingredients of urinary sediments; calculi consisting of xanthin have been found by some investigators.

CYSTIN crystals are six-sided colorless plates, which are soluble in ammonia and insoluble in acetic acid and water. Von Jaksch recommends the following microchemical test for their detection: A drop of hydrochloric acid is added to the urinary sediment. When the acid comes in contact with cystin, there develop prismatic crystals (hydrochlorate of cystin) which are grouped in masses suggesting a rosette form. Cystinuria is a rare condition. In some cases it is unattended with symptoms, while in others it is responsible for calculus formation.

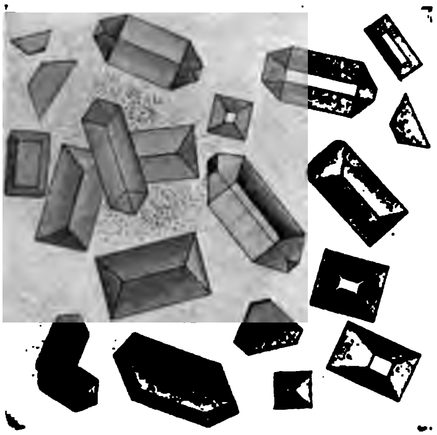


FIG. 111.—Triple phosphate crystals

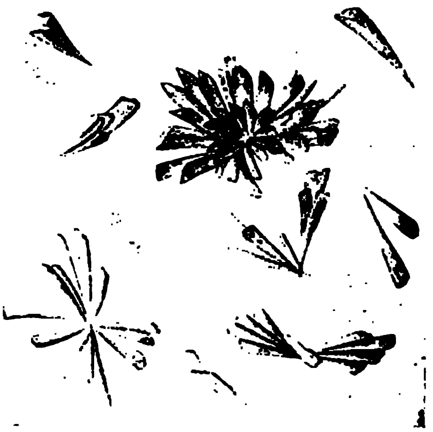


FIG. 112.—Neutral calcium phosphate crystals.

SOAPS OF LIME AND MAGNESIA consist of needles arranged in bundles or sheaves, sometimes radiating from a central point forming a sphere. They resemble tyrosin crystals in form and arrangement. These crystals are rarely found. They have been noted in septicæmia.

FAT GLOBULES can be recognized by their highly refractive appearance and solubility in ether. Among the conditions in which lipuria is seen, may be mentioned, acute and chronic parenchymatous nephritis, diabetes mellitus, bone disease and injury, chyluria, phosphorus poisoning, and certain diseases of the liver and pancreas.

Crystalline and Amorphous Substances found in Alkaline Urine.—AMMONIOMAGNESIUM PHOSPHATE or triple phosphate crystals are colorless and vary considerably in size and appearance. In their most characteristic form they occur as the so-called "coffin-lid" crystals; others resemble fern leaves in outline, while some are arranged in the shape of the letter "X." Triple phosphate crystals are found in association with amorphous deposits of phosphates, carbonates, and at times with ammonium urate.

NEUTRAL CALCIUM PHOSPHATE occurs in alkaline, faintly acid, or amphoteric urine in the form of sheets or needle-like crystals, the latter being arranged singly or in masses forming dumb-bell or star-shaped figures. These bodies are soluble in acetic acid.

NEUTRAL MAGNESIUM PHOSPHATE crystals are colorless, refractile, elongated plates with irregular or bevelled edges; they are soluble in acetic acid.

CALCIUM CARBONATE crystals are found in alkaline urine associated with amorphous carbonates. They are dumb-bell shaped bodies which dissolve in acetic acid with the liberation of gas.

AMORPHOUS PHOSPHATES of calcium and magnesium and AMORPHOUS CARBONATES are of common occurrence in alkaline urine. The addition of a fixed alkali to urine will precipitate amorphous phosphates and carbonates. On heating urine having a low acid or alkaline reaction, a white cloud similar to that produced by albumin appears which consists of phosphates or carbonates. On the addition of acetic acid, phosphates and carbonates are dissolved. The solution of the latter is attended with the evolution of gas. Amorphous phosphates or carbonates are often responsible for a turbid urine with a heavy sediment. Microscopically, these bodies appear as colorless, coarse granules, which are soluble in acetic acid. Phosphatic sediments are occasionally symptomatic of certain types of dyspepsia, of neurasthenia, of diseases associated with marked gastric acidity, and of some cases of cystitis. A deposit of phosphates does not of itself indicate an increased output of phosphoric acid; this can only be determined by quantitative analysis.

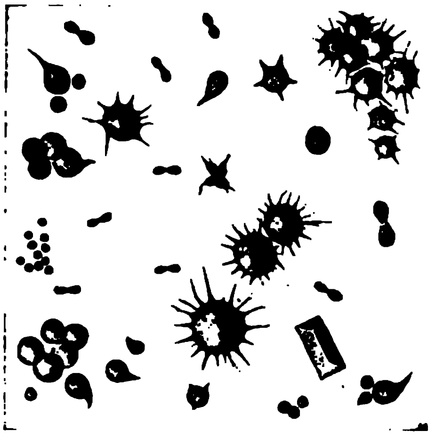


FIG. 113.—Ammonium urate crystals.

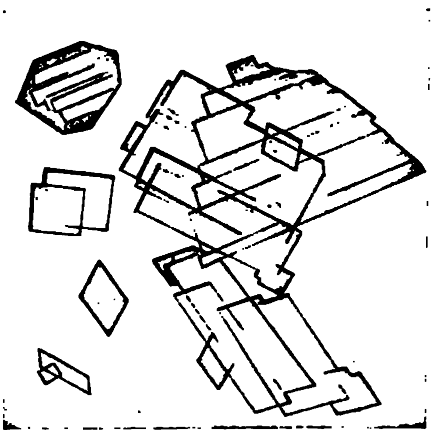


FIG. 114.—Cholesterin crystals.

AMMONIUM BIURATE appears as dark brown spheroidal bodies from the surface of which spicules project, the so-called "thorn-apple" or "hedgehog" crystals, and in the form of coarse yellow needles grouped in clusters. Ammonia biurate may be associated with triple and amorphous phosphates.

Acetic acid will cause solution of these crystals with the formation of uric acid. Ammoniacal fermentation of the urine, occurring in the bladder or after it has been voided, is responsible for the precipitation of ammonium biurate.

CHOLESTERIN crystals occur as colorless thin plates. They are rare constituents of urinary sediment and have been observed in hydatid cystic kidney, pyonephrosis, hydronephrosis, and cystitis.

INDIGO, a rare ingredient of the urinary sediments, is found in the form of a blue crystalline body consisting of needles grouped in a stellate manner or as rhombic plates, and also as bluish amorphous granules. The amorphous material is not infrequently present in decomposing urine. Indigo is a rare constituent of urinary calculi.

Cellular Deposits.—EPITHELIAL CELLS.—Epithelial cells in small numbers, not sufficient to impart a sediment or cloudiness to the urine, can be found in every specimen. In many instances their number is so large as to justify a diagnosis of an inflammatory, atrophic, or degenerative lesion involving the genito-urinary tract. The predominance of one or several of the types of epithelial elements, unless correlated with other clinical data, has little significance. It is impossible to locate definitely a lesion of the genito-urinary tract from the morphological characters of epithelium alone. This difficulty is apparent when we consider, (1) similarity of many of the cells of different parts of the tract, notably those derived from the pelvis of the kidney, ureters, and bladder, (2) alterations in the shape of these delicate formations due to the action of the urine, and (3) the influence exerted on these cells by morbific factors, such as necrosis, pressure, etc. Therefore, little importance should be attached to their histological structure. Round cells, having a relatively large nucleus, are derived from the tubular structures of the kidney and the deeper layers of the renal pelvis. When casts are beset with round cells it points to a renal origin of this epithelium. An abundance of round cells in the absence of casts, especially when pus-cells and subjective symptoms pointing to pyelitis are present, is suggestive of origin from the renal pelvis. This opinion is strengthened by the presence of polygonal and conical cells, some of which have a tail-like elongation of their protoplasm; these cells are often arranged in a stratified manner. Round cells are also derived from the male urethra, while small conical and polygonal cells originate in the superficial layers of the pelvis of the kidney. Cylindrical cells with

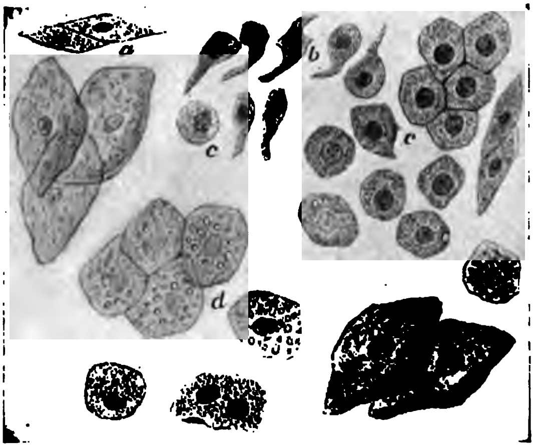


FIG. 115.—Epithelial cells. *a*, flattened cells; *b*, conical cells with tail-like prolongations; *c*, round and polygonal cells; *d*, degenerated cells.

bluntly pointed ends are found in the superficial layers of the male urethra. Flattened, oval or circular, or polygonal cells line the superficial layers of the ureter, bladder, prepuce, fossa navicularis, and vagina. Large squamous cells are generally derived from the vagina or prepuce. Protoplasmic degeneration of the epithelial cells is extremely common. A final diagnosis should never depend on the characters of the epithelia in the absence of clinical findings.

Leucocytes.—A careful search in every normal or morbid specimen of urine will reveal a few leucocytes. The action of the urine upon these cells causes alterations in their structure. In acid urine they have a distinct nucleus, while in alkaline urine their protoplasm is swollen and cloudy, obscuring the nucleus. By treating a specimen of urine having a weakly acid or alkaline reaction with acetic acid, the nuclear outline becomes sharply marked. The leucocytes stain a mahogany brown with a solution of iodo-potassic iodide (glycogen reaction), while epithelial cells are tinted a light yellow with this reagent. Pus-cells in considerable or large numbers frequently indicate inflammatory disease of some part of the genito-urinary tract. They occur in renal hyperæmia, nephritis, abscess and tuberculosis of the kidney, pyelitis, urethritis, cystitis, prostatitis, epididymitis, and orchitis. A leucorrheal discharge is a common cause of pyuria. Urine containing many pus-corpuscles generally gives a positive reaction for albumin. Renal and extrarenal albuminuria may coexist.

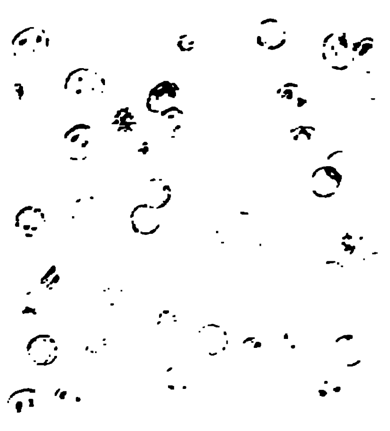


Fig. 116.—Red blood-cells and leucocytes.

Red Blood-cells.—Hæmaturia, or the presence of red corpuscles in the urine, is always pathological except during menstruation. In certain uterine diseases attended with bloody discharge some of the erythrocytes may be washed into the urine. A microscopic inspection serves to recognize red blood-cells in almost every instance, so that chemical tests for their detection are rarely required. In some urines the red cells are unaltered, while in others changes in their structure are found, such as decided shrinkage of the cell, or crenation, or they may be partially or completely decolorized, and appear as pale yellow disks or as faintly outlined rings (phantom corpuscles). The quantity of blood may be sufficient to tinge the urine pale or dark red, but in many cases the amount is so small that the microscopic test is essential in the diagnosis of hæmaturia. When the erythrocytes are intimately mixed with the urine, this suggests a hemorrhage in the kidneys, renal pelvis, or ureters. The presence of dehæmoglobinized corpuscles is noted in kidney lesions, such as congestion and inflammation. Unaltered blood or blood-tinged urine passed at the beginning of micturition is of urethral origin; on the other hand, when blood appears at the end of urination, its source is generally the neck of he bladder. Bleeding may cause coagula of certain shapes; cylindrical lots of large size suggest urethral hemorrhage, those of small diameter ay indicate ureteral hemorrhage, while irregular clots often form in the ladder. Neither the morphological characters of red blood-cells nor the ize and outline of clot can be relied upon to definitely determine the ite of a hemorrhage, unless these findings are supported by other clinical lata. The use of the cystoscope, urethral and ureteral catherization, and xamination with X-rays, especially for renal calculus, afford valuable adjuncts in the diagnosis of hæmaturia. An albumin reaction is invariably obtained when red blood-cells are abundant in the urine, but when only small numbers exist, a negative test is the rule. The quantity of albumin is proportionate to the amount of blood.

Tube-casts.—These are cylindrical bodies moulded in the uriniferous tubules. Their structure is variable, and may consist of a hyaline or waxy material, of cellular bodies, of granular elements, of fat globules, and in rare instances of bacteria or of amorphous substances.

HYALINE CASTS.—These, by far the most common, are slightly refractile, transparent, of regular outline, with rounded ends. They are invisible in a brightly illuminated field on the microscope, so that it is necessary to cut off much of the reflected light with the iris diaphragm in order to bring out their outline. Epithelial cells, leucocytes, red blood-corpuscles, and granules frequently beset these casts, and, indeed, it is not uncommon to find adherent cells or granules so numerous that the hyaline material is obscured. Casts covered with granules are termed hyalogranular. This appearance may make it impossible to distinguish hyaline casts from those composed principally of granules. Clinically, however, the significance of hyaline casts coated with granules and those composed wholly or mostly of granules is identical. It should be remembered that a sharp distinction cannot be drawn between these forms. Hyaline casts are soluble in acetic acid.

WAXY CASTS appear as highly refractile, sharply defined, colorless or yellowish cylinders, showing a tendency to transverse fragmentation. Like hyaline casts they may be studded with cells or granules. They may exhibit an amyloid reaction, but this is no criterion that lardaceous renal disease exists, but, on the contrary, amyloid disease of the kidney is, as a rule, not associated with casts giving this reaction. Not infrequently, casts are observed that cannot be definitely classified as belonging to the waxy or hyaline varieties.

GRANULAR CASTS are composed of fine or coarse granules. Cells forming leucocytic or epithelial casts may show a decided granular protoplasm, so that it becomes difficult to distinguish their outline; these types constitute border line varieties between cellular and granular casts. Clinically, this is of little moment, since the granular or fatty casts represent products of degenerated cells. Acetic acid dissolves granular casts.

FATTY CASTS consist of fat globules derived from degenerated cells. Ether dissolves fatty casts.

EPITHELIAL CASTS are made up of renal epithelial cells, many of which present degenerative changes. These casts may have tubular form.

LEUCOCYTIC CASTS consist of white blood-cells. They are generally recognized at a glance, but should uncertainty arise as to the character of the cells forming these cylinders, this doubt can be settled by treating the specimen with a droplet of acetic acid, which clarifies the protoplasm of the leucocytes and causes the nucleus to become distinct.

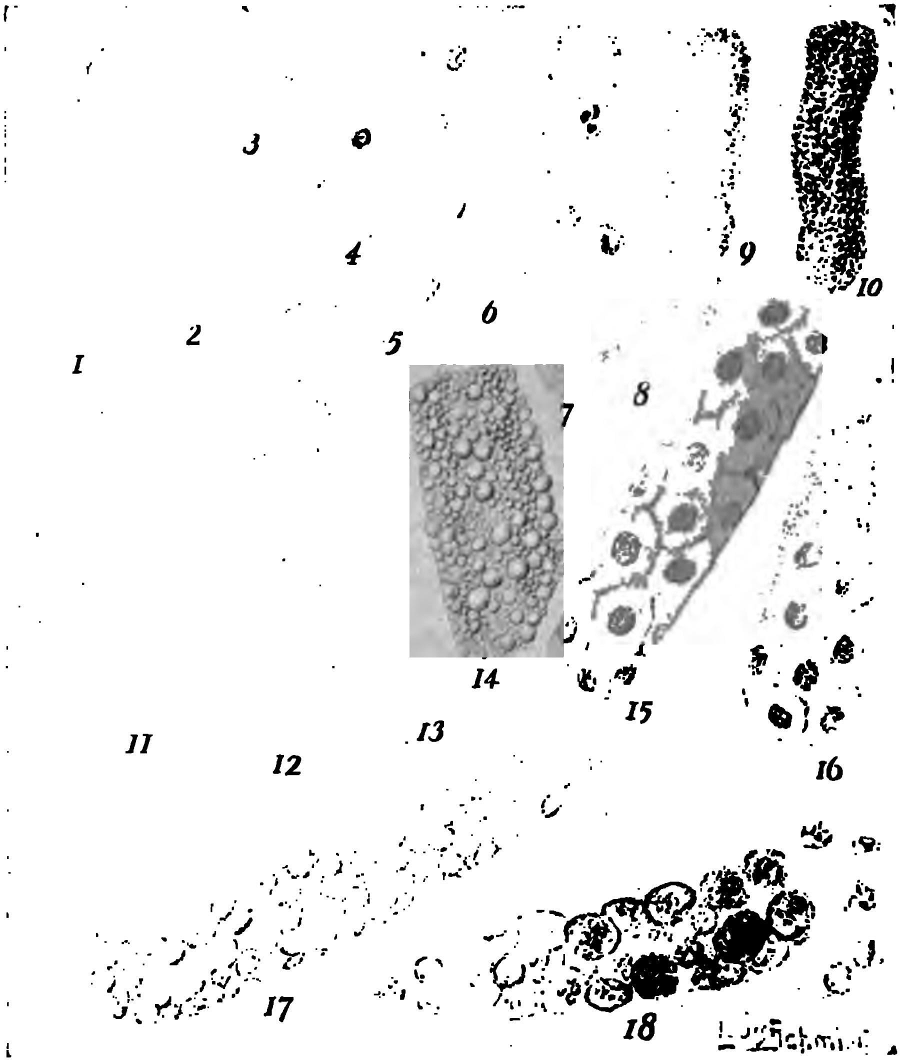


FIG. 117.—Tube-casts 1, 2, 3, 5, hyaline casts; 4, 6, hyaline casts beset with epithelial cells; 7, hyaline cast—one end of which is coated with fine granules; 8, hyaline cast beset with leucocytes; 9, finely granular cast; 10, coarsely granular cast; 11, 12, 13, waxy casts; 14, fatty cast; 15, 16, epithelial casts; 17, blood-cast; 18, leucocytic cast.

BLOOD-CASTS consist of erythrocytes, many of which may be altered by crenation or dehæmoglobinization. Pus- and blood-casts are rarely encountered. Casts formed of hæmoglobin, of bacteria, or of urates are rare.

CYLINDROIDS are of two forms; one variety appears as long twisted or curved ribbon-like structures, composed of mucus, and therefore insoluble in acetic acid. This cylindroid is readily distinguished from true hyaline casts, because of its length and flattened appearance. A second group consists of elongated cylindrical bodies. They show considerable variation in their short diameter, and are composed of a hyaline material. This cylindroid often tapers into a long thread-like tail. Some of the latter variety closely resemble hyaline casts, but can generally be distinguished from glassy casts by their irregular diameter. The material composing the latter type is soluble in acetic acid. Some authorities contend that this form of cylindroid has the same clinical significance as the hyaline cast. The mucous cylindroids are in the main formed in the bladder.

Clinical Significance of Tube-casts.—Tube-casts, especially of the hyaline variety, are often found in the urine of morbid states and occasionally of apparently healthy persons. Some observers maintain that their presence in the urine of so-called healthy persons can be explained by temporary circulatory disturbances, such as result from violent physical exercise or from overstimulation, as with alcohol. These circulatory derangements, although of a temporary nature, cannot be regarded as strictly physiological; therefore, the presence of casts under such circumstances reflects an abnormality of the renal function. The finding of casts over a long period generally warrants a diagnosis of structural changes in the kidneys. The number of casts present in a specimen of urine is sometimes an index of the extent or severity of renal involvement. In acute diffuse nephritis their number is generally large, in chronic parenchymatous nephritis they are usually fairly abundant, while in the interstitial form only a small number is noted. In passive renal congestion, amyloid disease, and in the degenerations attending febrile diseases, casts are generally few in number, but occasionally plentiful. The size of casts varies considerably. Large cylinders at times exceed 1 mm. in length. The size of tube-casts has no special diagnostic significance. The predominance of one or several varieties of casts may be of value in deciding the character of a renal lesion. Hyaline casts do not signify any special morbid change, as they occur under a variety of circumstances. Often

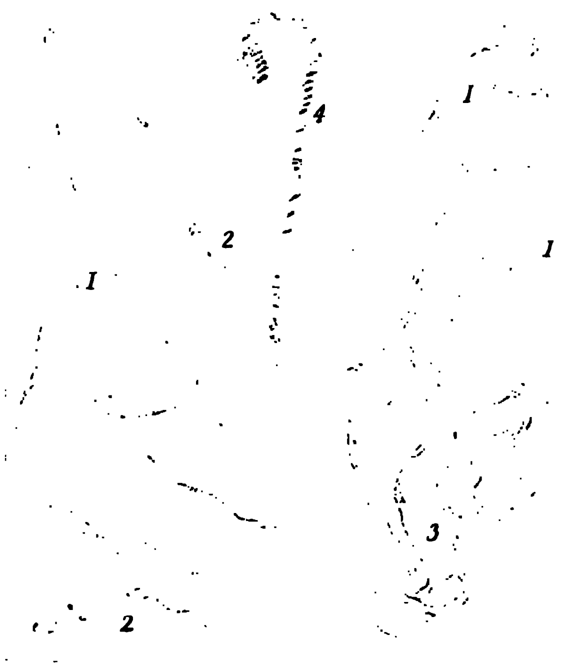


FIG. 118.—Cylindroids. 1, Cylindroids resembling hyaline tube-casts; 2, cylindroids stippled with granules; 3, ribbon-like mucous cylindroids; 4, spiral form of cylindroid.—Modified from Emerson.

they appear when there is only a slight functional derangement of the kidneys, but they are invariably present in organic renal disease. Waxy, granular, epithelial, and fatty casts point to degeneration of the renal parenchyma, while pus-casts may indicate purulent kidney disease. Blood-casts signify hemorrhage.

SPERMATOZOA AND TESTICULAR CASTS. — Spermatozoa are found in the urine after coitus, pollution, and rarely after convulsions. The urine which contains spermatozoa occasionally reveals testicular casts. These casts closely resemble renal casts. They can, however, be distinguished from the latter, since they occur only in the first part of the urine voided while renal casts are present in the entire specimen. Their recognition depends mainly upon the finding of spermatozoa with these casts in the first urine of a two-glass test, and an absence of both of these elements in the second specimen of urine.

Irregular shreds and ribbon-like threads (Tripperfäden) are seen without magnification in the urine after acute gonorrhœa and in chronic urethritis. They consist of shreds of coagulated mucus, to which are generally adherent leucocytes and epithelial cells.

Bacteria; Animal Parasites.—BACTERIA.—The presence of bacteria in abnormal urine depends upon, (1) contamination of urine after it is voided, (2) existence of infectious lesions of the genito-urinary tract or communicating with it, and (3) elimination of bacteria from the blood by the kidneys. In large numbers bacteria impart turbidity to the urine, which does not clear up completely by centrifugating or by passing the urine through filter-paper.

The Micrococcus ureæ is considered responsible for ammoniacal fermentation. The colon bacillus, tubercle bacillus, typhoid and paratyphoid bacillus, plague bacillus, ray fungus, sarcinæ and moulds are the commoner micro-organisms found in pathological urine. The typhoid bacillus, which is eliminated by the kidney in every case of enteric fever, is occasionally the exciting factor of inflammatory disease of the bladder and renal pelvis. Tubercle bacilli in the urine may indicate a tuberculous focus in the urinary tract. Their elimination by the kidneys from the blood in the absence of genito-urinary tuberculosis has been suggested. The smegma bacillus, which exists in the secretions of the external genitals, is at times washed into the urine. This contamination can be avoided by carefully cleansing the external genitals, or by securing the urine with a catheter. Pappenheim's stain is highly recommended as means of differentiating the tubercle from the smegma bacillus. Yeast cells are often found in diabetic urine and may give rise to pneumaturia.

ANIMAL PARASITES.—The Trichomonas vaginalis is rarely noted and its presence is probably dependent upon contamination of the urine with a vulvovaginal discharge containing this parasite. Ova of the Distoma hæmatobium are sometimes seen in the urine when the adult worm resides in the mucous membrane of the renal passages. Distomiasis is essentially a tropical disease which is occasionally responsible for hæmaturia. Filarial embryos have been found in certain cases of tropical hæmaturia. Echinococcus hooklets or fragments of cysts may be present in cases of hydatid disease of the urinary system. There are a few instances on record in which the Eustrongylus gigas was noted in the urine.

Calculi.—Urinary calculi of renal and vesical origin vary as to size and outline. Stones small enough to pass through the urinary passages are more common than the large calculi found in the renal pelvis or bladder.

Uric acid stones vary in size from that of a grain of sand to concretions large enough to fill up the renal pelvis. These calculi are reddish-brown or dark gray, very dense, have a smooth or slightly roughened surface, dissolve in alkalies, and, when treated with sodium hydrate, generate ammonia. They give murexide test. Sometimes calcium oxalate is present n uric acid concretions. Ammonium urate stones have a waxy consistence, give the murexide test and liberate ammonia when treated with sodium hydrate. These calculi are rare. At times they are found in adults, and are occasionally discovered in the new-born. Calcium oxalate stones are responsible for severe attacks of renal colic and hæmaturia. These calculi are very hard, their surface is generally irregular, often showing sharp projections, and their color is dark gray or black. Hydrochloric acid dissolves them, and acetic acid will also cause solution when added to the powdered stone. Phosphate stones have a soft texture, are white or pale yellow, and have a rough surface. They are soluble in acetic acid without gas formation. They are formed in the bladder much more frequently than in the renal pelvis. Cystin stones have a wax-like consistency, are white or yellowish in color, dissolve in ammonia, and give the reaction for cystin. They are of rare occurrence. Xanthin stones are hard, of a white or yellowish-brown color, and dissolve in ammonia. Indigo stones have a blue or bluish-gray color. Xanthin and indigo calculi are extremely rare. Calcium carbonate stones are white, have a chalk-like consistency, and are soluble in acetic acid with gas formation. Stones consisting of fatty acids and cholesterin have been recorded in a few instances.

Tumor fragments from carcinoma or sarcoma of the urinary tract are rarely present in the urine. Fecal matter has been found in the urine in cases of enterovesical fistula.

CHEMICAL EXAMINATION.

Nitrogenous Bodies.—The normal amount of nitrogen eliminated by he kidneys per day varies between 10 and 16 grammes. It may be educed to 5 or 6 grammes on a vegetable diet. Nitrogen, the best index of proteid metabolism, is principally eliminated in the form of urea, and o some extent as ammonia, uric acid, and extractives. Hammarsten's estimation of the percentage of nitrogen excreted in the principal nitrogenous bodies is as follows:

	Adults, per cent.	Infants, per cent.
Urea	84 to 91	73 to 76
NH$_3$	2 to 5	7.8 to 9.6
Uric acid	1 to 3	3 to 8.5
Extractives	7 to 12	7.3 to 14.7

Nitrogen is increased by a rich proteid diet, active exercise, in fevers, in cachexia, in diabetes, in poisoning by arsenic, antimony, phosphorus, and certain organic poisons, after hemorrhage, in dyspepsia, during the resolution stage of pneumonia, and from the absorption of exudates and transudates. It is lowered from lack of exercise, by a vegetable diet or one containing much carbohydrate, during the convalescence of fevers, in persons gaining weight rapidly, during pregnancy, during the formation of exudates and transudates, and in nephritis.

Urea.—QUANTITATIVE ESTIMATION; HYPOBROMITE METHOD.—This quantitative test is based upon the principle that an alkaline solution of hypobromite of soda will decompose urea into nitrogen, and carbon dioxide, which is absorbed in the excess of alkali. The amount of urea is estimated by the volume of nitrogen set free. Hüfner has shown that one cubic centimetre of nitrogen (at 0° C. and 760 mm. pressure) represents .00268 gramme of urea. A convenient method (Rice) of preparing the hypobromite reagent is as follows: (1) A solution is made by dissolving 100 grammes of NaOH in 250 c.c. of water; (2) a solution of bromine one part, potassium bromide one part, and water eight parts. These solutions are mixed in equal amounts. Special forms of apparatus have been devised for collecting the nitrogen and measuring its volume. The Heintz modification of the Doremus apparatus can be highly recommended because it is easy to operate and is sufficiently accurate for clinical purposes. With this apparatus the test is conducted as follows: The large tube is filled with hypobromite reagent and the small tube with urine up to the point indicated by the mark 1. By opening the stop-cock, one cubic centimetre of urine is allowed to flow very slowly into the large tube. The reaction occurs immediately and nitrogen gas is set free and collects in the upper part of the tube by displacing the fluid. The apparatus is then set aside for fifteen minutes, when the reading is taken. The amount of urea for one cubic centimetre of urine is indicated by a graduated scale at the upper level of the fluid. Albumin should always be removed before making this test. A considerable error will occur when the urine is rich in ammonia. There are other methods for estimating urea which give more accurate results, but they are objectionable because they are tedious and entirely too complicated for the general practitioner.

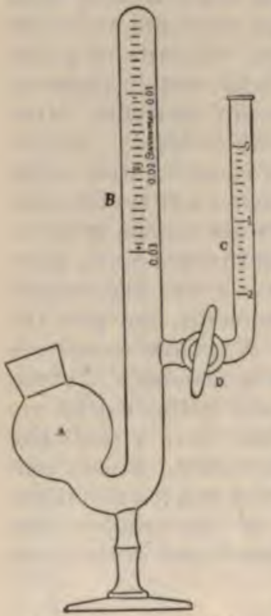


FIG. 119.—Heintz modification of Hüfner apparatus for urea determination. *A*, bulb; *B*, graduated tube to collect and measure the nitrogen; *C*, tube for urine; *D*, stop-cock.

On an ordinary diet the daily amount of urea varies between 20 and 40 grammes, on a rich diet it may reach 100 grammes, while on a restricted diet it is sometimes reduced to 15 grammes. As a rule the quantity of urea and the total nitrogen output are parallel, so that for clinical purposes the amount of urea is generally determined instead of the total nitrogen. Urea may show a reduction with a rise in the ammonia elimination. This is observed in certain forms of liver disorders, although in many cases of hepatic disease the urea output is unaltered.

Uric Acid.—FOLIN'S MODIFICATION OF HOPKINS'S TEST.—Three hundred cubic centimetres of urine are treated with 75 c.c. of a reagent prepared as follows: 500 grammes of ammonium sulphate and 5 grammes of uranium acetate are dissolved in 650 c.c. of water, to which are added 60 c.c. of a 10 per cent. acetic acid solution, and water enough to bring the amount up to 1 litre. After standing for about five minutes the urine so treated is filtered through two thicknesses of filter-paper. Into each of two beakers 125 c.c. of filtrate are poured, treated with 5 c.c. of concentrated ammonia, and set aside for twenty-four hours. The ammonium urate precipitate is next washed with a small quantity of a 10 per cent. solution of ammonium sulphate. The precipitate of ammonium urate collected on filter-paper is washed with 100 c.c. of water into a beaker, after perforating the filter-paper. The solution is finally treated with 15 c.c. of concentrated sulphuric acid and then immediately titrated with a 1/20 normal solution of potassium permanganate, until a faint red color tints the entire solution. This color disappears rapidly. Each cubic centimetre of a 1/20 normal permanganate solution represents .00375 gramme of uric acid.

Uric acid is an oxidation product of the xanthin bases. Its origin depends upon the nucleins derived from the food (exogenous uric acid) and from the body tissues (endogenous uric acid). The normal daily amount of uric acid found in the urine varies between .2 and 1.25 grammes, which represents from 1 to 2 per cent. of the total nitrogen output. Uric acid is increased by a diet rich in nuclear proteids, active muscular exercise, in fevers, in anæmia, in leukæmia, in pneumonia during the stage of resolution, in cirrhosis of the liver, and in diabetes mellitus. In gout the amount of uric acid is generally decreased between the acute attacks, and rises during and immediately after the paroxysm. In gout an increase of uric acid is found in the blood (uratæmia). The circumstances which bring about the separation of uric acid crystals in and about the joints and in other tissues are not definitely known. The mere existence of uratæmia does not justify the conclusion that it is the principal or primary factor of this disease; on the contrary it would appear that an increase of urates which occurs in a number of conditions, as anæmia, leukæmia, and during the resolution stage of pneumonia, does not in itself favor precipitation of biurate of sodium. It has been suggested that an excess of sodium salts in the blood, lymph, and especially in synovial fluid, determines the precipitation of urates. Solutions of uric acid have been shown to possess only slightly toxic or harmless properties when injected into the tissues of animals.

The quantity of uric acid in the urine is decreased on a restricted diet, especially one poor in substances containing nucleins, after the administration of large doses of quinine, in nephritis, and in certain chronic diseases. At the present time a final opinion as to the rôle played by uric acid in the so-called uric acid diathesis cannot be given.

Xanthin Bases.—Under this heading is included a group of substances found in the urine in very small amounts and regarded as being formed from nucleins. In this group may be included xanthin, hypoxanthin, heteroxanthin, paraxanthin, guanin and adenin. In the main it may be said that the amounts of uric acid and the xanthin bases fluctuate in a parallel manner. The xanthin bases are increased in the urine in leukæmia, after a diet rich in nucleins, and in pneumonia. Rarely, calculi consist of xanthin.

Ammonia.—The normal daily output of ammonia is about 0.7 gramme, which represents slightly over four per cent. of the total nitrogen elimination. It exists in combination with some of the urinary acids. Its presence in the urine is accounted for by a small amount of ammonia which is not transformed into urea in the liver. Ammonia is increased in conditions associated with deficient oxidation, as cardiac dyspnœa, in certain diseases of the parenchyma of the liver, such as acute yellow atrophy and phosphorous poisoning, in diabetes mellitus, and, notably, in pernicious vomiting of pregnancy.

Chlorides.—QUANTITATIVE DETERMINATION.—Ten cubic centimetres of urine are diluted with 90 c.c. of water, to which are then added a few drops of a strong potassium chromate solution. A standard silver solution (1 c.c. of which represents .0035 gramme of chlorine, or .0058 gramme of NaCl) is then slowly added from a graduated burette. The development of a permanent orange color indicates that all the chlorine has been precipitated.

The excretion of chlorides, which varies from 10 to 15 grammes per day, depends almost exclusively upon the quantity of chlorides ingested. A decreased elimination is present on a diet poor in chlorides, in the acute fevers (probably due to a deficiency of chlorides in the fever diet), before the crisis in pneumonia, in acute and chronic nephritis, in many chronic diseases, in gastric disorders associated with vomiting, in diseases attended with diarrhœa, and during the formation of transudates and exudates. An augmented elimination is observed after a diet rich in chlorides, after the acute fevers, especially during the stage of resolution of pneumonia, in diabetes insipidus, and from rapid resorption of transudates and exudates.

Phosphates.—Phosphoric acid of the urine is combined with sodium, potassium, ammonium, calcium, and magnesium. The daily amount of phosphoric acid excreted by the kidneys varies between two and three grammes. A diminished excretion has been noted in some febrile diseases, in cases of arthritis, between the paroxysms of gout, in pregnancy, in acute yellow atrophy of the liver, in nephritis, in Addison's disease, and in chronic lead poisoning. An increased elimination has been noted on a diet rich in meat, during the attack of gout, in diabetes mellitus, in neurasthenia, in hysteria, in leukæmia, and after active muscular exercise. The existence of a phosphatic deposit in the urine is not necessarily a sign of increased elimination, and is frequently due to alkalinity of the urine. A quantitative estimation of phosphoric acid is necessary to establish an increased output. Neubauer's method consists in titrating the urine with a uranium nitrate solution, using cochineal as an indicator. For the details of this method special works on urinary chemistry should be consulted.

Sulphates.—Sulphuric acid exists in the urine as mineral, preformed or neutral sulphates, and as conjugate or ethereal sulphates. The total daily output of sulphuric acid varies between 2 and 3 grammes, nine-tenths of which is eliminated as mineral sulphates and the remainder as ethereal sulphates. Ethereal sulphates occur in combination with certain aromatic bodies, the most important of these being phenol, indoxyl, skatoxyl, and cresol. The sulphate elimination is controlled principally by proteid metabolism, so that the amount is increased after a diet rich in meat, by muscular exercise, in the acute febrile diseases, in acute inflammatory diseases of the brain and spinal cord, and by certain poisons which augment proteid destruction. The output of sulphates is reduced by a vegetable diet or one poor in proteids, during the period of convalescence from the acute fevers, and in many chronic diseases. The quantity of ethereal sulphates depends mainly upon putrefactive changes occurring in the intestinal tract, and sometimes in other parts of the body. The normal proportion of ethereal sulphate to neutral sulphate varies considerably. The conjugate sulphates are diminished by starvation, by the administration of calomel and hydrochloric acid, and are increased by the ingestion of alkalies and carbolic acid, in intestinal diseases associated with increased putrefaction, as in constipation, enteric fever, and tuberculous enteritis.

Indican.—OBERMAYER'S TEST.—The reagent for this method is made by dissolving two parts of ferric chloride in 1000 parts of concentrated hydrochloric acid. A small amount of urine is treated with an equal part of Obermayer's reagent and the mixture shaken with 2 or 3 cubic centimetres of chloroform, which extracts indican. It is light blue or colorless when a normal amount is present, while an increased quantity is shown by a dark blue color.

JAFFE'S TEST MODIFIED BY STOKVIS.—Equal volumes of hydrochloric acid and urine are mixed. The liquid is treated with a droplet of a concentrated solution of sodium or calcium hypochlorite and then shaken with a few c.c. of chloroform. A blue color is imparted to the chloroform by the indigo. An approximate estimate of the amount may be formed by the depth of this color. Iodine in the urine tints the chloroform pink. Bile pigment should always be removed with lead subacetate before testing for indican. Indol is formed in the intestines as a result of putrefactive processes; in the blood it is oxidized and combines with sulphuric acid, being eliminated as sodium or potassium indoxyl sulphate or indican. As putrefaction is essential for the formation of indican, only small traces of this substance occur in the urine of healthy persons, since intestinal decomposition is slight under normal conditions. The quantity of indican is influenced by the character of food, being smaller upon a milk than on a full mixed diet. Jaffe found that 6.6 mg. was the average normal amount for 1000 c.c. of urine.

Pathological indicanuria occurs in carcinoma of the stomach, in certain forms of gastritis, and in conditions associated with inhibited intestinal peristalsis, as constipation, intestinal obstruction, and peritonitis. The amount of indican is augmented in putrid bronchitis, in empyema, and in gangrene and abscess of the lungs.

Urinary Pigments.—The color of normal urine depends chiefly upon urochrome. The following pigments are responsible for the color of many abnormal urines: pathological urobilin, uroerythrin, hæmoglobin, methæmoglobin, urohæmatin, uroroseinogen, biliary pigment, and melanin. After the ingestion of senna, santonin, iodine, phenol, and creosote abnormal pigmentation of the urine often occurs.

Biliary Pigments.—ROSENBACH'S MODIFICATION OF GMELIN'S METHOD.—The urine is filtered through thick filter-paper. A drop of concentrated nitric acid is then placed upon the urine-soaked filter-paper. A play of colors, consisting of red, yellow, green, blue, and violet, in which the green predominates, will develop in the presence of biliary pigment.

SMITH'S TEST.—A small amount of tincture of iodine diluted with 10 parts of alcohol is added to 5 or 10 c.c. of urine, so that the iodine solution forms a layer above the urine. An emerald color forms at the zone of contact of two fluids when bilirubin is present. Biliary acids are associated with bilirubin so that their clinical significance is practically the same. The tests for biliary acids are attended with considerable difficulty.

The biliary pigments are bilirubin, biliverdin, bilifuscin, and biliprasin. Bilirubin is found in freshly voided urine only, while the other pigments may appear after the urine has stood for a time. Biliary pigment occurs in the urine in both toxæmic and obstructive jaundice.

Phenol.—SALKOWSKI'S TEST.—About 10 c.c. of urine are treated with a few c.c. of nitric acid and boiled. On cooling, bromine water is added. An increased amount of phenol is shown by the development of a decided cloudiness or precipitate.

The amount of phenol eliminated is very small (.03 gramme daily under normal conditions). This substance is increased whenever putrefactive processes occur in the body, as in gangrene, putrid bronchitis, empyema, and, rarely, from intestinal decomposition. It has also been demonstrated in tuberculosis, meningitis, peritonitis, erysipelas, scarlet fever, and from poisoning with phenol or some of its derivatives, such as salicylic acid, pyrocatechin and hydroquinone. The urine containing phenol may become dark brown or black on standing.

Pathological Urobilin.—BRAUNSTEIN'S TEST.—About 20 c.c. of urine are mixed with 5 c.c. of a reagent which consists of 100 parts of a concentrated solution of cupric sulphate, 6 parts of hydrochloric acid, and 3 parts of ferric chloride. A small amount of chloroform is added to the mixture. On shaking, the chloroform becomes rose colored.

This pigment is closely related to urochrome and can be differentiated from the latter by the spectroscope. Urobilin and its chromogen are soluble in chloroform and precipitated with ammonium sulphate. Pathological urobilin is sometimes encountered in the urine in febrile diseases, cirrhosis of the liver, pernicious anæmia, cancer, cerebral hemorrhage, scurvy, Addison's disease, hæmophilia, and syphilis.

Melanin and Melanogen.—These substances are occasionally found in the urine of persons suffering from melanotic tumors, chronic malaria, and certain wasting diseases. The urine containing melanin and melanogen may have a normal yellow color when voided, but becomes darker when exposed to the air.

Albumins.—The proteids found in the urine are serum albumin, serum globulin, nucleo-albumin, albumose, Bence-Jones's albumin, hæmoglobin, fibrin and histon. The most important of these from a clinical standpoint is serum albumin.

Serum Albumin.—The most useful tests for the detection of albumin are the boiling and acidulation tests and Heller's test, because they afford uniformly satisfactory results, are simple and easily applied. It is claimed that these tests are less sensitive than many others, such as Speigler's and Tanret's. Before testing for albumin the urine should be clear, and, if cloudy, must be filtered through several layers of filter-paper. Bacteria cannot be completely removed by filtration through ordinary filter-paper. It is desirable to have a fresh specimen for testing. In certain cases several samples should be secured, *i.e.*, the first urine passed in the morning on arising, and that voided late in the afternoon. Albumin reactions are sometimes less distinct in concentrated specimens than in those of low specific gravity, and it is, therefore, advisable to dilute an inspissated urine before applying albumin tests.

BOILING AND ACIDULATION TEST.—Clear urine is boiled in a test-tube. When a precipitate forms this is generally due to either phosphates or albumin (serum albumin in conjunction with serum globulin). The turbidity caused by phosphates clears on the addition of a few drops of colorless nitric acid, while the cloud due to albumin remains or even is intensified after acidulation. A precipitate of carbonates, developing on heating, will disappear upon the addition of nitric acid with the liberation of gas (CO_2). If on boiling the urine remains clear but subsequently on cooling a cloud develops, this is due to albumose. This turbidity will again disappear on heating. Certain resinous bodies, as copaiba, benzoin, cubebs, and turpentine, also produce a precipitate on heating. This cloud can be distinguished from that produced by albumin by the fact that alcohol dissolves the turbidity produced by these substances. When employing acetic acid, it is best to add a few drops before boiling, care being taken to avoid an excess, since albumin may not precipitate on boiling. If a cloud forms after the urine is treated with acetic acid, this is caused by nucleo-albumin and should be removed by filtration before testing for serum albumin. The most accurate results are obtained with this method when a dilute acetic acid solution is employed (2.5 per cent.).

HELLER'S TEST.—Colorless nitric acid is allowed to flow slowly from a pipette into a test-tube or a conical glass vessel containing a small quantity of urine, so that the urine forms a distinct layer above the acid. In order to prevent mixing the acid and urine, the test-tube or conical vessel should be inclined while adding the nitric acid. When serum albumin is present a white disk appears at the zone of contact between the urine and acid. When a small amount of albumin exists the precipitate does not form immediately but in the course of several minutes. An approximate quantitative estimate of albumin can be formed from the thickness of the coagulated layer. A pale red or reddish-violet disk, at or above the plane of contact, is noted in many normal and abnormal urines. A white precipitate is also caused by serum globulin and albumose. The latter disappears on heating and reappears on cooling. Nucleo-albumin in large amounts may give a positive reaction, but this is so uncommon that it can be disregarded for practical purposes. Certain resinous bodies, indicated in the discussion of the boiling and acidulation test, produce a white cloud which disappears when treated with alcohol.

ACETIC ACID AND POTASSIUM FERROCYANIDE TEST.—A few drops of 10 per cent. solution of potassium ferrocyanide or platinocyanide are added to a small amount of urine previously acidified with acetic acid. A precipitate indicates albumin or albumose. If, on heating, the turbidity disappears completely, the presence of the latter substance is indicated, or, if the cloud partly clears on warming, the presence of both substances may be inferred. When a precipitate, due to nucleo-albumin, forms on addition of acetic acid, the urine should be filtered and the test repeated.

SPIEGLER'S TEST.—The test solution as modified by Jolles consists of mercuric chloride 10 grammes, succinic acid 20 grammes, sodium chloride 20 grammes, and distilled water 500 c.c. The reagent is added slowly by means of a pipette to a small amount of urine contained in a test-tube, so that the urine forms a layer above the test solution. A white cloud at the junction of the fluids indicates albumin, nucleo-albumin or albumose. When the urine contains iodine, a precipitate of mercuric iodide forms, which is soluble in alcohol. This test is very sensitive.

Many other methods for the detection of albumin are recommended by different authorities, as tests with picric acid, metaphosphoric acid, phosphotungstic acid, and trichloracetic acid.

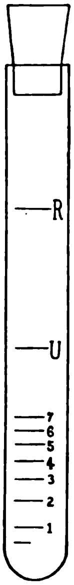


FIG. 120.—Esbach's albuminometer. — Emerson.

QUANTITATIVE DETERMINATION OF ALBUMIN. ESBACH'S METHOD. — The test solution is prepared by dissolving 10 grammes of picric acid and 20 grammes of citric acid in 1000 c.c. of distilled water. A special graduated test-tube devised by Esbach and known as an albuminometer is required for this method. The urine should have an acid reaction. It is poured into the albuminometer to the mark "U"; the reagent is then added until the fluid reaches to the mark "R." The fluids are then mixed and the test-tube set aside for twenty-four hours, when the reading is taken. The height of the column of coagulated albumin, as measured by the scale on the tube, represents the amount pro mille. Esbach's reagent precipitates serum albumin, serum globulin, albumose, uric acid, and creatinin. When the specific gravity exceeds 1.008, or when a large amount of albumin exists, the urine should be diluted with one or several volumes of water before applying the test. The reading is multiplied by the number of dilutions. Esbach's method, although not so accurate as the gravimetric determination, is quite satisfactory for general clinical purposes.

BOILING TEST.—An approximate estimate of the quantity of albumin can be formed by boiling acidified urine in a test-tube and allowing the precipitate to settle for twenty-four hours. The error with this method may be considerable, because albumin sometimes separates in large and at other times in small flakes.

GRAVIMETRIC METHOD.—One hundred cubic centimetres of urine are sufficiently acidulated with acetic acid to insure separation of all the albumin. It is then boiled and passed through a filter of known weight. The precipitate collected on the filter is washed with hot water until the washings cease to give a reaction for chlorides. The precipitate is next washed successively with alcohol and ether to remove fat. The filter containing the precipitate is now dried at a temperature of 120° to 130° and then carefully weighed. The weight of the albumin is obtained by subtracting the weight of the filter-paper from the combined weight of the filter-paper and dried precipitate.

Albuminuria. — The term albuminuria implies the presence in the urine of coagulable albumin, and refers particularly to serum albumin. One or more albuminous bodies are almost invariably associated with serum albumin. Albuminuria is symptomatic of a large number of morbid states, from minor disturbances in health to malignant diseases.

1. RENAL ALBUMINURIA.—When albumin is eliminated by the kidneys the condition is termed renal albuminuria.

(a) *So-called physiological albuminuria* is occasionally noted in healthy individuals after violent exercise or severe nervous stress. Whether albuminuria is ever physiological is still a mooted question. Albuminuria often occurs in pregnancy, especially in the later stages. The so-called albuminuria of adolescents is probably pathological.

(b) *Albuminuria of Organic Kidney Disease.* — In this variety the presence of albumin in the urine depends directly on structural changes in the renal tissues, and in nephritis, and amyloid, tuberculous, malignant and cystic disease of the kidney. In acute and chronic parenchymatous nephritis the amount is generally large, while in amyloid disease it is moderate or small, and in contracted kidney it is small. The mere presence of albumin in the urine never warrants a diagnosis of organic renal disease; on the other hand mere traces occur in granular kidney, and, indeed, albumin may be absent for a time in this disease. Large quantities of albumin usually justify a diagnosis of organic kidney disease.

(c) *Febrile Albuminuria.*—A discharge of albumin of slight or moderate degree in fevers and inflammatory diseases is suggestive of a simple parenchymatous degeneration of the kidney and of vascular derangements, incident to the febrile or inflammatory process, while a high grade of albuminuria, noted in a limited number of these cases, points to marked renal degeneration, often associated with decided congestion. The difference between albuminuria of febrile and inflammatory disorders and that of acute Bright's disease is essentially one of degree, so that a sharp distinction cannot be made between these forms. Albuminuria is symptomatic of many of the infectious diseases, especially enteric fever, typhus fever, pneumonia, cerebrospinal fever, yellow fever, plague, cholera, malignant endocarditis, diphtheria, erysipelas, and variola.

(d) *Toxic Albuminuria.*—Under this heading is included the albuminuria produced by drugs, such as salicylic acid, potassium iodide, salol, urotropine, phenol, alcohol, ether, chloroform, lead, mercury, phosphorus, and a number of other toxic substances.

(e) *Albuminuria occurring in blood disorders* is seen in severe secondary anæmias, pernicious anæmia, chlorosis, and leukæmia.

(f) *Alimentary Albuminuria.*—The ingestion of very large amounts of albumin, such as raw eggs, may excite albuminuria, but a moderate quantity of albuminous food will never produce albuminuria in a healthy person. An antecedent chronic albuminuria may be intensified by a moderate consumption of albumin.

(g) *Albuminuria dependent upon circulatory disturbances of the kidneys* is seen in cardiac disease, especially during the stage of ruptured compensation, in pulmonary disease with venous stasis, from pressure on the renal veins by a tumor, cyst or peritoneal effusion, and by a thrombus in these vessels. In floating kidney albuminuria sometimes depends on kinking of the renal veins so that it may be present only while the individual is in the erect posture, disappearing when in the recumbent position (orthostatic albuminuria).

(h) *Albuminuria in nervous diseases* is common when organic lesions of the nervous system exist, such as apoplexy, brain tumor, and spinal sclerosis, but it is infrequent in functional disorders, such as neurasthenia and migraine.

(i) *Albuminuria caused by obstruction in the urinary passages* occurs in nephrolithiasis, when the stone blocks up the ureter for a time, and also when the ureter is compressed by a tumor or is twisted. The urine which has been impeded in its passage shows albumin in many instances.

2. ACCIDENTAL ALBUMINURIA.—When the urine contains albumin derived from the renal passages or genital organs it is designated accidental or extrarenal albuminuria. The presence of pus, blood, leucorrhœal discharge, and chyle in the urine, as a rule, causes a slight, and rarely, a moderate albumin reaction. This type occurs in pyelitis, ureteritis, cystitis, prostatitis, vesiculitis, epididymitis, urethritis, vulvovaginitis, and during menstruation. A vaginal discharge is often washed into the urine. The diagnosis of accidental albuminuria is generally unattended with difficulty, provided the results of microscopic examination and the clinical investigation are carefully considered. In general terms it may be said that the intensity of the albumin reaction is directly proportionate to the amount of cellular deposit. The differentiation between renal and extrarenal albuminuria rests on the data obtained by a careful urinalysis with other clinical findings. Both conditions often coexist. The presence of tube-casts and many pus-cells with an albumin reaction greater than the number of leucocytes would indicate, argues in favor of a coexistent renal and accidental albuminuria.

Serum Globulin.—KAUDER'S TEST.—The urine is treated with a sufficient quantity of ammonia to separate the phosphates, which are removed by filtration. An equal bulk of a saturated solution of ammonium sulphate and filtrate are mixed. A precipitate represents serum globulin.

Serum globulin and serum albumin are almost invariably associated, so that their clinical significance is similar. As a rule serum albumin is found in excess of serum globulin, although exceptions to this rule are recorded in amyloid disease, diabetes, and severe nephritis.

Nucleo-albumin. — This body is precipitated by strong acetic acid. Concentrated urines should always be diluted with two or three volumes of water before applying this test. Urine containing much serum albumin and serum globulin should be boiled and filtered in order to remove these substances before testing for nucleo-albumin.

OTT'S METHOD.—Add to the urine an equal volume of saturated solution of sodium chloride, and treat the mixture with Almen's tannin solution. The presence of nucleo-albumin is shown by the formation of an abundant precipitate. Almen's solution consists of 5 grammes of tannic acid, 10 c.c. of a 25 per cent. solution of acetic acid, and 240 c.c. of 50 per cent. ethyl alcohol. Nucleo-albumin can be removed from the urine with neutral lead acetate.

With certain delicate tests nucleo-albumin can be demonstrated in many normal and abnormal specimens, so that its presence in small amount may be regarded as physiological. When nucleo-albumin can be detected by tests generally employed in routine clinical work, it is probably pathological. Nucleo-albuminuria occurs in inflammatory diseases, especially of a catarrhal nature, of the urinary tract, as cystitis and pyelitis. In febrile diseases associated with albuminuria, in leukæmia, in jaundice, and in acute nephritis, nucleo-albuminuria is not uncommon. In the last named disease nucleo-albuminuria sometimes precedes and follows serum albuminuria.

Albumose.—To the urine strongly acidulated with acetic acid, is added an equal amount of a saturated solution of sodium chloride. The presence of a precipitate, which disappears on boiling and returns on cooling the urine, consists of albumose. When serum albumin coexists with albumose, this must be removed by boiling and filtering before applying the test.

Albumosuria is referred to by some writers as peptonuria, a term which Kühne restricts to the presence of true peptone. According to Kühne, peptonuria has been found in pneumonia, phthisis, and gastric ulcer. The chief clinical significance of albumose in the urine relates to morbid lesions, characterized by a destruction of leucocytes, with the absorption of the disintegrated products. In many diseases showing these pathological features, especially in purulent collections, the occurrence of albumosuria may be a useful sign in diagnosis. In this connection it must be pointed out that, since the group of conditions in which it occurs is a vast one, its significance is of less value in diagnosis than any other urinary findings. Albumosuria has been noted in pneumonia during the period of resolution, in suppurative meningitis, in liver abscess, in septicæmia, in leukæmia, in endocarditis, in myxœdema, in diphtheria, in measles, in rheumatic fever, in scarlet fever, in acute yellow atrophy of the liver, in scurvy, in dermatitis, and in intestinal diseases characterized by ulceration, as enteric fever, tuberculosis, and carcinoma. Albumosuria may be associated with, or occur independently of, serum albuminuria.

Bence-Jones's Albumose.—The recognition of this proteid depends upon the fact that its precipitation occurs at a temperature of 52° to 60° C. Upon boiling, the cloud entirely or partially disappears, to return again on cooling. With Heller's nitric acid test Bence-Jones's albumin gives a reaction like that of serum albumin.

This proteid, first described by Bence-Jones, occurs with considerable frequency in myelomatous growths in the bones. It is generally designated as albumose, but probably incorrectly. The researches of Simon and Magnus Levy indicate that it is a true albumin.

Hæmoglobin.—The spectroscopic examination, as a rule, shows absorption bands of methæmoglobin, sometimes of oxyhæmoglobin.

DONOGANY'S TEST.—If, on the addition of 1 c.c. of ammonium sulphide solution and an equal quantity of pyridine to 10 c.c. of urine, an orange color develops, the presence of blood may be inferred. When the result is doubtful, a spectroscopic examination should be made of the mixture.

The physiological destruction of red corpuscles is not followed by hæmoglobinuria, because the coloring matter set free from the disintegrated erythrocytes is converted wholly, or in part, in the liver into bile, and, perhaps, a fraction of the amount is redeposited in the tissues and stored there for the future demands of the system. The explanation generally offered to elucidate hæmoglobinuria is based upon an erythrocytolysis so excessive that a part of the hæmoglobin liberated into the plasma (hæmoglobinæmia) is secreted by the kidneys. Hæmoglobinuria occurs in some cases of malarial fever (black water fever). It has been observed in yellow fever, variola, icterus gravis, scarlet fever, enteric fever, syphilis, Raynaud's disease, and from the toxic action of phenol, potassium chlorate, snake venom, hydrogen sulphide, carbon monoxide, and after exposure to the cold. The etiological factor responsible for paroxysmal hæmoglobinuria has not been definitely determined. Some writers claim that exposure to cold is the exciting cause, while others hold that it is of nervous origin. Hæmaturia is much more common than hæmoglobinuria.

Fibrin.—The suspected fibrin clots are separated from the urine by filtration, then thoroughly washed with water and dissolved by boiling in a 5 per cent. solution of hydrochloric acid. The solution thus secured gives the test for serum albumin when the coagulum consists of fibrin.

Fibrinuria has been noted in hæmaturia, chyluria, and in pseudomembranous inflammation of the urinary tract.

An acetosoluble albumin referred to by Simon as Patein's albumin has been reported in cystic kidney and nephritis.

Glucose.—Fresh urine is desirable for quantitative examinations for sugar. When albumin is present, this should be removed from the urine before testing for glucose.

TROMMER'S TEST.—To a small amount of urine rendered strongly alkaline with a solution of sodium hydrate, is added drop by drop a 10 per cent. solution of cupric sulphate, until the cupric oxide which forms ceases to be dissolved. On heating the urine, treated in this manner, a yellow or red precipitate develops when sugar is present. Small traces of sugar often give negative results unless the urine is boiled. Cupric oxide is often reduced by other substances. This may occur after the ingestion of benzoic acid, chloral, salicylic acid, sulphonal, chloroform, and from the presence in the urine of uric acid, creatinin, creatin, bile pigment, and hydroquinone. Glucose causes precipitation of cupric oxide at a temperature below the boiling point, which affords a means of distinguishing it from other reducing substances.

FEHLING'S TEST.—This method is a modification of Trommer's test. Two solutions are required, an alkaline and a copper solution, which should be mixed just before applying the test. Fehling's reagent deteriorates in a few days to such an extent that it is unsuited for testing; therefore, it is necessary to keep the alkaline and copper solutions in separate bottles supplied with well-fitting rubber corks. The alkaline solution consists of potassium and sodium tartrate 173 grammes, potassium hydrate 60 grammes, and 500 c.c. of distilled water. The copper solution consists of cupric sulphate 34.64 grammes, dissolved in 500 c.c. of distilled water. Equal volumes of these solutions are poured into a test-tube and shaken; the mixture is then diluted with four parts of water and boiled. After removing the test-tube from the flame the urine is added in small amounts, and after each addition the mixture heated but not boiled. When sugar is present a yellow or red precipitate of cupric suboxide separates. A change of the blue color of Fehling's solution to green, with a slight turbidity of the liquid after the addition of the urine, is very often seen, and may not be caused by glucose. Nearly every reducing substance except sugar requires boiling to produce precipitation of cupric suboxide.

PHENYLHYDRAZINE TEST.—About .5 gramme of phenylhydrazine hydrochloride and 1 gramme of sodium acetate are added to about 8 c.c. of urine contained in a test-tube. If the salts do not dissolve on warming the urine, water is added to effect solution. The tube is now placed in boiling water for 20 or 30 minutes, then removed, and rapidly cooled by placing the test-tube in cold water. The formation of a bright yellow precipitate indicates the presence of sugar. Mere traces of glucose cause a small amount of precipitate which should be examined microscopically for phenylglucosazone crystals. These consist of yellow needles arranged singly or in clusters. Their melting point is 205° C. In experienced hands this test is generally considered the most sensitive.

NYLANDER'S MODIFICATION OF BOETTGER'S TEST.—Almen's reagent, required for this method, consists of 4 grammes of potassium and sodium tartrate, 2 grammes of bismuth subnitrate, and 10 grammes of sodium hydrate dissolved in 90 c.c. of water. This solution is then boiled and, after cooling, it is filtered. A small quantity of Almen's reagent is added to the urine, approximately in the proportion of 1 to 11, and the resultant mixture is boiled. In the presence of sugar a dark gray or black precipitate of metallic bismuth separates. A positive reaction may be given by albumin, melanin, melanogen, and other reducing substances found in the urine after the ingestion of salol, benzol, sulphonal, trional, turpentine, quinine, rhubarb, and senna.

FERMENTATION TEST.—The principle of this method is based on the fact that glucose is decomposed by yeast into alcohol and carbon dioxide. Special fermentation tubes, as designed by Einhorn, are convenient in conducting this test. The method is carried out by mixing a bit of a cake of compressed yeast with urine in a test-tube. Einhorn's fermentation tube is filled with this mixture, care being taken to exclude air bubbles from the top of the tube. The saccharometer is kept at a temperature of from 25° to 38° for twenty-four hours, during which time the CO_2 collects in the upper part of the tube. A temperature of 34° C. gives the most satisfactory results. A control test should always be made with normal urine, since slight fermentation occurs in every specimen. With Einhorn's tube, an approximate estimate of the quantity of sugar can be formed, but for accurate quantitative analysis Robert's differential method is to be preferred. The fermentation test serves to differentiate fermentable sugar from other reducing substances.

QUANTITATIVE ESTIMATION OF SUGAR. FEHLING'S TITRATION METHOD.—10 c.c. of Fehling's solution diluted with 40 c.c. of water are boiled. At this temperature saccharine urine is added drop by drop from a graduated burette, until the blue color of the test solution disappears, which indicates complete reduction of cupric oxide. The presence of reduced copper held in suspension obscures the color of the solution, so that it is necessary to allow the cuprous oxide granules to settle from time to time in order to detect the tint of the fluid. The cupric oxide contained in 10 c.c. of Fehling's solution is reduced by .05 gramme of glucose.

ROBERT'S DIFFERENTIAL DENSITY METHOD. — For general clinical work, Robert's method is most satisfactory. The principle of this method rests on determining the specific gravity before and after fermentation; each .001 degree of difference in the specific gravity represents .23 per cent. of sugar. The test is carried out by noting the specific gravity of 200 c.c. of urine taken from a mixed 24-hour specimen. A portion of a cake of compressed yeast is mixed with the urine, which is then set aside for 24 or 48 hours. The glucose generally disappears in 24 hours, but, in order to ascertain whether all the sugar has been decomposed, the urine is tested by Fehling's method. After all the sugar has been decomposed, the specific gravity of the fermented urine is taken and the difference between the two readings determined. The small urinometers employed in clinical work are not suited for exact determination, therefore it is convenient to use larger instruments. Accurate estimations can be taken with a set of four or five hydrometers, each of which represents a part of the specific gravity range ordinarily encountered in diabetic urine. For example, hydrometer number 1 indicates the scale from 1.000 to 1.010; number 2 ranges from 1.010 to 1.020; number 3 ranges from 1.020 to 1.030; number 4 ranges from 1.030 to 1.040; number 5 ranges from 1.040 to 1.050. The specific gravity observations should be taken at, or nearly, the same temperature. Evaporation of the urine should be reduced to a minimum during fermentation. The first specific gravity determination is taken before the yeast is added to the urine, and the second reading is made after the fermented urine has been filtered.

The quantitative determination for sugar by the polariscope is recommended highly by many workers. A polariscope designed for this estimation is an expensive instrument. The rapidity with which a determination can be made is one of its chief advantages over other methods.

Physiological Glycosuria.—The presence of traces (.5 pro mille) of glucose in the urine of healthy persons is conceded by most authorities. This quantity cannot, however, be detected by the tests employed in routine work.

Pathological Glycosuria.—This condition may be said to exist when glucose can be recognized by the tests generally in vogue in clinical work. Glycosuria may be transitory, intermittent, or constant. The latter variety is one of the cardinal symptoms of diabetes mellitus.

Glycosuria depends directly on an excess of sugar (above .2 per cent.) in the blood. A possible exception to this rule relates to the glycosuria following the administration of phloridzin. It is thought that this substance produces such alterations in the renal epithelium as to permit of increased glucose elimination. A renal form of diabetes has been suggested. The sugar of the blood is derived principally from the carbohydrates of the food, and in all likelihood some glucose is produced from the albumins of the food. In certain cases of diabetes, characterized by rapid emaciation, body proteids are concerned in its formation. Although many factors involved in the physiology of glucose metabolism remain unexplained, much clinical and experimental evidence supports the view, (1) that sugar metabolism is to a great extent regulated by the nervous system, (2) that the liver is chiefly concerned in converting sugar into glycogen, and also in forming glucose, and (3) that the pancreas secretes a sugar-destroying ferment. A hypothetical conception of pathological glycosuria based on this theory may be said to depend on a failure on the part of the liver to form and store up glycogen, a disturbance which might result from a loss of nervous control or from disease of the hepatic cells; or on an inability on the part of the system to consume sugar, which is ascribed to a disturbance in the function of the pancreas inhibiting or suppressing the secretion of the glycolytic substance. Clinically, glycosuria occurs under a variety of circumstances: Disorders of the nervous system. Temporary or permanent glycosuria is observed in brain tumors, meningitis, injuries to the nervous system, neurasthenia, exophthalmic goitre, and may follow worry, fright, or mental overwork. Diseases of the pancreas. Permanent glycosuria is often associated with sclerosis, and sometimes with atrophy or tumors of the pancreas, while temporary glycosuria is at times symptomatic of acute inflammation of this organ. Hepatic disease, abscess and cirrhosis of the liver may be attended with the temporary or constant presence of sugar in the urine. Toxic agents. The occasional occurrence of glucose in the urine is noted in the infectious diseases, as syphilis, influenza, enteric fever, diphtheria, rheumatic fever, and malaria, and from poisoning by chloral, alcohol, and morphine. The explanation of glycosuria occurring under these circumstances might be found in the development of a disorder of the function of the liver, the pancreas, or the nervous system, produced by these toxic agents. This variety is mainly observed as a transitory form, although occasionally diabetes develops after an acute infectious disease, which suggests permanent morbid processes of the hepatic or pancreatic tissues excited during the acute stage of the disease.

The power possessed by the system to consume sugar varies in health and in disease. Carbohydrate tolerance can be determined by the administration of glucose by the mouth. The urine of healthy persons generally does not show glucose unless the amount ingested exceeds 250 grammes. When glycosuria follows the taking of 100 grammes, an abnormal sugar metabolism probably exists (pathological alimentary glycosuria). Carbohydrate tolerance is lessened by age, and is often reduced in obesity and gout.

Lactose.—The presence of milk sugar in the urine is indicated by a positive reaction with Trommer's and Nylander's tests after prolonged boiling, when negative results are obtained with the phenylhydrazine and fermentation tests. Lactose is found in the urine during the last weeks of pregnancy and in nursing women. Glycosuria and lactosuria are occasionally associated. The ingestion of more than 120 grammes of lactose often causes a lactosuria.

Levulose.—The presence of fruit sugar may be inferred when the urine gives sugar reactions with Trommer's, Fehling's, the fermentation and phenylhydrazine tests, and does not rotate polarized light to the right.

Levulose at times rotates polarized light to the left. Levulose occurs in the urine in some cases of diabetes and, at times, in the urine of healthy persons after the ingestion of levulose.

Pentose.—Pentose can be recognized by the fact that it does not undergo fermentation with yeast, but gives a positive reaction with Fehling's, Nylander's, and the phenylhydrazine tests. Pentose has been discovered in the urine after eating plums, pears, apples, cherries, and huckleberries, from the ingestion of 50 grammes or more of pentose, and occasionally in diabetes. A family tendency has been recorded.

Dextrin.—This substance reduces Fehling's solution, the copper separating first as a green, then changing to a yellow precipitate, and sometimes as a dark brown sediment. Dextrin has been found in the urine in the absence of glucose. Some authorities regard the presence of traces of dextrin as normal.

Acetone.—Legal's Test.—A few drops of freshly prepared concentrated solution of sodium nitroprusside are added to a small amount of urinary distillate, and the mixture treated with sodium or potassium hydrate. When a ruby color develops, rapidly changing to yellow, it signifies the presence of acetone. This test is usually negative with mere traces of acetone.

Lieben's Test.—A few drops of potassium hydrate solution and a small quantity of iodopotassic iodide are added to the urinary distillate, and the mixture warmed. Acetone is indicated by the formation of iodoform, which appears as hexagonal or stellate crystals, and can be recognized by its characteristic odor.

Dunning's Test. — Tincture of iodine, or Lugol's solution, is added to the urinary distillate, and the mixture treated with ammonia until a black precipitate develops, which slowly disappears, leaving a yellow deposit of iodoform crystals.

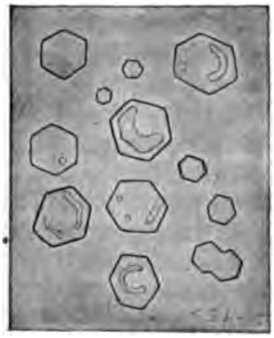


FIG. 121.—Iodoform crystals formed from the distillate of the urine of a case of diabetes. —Emerson.

Acetone occurs in normal urine in small quantities, not exceeding 10 mg. in twenty-four hours. It is increased by restricting or withholding carbohydrates from the diet, especially when large amounts of proteids are consumed. It is also augmented in febrile diseases, in certain cachexias, in gastric ulcer, and follows the administration of phloridzin, and chloroform narcosis, and in severe forms of diabetes mellitus, notably before and during diabetic coma.

Diacetic or Aceto-acetic Acid.—Gerhardt's Test.—10 or 15 c.c. of urine are subjected to the action of a solution of ferric chloride. When a precipitate forms on the addition of the ferric chloride, it is removed by filtration, and to filtrate is again added the test solution. Diacetic acid may be inferred when a Bordeaux red color develops, which may completely disappear in from 24 to 48 hours. Salicylic acid, salol, aspirin, diuretin, sodium acetate, and antipyrin may give a similar reaction. Prolonged boiling of the urine containing diacetic acid will cause a complete or partial disappearance of this substance.

Diacetic acid is rarely found in normal urine. It occurs in conjunction with large amounts of acetone, and the clinical significance of aceto-acetic acid is similar to that of acetone. Oxybutyric acid may also be associated with diacetic acid. Diaceturia is of special importance in diabetics, since it is a trustworthy sign of acidosis, and is always a forerunner of diabetic coma. Aceto-acetic acid has been noted in the urine in febrile diseases, in gastro-intestinal disturbances, especially those attended with starvation, and occasionally in individuals who have consumed a rich proteid diet for a number of days.

β-OXYBUTYRIC ACID.—The urine is evaporated to the consistency of a syrup, and an equal volume of concentrated sulphuric acid is added. By distillation crotonic acid is obtained. Crystals of crotonic acid separate on cooling the distillate. If crystallization does not occur readily, an ethereal extract is obtained, evaporated, and the residue dissolved in water and allowed to crystallize. The presence of β-oxybutyric acid may be inferred by these crystals. If fermented diabetic urine containing oxybutyric acid be subjected to polariscopic examination, polarized light is rotated to the left.

β-oxybutyric acid is the mother substance of diacetic acid, while acetone is derived from the latter substance. Its presence may be suspected when diacetic acid exists in the urine in large amounts. β-oxybutyric acid occurs less frequently than diacetic acid and acetone, and in general terms may be said to arise under conditions similar to those causing acetonuria. It is found in the urine in severe infectious fevers, during starvation, and in grave forms of diabetes. β-oxybutyric acid is generally regarded as the cause of diabetic coma. Some attribute the symptoms of this condition to a lowering of the alkalinity of the blood (alkali starvation), others contend that its toxic action is responsible.

Alkaptone Bodies.—The urine containing alkaptone bodies reduces Fehling's reagent, causing this test solution to blacken. This reaction serves to differentiate it from glucose. Nylander's, the phenylhydrazine and the fermentation tests are negative with urine containing alkaptone bodies.

Urine of alkaptonuric individuals appears normal when voided, but on standing its color changes to a reddish-brown or black. This peculiar characteristic of the urine is thought to be due to homogentisinic acid and uroleucinic acid. The cause of this condition is not known. The condition is compatible with good health, and is often peculiar to several members of a family, but inheritance does not seem to be an important factor in its production.

Ehrlich's Diazo Reaction.—This test, introduced by Ehrlich, depends on certain diazo bodies, which probably combine with aromatic compounds, giving a color reaction. The test is conducted as follows: A solution consisting of 5 parts of sulphanilic acid, 50 parts of hydrochloric acid, and 1000 parts of water, is mixed with a .5 per cent. solution of sodium nitrite in the proportion of 50 of the former to 1 of the latter. An equal volume of urine is added to this mixture and shaken. Upon the addition of a few drops of ammonia, a cherry-red color develops at the zone of contact, indicating a positive diazo reaction. On shaking, the entire fluid becomes red. A brown or salmon color constitutes a negative reaction. The chief clinical significance of this reaction relates to its almost constant presence in enteric fever, but is without value as a differential sign, since it occurs in a number of diseases. It is frequently present in measles, and occasionally in pneumonia, scarlet fever, diphtheria, phthisis, rheumatic fever, meningitis, and at times in non-febrile diseases, such as chronic nephritis, carcinoma of the stomach, and leukæmia. The administration of salol, phenol, and betanaphthol may interfere with this reaction.

Fat.—Normal urine does not contain fat, but it is present in small amounts, rarely in large quantities in chronic parenchymatous nephritis, occasionally when fat occurs in excessive amounts in the blood, and after the administration of large doses of cod-liver oil. It has been observed in bone diseases in which there is a destruction of the bone-marrow, in diabetes mellitus, leukæmia, pancreatic diseases, chronic tuberculosis of the lungs, and obesity. In chyluria or galacturia the milky appearance of the urine is due to fat globules. Chylous or chyliform urine, in addition to fat, may also contain leucocytes, red blood-cells, fibrin, albumin, and occasionally leucin, tyrosin, and cholesterin.

Cryoscopy of the Urine.—The determination of the freezing point of the urine permits one to measure its molecular concentration. The apparatus devised by Beckmann is generally employed in ascertaining the freezing point. The average freezing point in normal individuals, as determined by Koranyi, is −1.7° C., although wide variations are noted. Cryoscopy of the urine is rarely employed in routine clinical work, since the results have not been satisfactory.

Cammidge's Test.—A test for the detection of pancreatic disease has been suggested by Cammidge. He holds that this reaction is due to the presence in the urine of a peculiar body, probably pentose. For the details of this elaborate procedure the reader is referred to the manuals on special laboratory work. Cammidge claimed that a positive reaction occurs in all cases of active inflammation of the pancreas, and that by this means acute inflammation of the pancreas can be differentiated from intestinal obstruction, and chronic inflammation of the organ giving rise to occlusion of the common duct can be diagnosticated from gall-stone disease. It was asserted also that "a positive reaction is indicative of altered carbohydrate metabolism due to disturbance of the internal secretion of the pancreas." The enthusiasm excited by this announcement was followed by a speedy reaction.

The results of critical studies in a series of cases at the Mayo Clinic in 1911 justify the conclusion that "if knowledge of the clinical histories and other factors of the personal equation be eliminated, the end results, judged by Cammidge's own criteria, must be considered, as a means of diagnosing diseases of the pancreas, to be both valueless and misleading." Similar conclusions followed prolonged and careful studies in the surgical clinic of the German Hospital in Philadelphia and elsewhere. It has been shown that rapid disintegration of any of the body-cells, and especially of the polynuclear leucocytes, may cause the reaction in the urine.

Test for Renal Function.—The phenolsulphonephthalein test of Rowntree and Geraghty is the most practical single test for total renal function. The solution consists of 6 mg. of the dye to 1 c.c. of salt solution. The injection is made either intravenously or intramuscularly under aseptic requirements. At the end of one hour the urine excreted is collected and again at the end of two hours, diluted with alkaline water to a fixed quantity and the phthalein content estimated by colorimetry, the colorimeter of DuBoscq or that of Autenrieth-Königsberger or test-tubes containing the dye in serial dilutions being used for the purpose. Normally after intramuscular injection 40 to 60 per cent. is excreted in one hour and 60 to 80 per cent. in two hours.

In a small proportion of the cases the test is unreliable. This variation has been explained by the assumption that a diseased kidney may be freely permeable to one substance as a dye and impermeable to another as a poison causing certain symptoms. Cases of fatal uræmia have been reported in which a nearly normal output of phthalein followed the test employed shortly before death. The general value of the test in prognosis is, however, not greatly impaired by these rare exceptions.

VII.

THE EXAMINATION OF THE SPUTUM.

Systematic examination of the sputum furnishes important clinical data in a considerable group of diseases (see also pp. 451–459).

MICROSCOPICAL EXAMINATION.

Leucocytes.—The mere presence of leucocytes has no special significance, since they occur in every specimen. A sputum containing an abundance of white blood-corpuscles generally indicates a pathological disturbance of some part of the respiratory tract, as chronic bronchitis, bronchiectasis, pulmonary abscess, tuberculosis with cavity formation, or may be due to a rupture of an extrapulmonary purulent collection into the lungs. The polynuclear neutrophile leucocytes are most often found in sputum, although in a limited number of diseases, particularly bronchial asthma, eosinophiles are noted. The sputum in asthma is usually loaded with eosinophiles, some of which have the characteristic morphology and staining reaction of the hæmic eosinophiles, while others are supplied with a circular nucleus. In certain cases of bronchitis, tuberculosis, and after hæmoptysis, eosinophiles are present in the expectoration.

Epithelial Cells.—Every specimen of sputum contains epithelial cells. Pavement epithelium may be derived from the mouth, the pharynx, and the upper half of the larynx, while cylindrical cells may come from the nose, the lower part of the larynx, trachea, and bronchi. Catarrhal inflammation, especially in its early stages, generally determines the presence of large numbers of epithelial elements. Ciliated cells are occasionally found in asthma and acute bronchitis, provided the specimen be examined immediately after expectoration. Alveolar epithelial cells which occur in the sputum in almost every pulmonary disease, as well as in the "so-called" normal expectoration, are large, of an oval, round, or polygonal shape, supplied with one or several relatively small vesicular nuclei, imbedded in protoplasm which often contains albuminous granules, myelin droplets, fat globules, particles derived from hæmoglobin, or coal pigment. These cells occur in abundance in acute inflammatory pulmonary disease and tuberculosis. Myelin granules have an irregular outline, often present a concentric arrangement, and are found either intra- or extracellularly. Myelin probably consists mainly of protagon and of small amounts of lecithin and of cholesterin. These droplets dissolve in alcohol, stain light yellow with iodine, poorly with aniline dyes, and are not blackened with osmic acid. Alveolar epithelium, containing granules of altered blood pigment, is seen in the sputum of congestion of the lungs, notably in that form due to heart disease, hence the term "heart disease cells" is applied to them.

Red blood-cells occurring in small numbers are commonly observed in the sputum of many diseases of the respiratory tract and, therefore, have no special importance, but when present in considerable or large numbers indicate a morbid lesion. Expectoration of blood (hæmoptysis) is due to a variety of causes (see page 458). Erythrocytes in the sputum, as a rule, exhibit alteration of structure, so that crenated, dehæmoglobinized, and fractured cells are common.

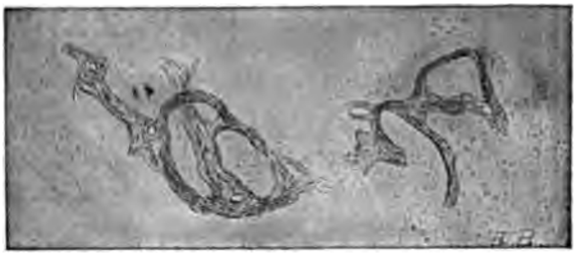


FIG. 122.—Elastic tissue from lung showing alveolar arrangement. × 50.—Emerson.

Elastic tissue, in considerable amounts, can be readily demonstrated by the following method: A thin layer of sputum, obtained by pressing it between two glass plates, is examined with the aid of a hand lens. When elastic tissue cannot be recognized by this method, the microscope should be employed; a suspected particle, which generally has a gray or yellow color, is placed upon a slide and studied by low magnification. Elastic tissue may also be demonstrated by treating the sputum with an equal quantity of a 10 per cent. solution of potassium or sodium hydroxide and boiling the mixture until it becomes homogeneous. The solution is shaken with four or five parts of water and the mixture centrifugated. The sediment is then examined microscopically. Elastic tissue is found as long slender threads, generally having a waxy appearance, and at times these fibres conform to the outline of alveoli. The presence of elastic fibres indicates disintegration of bronchial or pulmonary tissue, the latter being positively affirmed when the fibres have an alveolar arrangement. Elastic tissue is noted in bronchiectasis, pulmonary abscess, gangrene, tuberculosis, and tumors of the lungs.

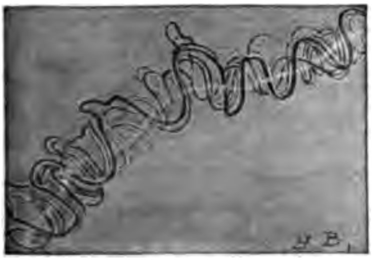


FIG. 123.—Curschmann's spiral, from the sputum of a case of asthma. × 200.—Emerson.

Curschmann's spirals are noted in the sputum in cases of bronchial asthma, occasionally in tuberculosis, croupous pneumonia, and bronchitis. Upon microscopic examination, they consist of delicate twisted threads, often wound around a central core. Many of these spirals are coated with mucus in which epithelial cells, eosinophiles, neutrophile leucocytes, and Charcot-Leyden crystals are imbedded. Curschmann's spirals consist chiefly of mucus, while the central core is held to be fibrin in some instances. Many authorities claim that these bodies are formed in the bronchioles.

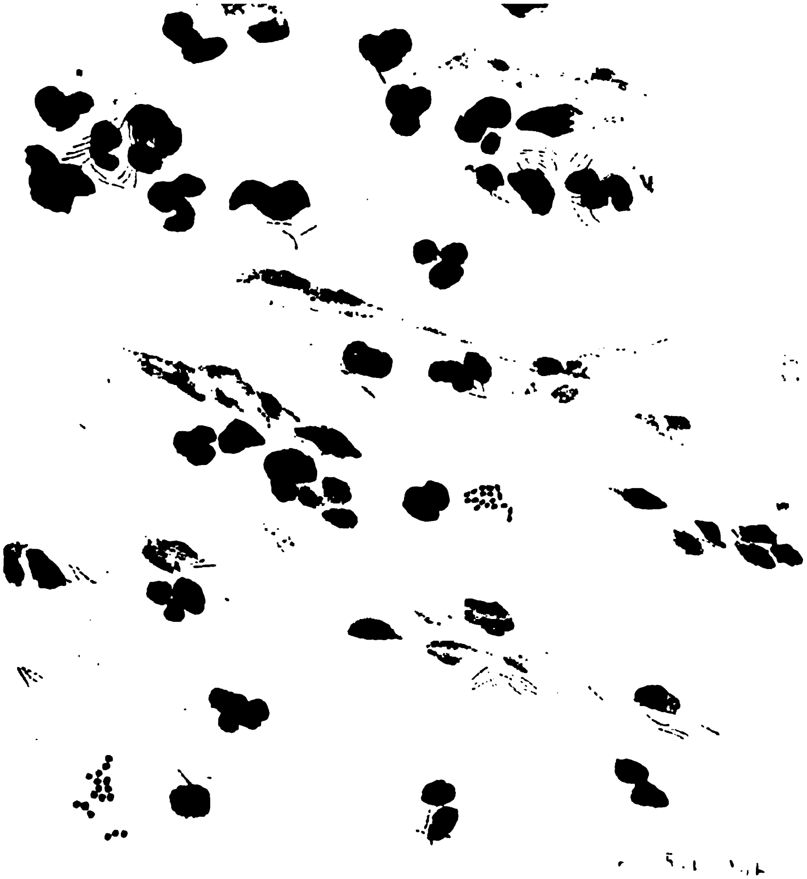
Crystals.—With the exception of Charcot-Leyden crystals, very little importance can as yet be attached to the presence of crystalline bodies. Charcot-Leyden crystals are colorless and have the shape of two elongated, sharply pointed, hexagonal, pyramidal figures with bases opposed. They stain with eosin. It was formerly thought that they were the exciting factor of bronchial asthma. This view is no longer entertained, since these crystals are occasionally found in other diseases, such as bronchitis and tuberculosis. They are probably formed from eosinophile cells. Crystals of fatty acids are noted in the sputum of tuberculosis, gangrene, bronchiectasis, and fetid bronchitis. CHOLESTERIN PLATES, which are rarely seen in the sputum, have been found in conjunction with fatty acid crystals in abscess of the lung, and phthisis. HÆMATOIDIN CRYSTALS occur in the putrid sputum of certain lung diseases, and in empyema and hepatic abscess with a bronchial outlet, and occasionally after hæmoptysis. Leucin and tyrosin crystals are at times present in purulent sputum, while calcium oxalate and triple phosphate crystals are rare ingredients of sputum.

Animal Parasites.—THE TRICHOMONAS PULMONALIS has been reported in a few instances in the sputum in lung gangrene, tuberculosis, abscess, and putrid bronchitis, while circomonads have been recorded in pulmonary gangrene. The sputum in cases of liver abscess perforating into the lung may show the Amœba coli. TÆNIA ECHINOCOCCUS.—Hydatid disease may cause pulmonary abscess or gangrene and is sometimes responsible for copious hæmoptysis. The sputum in this condition may contain shreds of cyst membrane, daughter cysts, scolices and hooklets of the worm. DISTOMA PULMONALE.—This parasite is responsible for a form of chronic pulmonary disease, characterized by hæmoptysis, seen in Japan, China, and Korea. This fluke and its ova are found in the sputum.

Vegetable Parasites.—A large number of micro-organisms have been found in the sputum. Among these may be mentioned: the tubercle bacillus, Diplococcus pneumoniæ, staphylococci, streptococci, sarcinæ, streptothrix, actinomyces, Micrococcus catarrhalis, and the influenza, smegma, typhoid, plague, diphtheria, and Friedlander's bacillus.

In the case of tubercle bacilli, their staining reaction, outline, and size, in the absence of biological tests, generally afford sufficient evidence to establish the diagnosis of this organism. With most bacteria occurring in the sputum this is not the case, so that their identity can only be determined provisionally but not finally by their tinctorial and morphological characteristics. This tentative opinion is, however, often strengthened by the correlation of the clinical data of the underlying pathological process. Cultural studies are as a rule essential, and inoculation experiments often required for a bacteriological diagnosis. Works on bacteriology should be consulted for bacteriological investigations.

TUBERCLE BACILLUS.—The finding of tubercle bacilli in the sputum is a valuable sign in establishing the diagnosis of tuberculosis of the lungs, although the absence of these organisms in the expectoration of an individual presenting pulmonary symptoms does not necessarily negative the diagnosis. The failure to find bacilli on a number of examinations in a suspected case, particularly of a chronic nature, is strong evidence against the existence of phthisis. In acute tuberculosis, especially in the early stages, they are frequently wanting in the sputum. There is no single characteristic presented by macroscopic examination of the sputum by which its tuberculous nature can be recognized. Rosenberger holds the view based on repeated observations that tubercle bacilli are present in the fæces of persons suffering from active pulmonary tuberculosis, even in the acute miliary form. The *technic* of the examination for tubercle



Tubercle bacilli in sputum stained with [illegible]

bacilli is as follows: Preferably a caseous mass or a bit of purulent or hemorrhagic sputum is placed upon a slide or cover-slip. In the absence of cheesy particles, specimens are selected from different parts of the sputum. A thin smear is made, carefully dried and fixed by rapidly passing the slide or cover-glass through a flame several times. The tubercle bacillus belongs to the group of acid-fast bacteria, which, after staining, resist to a marked degree decolorization with solutions of mineral acids.

To concentrate the bacteria in the specimen of the sputum, the method of Mülhäuser-Czaplewski will be found most serviceable. From four to eight volumes of a 0.25 per cent. solution of sodium hydrate are added to the sputum, placed in a bottle, and shaken until the fluid has a uniform mucilaginous appearance. A few drops of phenol-phthalein solution are added and the liquid is boiled. A 2 per cent. solution of acetic acid is now added drop by drop until the pink color of the liquid just disappears. The material can now be centrifugated and the sediment examined.

Antiformin.—To a portion of sputum in a centrifuge tube add an equal amount of a 10–15 per cent. solution of antiformin; allow it to stand for a few minutes and then centrifugate. Decant the supernatant antiformin solution and again centrifugate. At this point a few cubic centimetres of alcohol may be added to lower the specific gravity and aid in the precipitation of the bacilli. The sediment is then smeared on the slide and stained by one of the usual methods.

Ziehl-Neelsen Method.—The stain consists of 10 c.c. of a concentrated alcoholic solution of fuchsin, dissolved in 90 c.c. of a 5 per cent. solution of carbolic acid. The film of sputum is covered with the stain. The cover or slide is then held over a flame until the solution is brought to the boiling point; or the specimen may be stained in cold carbol fuchsin for 24 hours. After a half minute, the excess of hot stain is poured off and the specimen washed with water. The stained preparation is next placed in a 25 per cent. solution of nitric acid for several seconds until the bright red color disappears, then washed in water and dried. The specimen may be counterstained with a watery solution of Bismarck brown or methylene blue for a minute or two. The cover-glass film is mounted on a slide in balsam or cedar oil. The specimen spread and stained upon a slide, the most convenient method, may be examined without a cover-glass.

Gabbett's Method.—The sputum properly spread and fixed upon a slide or cover-glass is covered with a reagent consisting of fuchsin 1 gramme, alcohol 10 cubic centimetres, and a 5 per cent. solution of carbolic acid 100 cubic centimetres, and held over a flame until the stain boils. After draining off the carbol fuchsin from the slide, the specimen is treated for two minutes with Gabbett's reagent, composed of methylene blue, 2 parts, dissolved in 100 parts of a 25 per cent. solution of sulphuric acid; then washed with water, thoroughly dried, and examined microscopically.

Pappenheim's Method. — This method affords the means of distinguishing tubercle bacilli from other acid-fast organisms. The stain is prepared by dissolving 1 part of corallin in 100 parts of absolute alcohol. This solution is then saturated with methylene blue, after which 20 parts of glycerin are added. After staining the specimen with a heated carbol fuchsin solution in the manner previously described, the excess of stain is drained from the slide and immediately Pappenheim's solution is placed upon it and allowed to act for a few minutes. Fresh solutions may be added several times if the spread is tinged red in any part. The slide is next washed in water, dried, and examined.

With these methods, tubercle bacilli appear as straight or slightly bent red rods, varying from 1.5 to 4 microns in length and from .1 to .2 micron in thickness. Occasionally they are tinted more deeply in certain parts, having the appearance of a streptococcus (beaded forms). Branching forms are rarely found. The older varieties of bacilli are thought to stain more intensely than the younger forms. As a rule a number of organisms can be found in preparations, many of which are frequently arranged in groups containing several or more organisms. It is most uncommon to find but a single bacillus in a specimen and, when this occurs, the possibility of contamination of the sputum from dust should be remembered. The number of germs in chronic cases often is an index to the extent of the ulceration in the lung, although, in acute cases, the degree of the tubercle involvement bears no relation to the abundance of bacilli. A lessening in the number of bacilli ofttimes is associated with a steady improvement in the patient, and a disappearance of the micro-organisms frequently points to quiescent or healed lesions.

Diplococcus Pneumoniæ.—The finding of pneumococci in the sputum, in the absence of other clinical data, is without diagnostic significance, since these organisms exist in the saliva of a considerable proportion of healthy individuals, as well as in the expectorated material in several diseases. Their presence in the sputum of a case exhibiting pulmonary symptoms often establishes an etiological diagnosis. Pneumococci are found in large numbers in the sputum of croupous pneumonia and occasionally in bronchopneumonia. This organism reacts positively to solutions of basic dyes. Stained specimens frequently show a colorless capsule about the diplococci.

VIII.

THE EXAMINATION OF TRANSUDATES, EXUDATES, AND THE CONTENTS OF CYSTS.

The results of the examination of transudates, exudates, and the contents of cysts by physical, chemical, microscopical, and bacteriological methods are diagnostic auxiliaries.

Exploratory Puncture.—An exploratory syringe, equipped with a large stout needle, is generally used for this purpose, but for some explorations the aspirator needle alone is employed, since the positive internal pressure of certain effusions expels the fluid. The operation of exploratory puncture must be performed under strict antiseptic precautions; the skin should be sterilized by thoroughly scrubbing with soap and hot sterilized water, followed by washing with hot sterilized water and then with an antiseptic solution. When possible an antiseptic dressing should be applied for some hours prior to performing the operation. The hands of the operator should be surgically clean and the instrument should be sterile. The technic of peritoneal, pleural, pericardial, and lumbar puncture is discussed in Part IV.

The differentiation between exudates and transudates is not, as a rule, difficult, since the internist is guided by associated clinical phenomena in determining the origin of the fluid. There are, however, cases in which the character (whether it be inflammatory or non-inflammatory) of the material cannot be ascertained by the symptoms or the history of the case, and the final distinction must rest with the laboratory, although in a few instances the various methods of laboratory research fail to solve the problem.

Transudates are generally light yellow or pale yellowish-green, at times reddish, due to blood staining, milky as noted in chylous effusions, and dark yellow when deeply tinged with biliary pigment.

The composition of transudates of the peritoneal, pleural, and pericardial sacs is nearly the same. They consist of water (95–96 per cent.), solids, proteids, extractives, inorganic salts, and uric acid. Allantoin. dextrose, fructose, urobilin, and biliary pigment have also been demonstrated in transudates. Their specific gravity is generally below 1.018, in many cases as low or even below 1.010, and occasionally above 1.020. Hydræmic transudates are of lighter specific gravity than those due to stasis. The specific gravity is influenced mainly by the proteids contained in the fluid, so that exudates, which are generally richer in albuminous bodies than dropsical fluids, are as a rule of higher gravity than transudates. The estimation of the total proteids content is therefore of value in differentiating between transudates and exudates. The percentage of proteids in stasis transudates generally ranges from 1 to 3, while in hydræmic effusion it is much lower, usually not above .5. Transudates either contain no fibrin or it exists only in minute amounts. A few endothelial cells and leucocytes, at times erythrocytes and cholesterin crystals, are found. In hydroperitoneum occurring in leukæmia, Charcot-Leyden crystals, mast cells, and eosinophiles have been recorded. In the main the chlorides exist in greater concentration in transudates than in exudates, and as a rule the degree of alkalinity of dropsical fluids is about that of the blood of the individual in question, while in an exudate it is lowered.

Exudates.—The chief varieties of exudates are serous, hemorrhagic, purulent, and putrid, and between these types there are gradations and combinations. The recognition by macroscopic inspection of purulent collections is generally a simple matter, although serous exudates, which contain a large number of fine fibrin flakes and chylous fluids, are of similar appearance. The uniform turbidity of purulent effusions serves to distinguish them from serofibrinous effusions, while the presence of fine granules of fat is characteristic of chylous fluids. In many inflammatory collections a coagulum forms immediately after the fluid is withdrawn from the body. Their specific gravity is generally above 1.018, the proteid content is usually above 4 per cent. and at times as high as 6 per cent. Serum albumin and globulin in considerable amounts, traces of fibrinogen and serosamucin are present in exudates; nucleo-albumin, albumoses, leucin, and tyrosin have also been noted.

RIVALTA'S TEST.—The principle of this test is based on the precipitation in many exudates of a peculiar body, the character of which has not been definitely determined, although regarded by some authorities as mucin and denominated serosamucin, while others hold that it is a globulin. This test is carried out by allowing a drop of the fluid to fall into a weak acetic acid solution (two drops of glacial acetic acid in 100 c.c. of distilled water). When the drop sinks and leaves a turbidity it indicates the presence of this substance, while the failure to produce cloudiness denotes the absence of this body (serosamucin). The intensity of the cloudiness and the rapidity with which it forms are an index to the amount present. This test is of importance in differentiating exudates from transudates.

Animal parasites, bacteria, many cellular elements, lymphocytes, polynuclear cells, endothelial cells, erythrocytes, and detritus occur in exudates.

BACTERIOLOGICAL EXAMINATION.—Bacteria rarely exist in transudates, but their presence in exudates, which is frequent, furnishes a most useful field for diagnosis and prognosis. The fluid for bacteriological examination is collected in a sterile flask, the neck of which is then immediately plugged with sterile cotton. (For technic consult works on bacteriology.) A diagnosis of tubercle bacilli can often be made by staining methods.

COLLECTING SEDIMENT.—Fluids removed by puncture often coagulate spontaneously. Since the coagulum entangles some of the cellular bodies and bacteria, the elements which remain in the fluid portion do not form an accurate basis for calculating the number or the percentages of the different varieties of cells. In order to prevent coagulation one-third or fourth volume of a 2 per cent. sodium citrate salt solution is added to the specimen. After centrifugalization or sedimentation the supernatant fluid is removed and the tube is filled with saline solution, then gently agitated and recentrifugated. Much of the albumin is removed from the fluid by this procedure, which insures better results in staining.

INOSCOPY, the method introduced by Jousset, was designed to aid in the diagnosis of tuberculosis. The exudate is allowed to coagulate spontaneously, but should this not occur the addition of horse serum will bring about clotting. The coagulum which holds many of the tubercle bacilli is then removed, broken up, and digested by means of a fluid consisting of NaF 3 grammes, pepsin 1 or 2 grammes, glycerin 10 c.c., HCl 40 per cent. 15 c.c., water 1000 c.c. The resulting liquid is then centrifugated and the sediment examined in the usual manner for tubercle bacilli.

CYTOLOGICAL EXAMINATION.—After securing the sediment of the citrated material, or the digested coagulum, it should be properly fixed. Treating the sediment with a ½ or 1 per cent. formaldehyde solution for several minutes is highly recommended by some workers. The sediment is spread into a thin film upon a slide or cover-glass, dried, and if not previously fixed is now subjected to such fixatives as methyl alcohol, or alcohol and ether, heat, or formalin solutions. The selection of the stain depends upon the structures desired to be demonstrated and upon the choice of the worker. Most of the Romanowsky modifications or double stains, as eosin and hæmatoxylon or methylene blue, give satisfactory results. The principle which involves the determination of the percentages of the various types of cells is the same as for differential leucocyte counting. Immediate citration of fresh specimens, followed by centrifugating, probably offers the best means of studying cellular elements and bacteria.

CYTODIAGNOSIS.—The cytological formula does not diagnose a disease, but rather suggests the acuteness or chronicity of a pathological condition, the stage and intensity of a morbid process, or the absence of inflammation. A rare exception relates to effusions which contain tumor fragments, the histology of which may be diagnostic.

The most important cellular elements entering into cytological studies are lymphocytes, polynuclear cells, endothelial cells, eosinophile cells, mast cells, erythrocytes, and tumor cells.

Endothelial Cells.—An increase of endothelial cells in a fluid is generally associated with non-inflammatory effusions of the serous cavities. Dropsical effusion due to passive congestion and hydræmic transudates shows endotheliocytosis. In the early stages of a tuberculous effusion a high percentage of endothelial elements is sometimes noted.

Lymphocytes. — An irritant of mild intensity is responsible for a lymphocyte predominance in an effusion. Such a reaction is essentially local and does not provoke a general stimulus. An irritation of low grade, especially when protracted over a long period, calls forth these cells. Lymphocytosis is the rule in tuberculosis, although a polynucleosis may precede a lymphocytic phase or in some instances it may follow. These variations are attributed to increased virulence of bacteria and to secondary or mixed infections. A lymphocyte preponderance preceded by a polynucleosis is regarded as having a favorable prognostic significance. The development of a polynucleosis taking the place of a lymphocytosis is suggestive of a complication. In the late stages of acute inflammations or when these tend to become chronic, a high lymphocyte percentage is often noted. Lymphocytosis is noted almost constantly in effusions of tuberculous origin and sometimes in those due to syphilis, uræmia, malignant tumors, and paresis.

Polynuclear Cells.—The exudates in acute inflammation or infections of serous sacs, such as are produced by staphylococci, pneumococci, streptococci, meningococci, colon bacilli, and typhoid bacilli, contain a high percentage of polynuclear leucocytes. In the early stage of tuberculosis a polynucleosis is sometimes noted, and frequently in tuberculosis pericardial effusions. As an acute inflammation subsides polynuclear preponderance becomes less marked, and this is often followed by a rise in the number of the lymphocytes, which may outnumber the multinuclear elements.

Eosinophilic cell increase has been recorded in effusion occurring in the course of rheumatic fever, tuberculosis, nephritis, syphilis, carcinoma, and following trauma.

Mast cells have been noted occasionally in effusions, especially those of long standing.

Erythrocytes.—Contamination of the fluid with blood from the wound made by puncture is unavoidable in many instances, but aside from this source red corpuscles in an effusion are at times the expression of malignant, renal, or tuberculous disease. They are also seen in effusion due to acute infections. The possibility of a hemorrhage, as in cerebral apoplexy with effusion into the ventricles of the brain, or a small leak of an aneurism into a serous sac, should always be borne in mind.

Cells derived from carcinomata and sarcomata when found singly in effusions of serous cavities are believed by some writers to possess certain features which may be of diagnostic value. These cells may show mitotic figures. The results of an histological examination of a tumor fragment may warrant a final diagnosis.

The recognition of some of the varieties of cells just described may not be so simple a matter. A cell having a single nucleus undergoing degeneration and fragmentation may resemble a multinuclear element. Polynuclear cells may be difficult to detect when the cell body undergoes shrinkage and becomes disintegrated.

Chylous fluids owe their turbidity to fine particles of fat. The amount of fat varies; it is often under 1 per cent., but in a case reported by Hammerfahr it reached 2.95 per cent. Other constituents of this variety of effusion are water (90 per cent. +), albumin, fibrin, globulin, cholesterin, lecithin, salts, soaps, fatty acids, and other substances. The fat is soluble in ether and gives the tests for this substance.

There are certain effusions designated chyloid or pseudochylous which closely resemble chylous fluids in their gross appearance but differ from them since the free, fine, fat particles are absent. The opalescence of these fluids probably depends on a variety of causes, while in some instances the milky appearance cannot be explained. The presence of endothelial or epithelioid cells with a fatty degenerated protoplasm is the explanation suggested by Quincke in some of these cases. Other observers hold that bacteria, globulins, lecithin, mucin, and certain proteids (other than globulin) are responsible for the turbidity which may in some instances suggest a purulent character rather than a milky appearance.

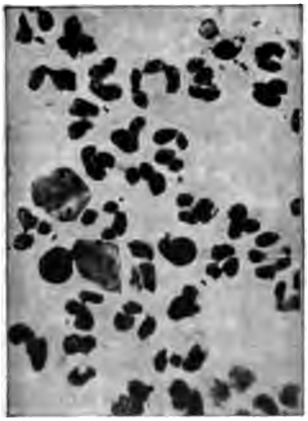


FIG. 124.—Smear of the spinal fluid of a case of epidemic cerebrospinal meningitis.—Emerson.

Chylous collections are not uncommonly noted, especially in the peritoneal cavity and pleural sacs, rarely in the pericardium. These effusions arise in a number of diseases in which pressure is exerted on the thoracic duct or the lymphatic vessels.

Cerebrospinal Fluid.—In health the cerebrospinal fluid obtained by lumbar puncture is colorless, clear, of alkaline reaction, has a low specific gravity, ranging from 1.003 to 1.007 due to the presence of from 1 to 1.5 per cent. of solids and cellular elements (endothelial cells and leucocytes), not exceeding 5 per c. mm. The amount under normal conditions has been set as varying between 5 and 10 c.c. although these figures are only approximate. The dural pressure as determined with an ordinary water manometer in the dorsal position ranges from 60 to 100 mm. in health, while in disease, as in meningitis and cerebral tumor, it may reach from 200 to 800 mm. Serious symptoms may arise on withdrawing the fluid when the pressure falls below 60 mm.

Urea, globulin, protalbumose, nucleoproteid, and a reducing substance probably similar to pyrocatechin, and sodium chloride and other inorganic salts are present. Serum albumin is said never to exist in the normal fluid. There is some doubt as to the presence of glucose; some authorities claim that it is a normal constituent (.4 to .5 per cent.), which disappears when meningitis develops (Lannois and Boulard), while others hold that it does not exist in health.

In pathological conditions the cerebrospinal fluid is often altered.

In disease the quantity varies from a few c.c. to more than 100 c.c. The amount is increased in acute hydrocephalus, in general paresis, dementia præcox, in some of the infectious diseases, in brain tumors, and in meningitis. It should be borne in mind that interference with the circulation of the cerebrospinal fluid from increased intracranial pressure, as in brain tumor, which cuts off the communication between the subarachnoid basilar spaces of the brain and those of the cord, may result in an absence or diminished amount.

The fluid is often pale yellow, cloudy, or creamy in appearance in acute meningitis, while in tuberculous meningitis, hydrocephalus, and brain tumors it is generally clear and colorless. In hemorrhage into the ventricles fluid blood may be obtained by puncture, while in icterus the fluid is yellowish. As a rule a turbid fluid points to an acute meningitis. Albumin may be present and the specific gravity is raised when acute inflammation of the meninges exists. Cholin, a substance which is derived from the destruction of nerve tissue, is present in the spinal fluid in cases of organic disease of the nervous system, notably in paresis, tabes dorsalis, syphilitic epilepsy, dementia paralytica, cerebral abscess, brain syphilis, myelitis, and spina bifida.

Bacteriological studies of the spinal fluid are most essential in diagnosis. The following are the more important bacteria which have been found by lumbar puncture: meningococcus, pneumococcus, staphylococcus, streptococcus, B. tuberculosis, B. coli communis, B. influenzæ, B. mallei, B. pyogenes fœtidus.

Trypanosomes are present in the spinal fluid in African sleeping sickness.

Cytological Examination.—The results of cytological studies of the cerebrospinal fluid fall in line with those previously mentioned. In tuberculosis a high lymphocyte count is the rule. Lymphocyte preponderance has also been noted in paresis, tabes, cerebrospinal syphilis, syringomyelia, cerebral tumors, pressure myelitis, in chronic and in later stages of cerebrospinal meningitis, in epilepsy, and in sleeping sickness. In acute meningitis, such as is determined by the meningococcus, staphylococcus, streptococcus, pneumococcus, B. typhosus, B. coli communis, a multinuclear cellular predominance exists.

Contents of Cysts.—PANCREATIC CYSTS.—The evidence that the fluid from an abdominal cyst has the property of digesting albumin in an alkaline medium suggests a pancreatic origin. A negative result does not rule out the possibility of pancreatic cyst, since trypsin disappears in collections of long standing.

OVARIAN CYST.—Fluid of ovarian cysts is often pale yellow, sometimes reddish or dark brown; the specific gravity shows wide fluctuation between 1.010 to 1.038; the consistency varies from a watery fluid to dense, viscid, "jelly-like" material.

Cystic collections of low specific gravity contain little albumin (serum albumin and globulin), while those of high specific gravity have large amounts of albumin. Puramucin is present in colloid cysts. Pseudomucin or metalbumin also exists in these cysts.

Ciliated cylindrical epithelial cells, squamous epithelium, erythrocytes, fat, fatty acid crystals, cholesterin plates, and hæmatoidin are also noted in the cysts.

Hydatid Cysts.--They may be recognized by the presence of cyst membrane, scolices, and hooklets. The fluid of these cysts is almost colorless, of very low specific gravity, contains little or no albumin, shows a considerable amount of sodium chloride, has a neutral or faintly acid reaction, and traces of sugar and succinic acid may be present. Granular and fatty detritus, calcareous fragments, hæmatoidin, cholesterin crystals, and granular cells are frequently found. In the event of suppuration leucocytes appear in the fluid.

Hydronephrosis.—The fluid of hydronephrosis does not always present features which are diagnostic. This applies especially to chronic hydronephrosis with complete occlusion of the ureter. In acute cases or those associated with partial occlusion of the ureter so that the kidney still functionates, the presence of a high urea content and uric acid, and especially when renal tube-casts and cells are found, renders the diagnosis a comparatively simple matter.

IX.

THE EXAMINATION OF THE NERVOUS SYSTEM.

PRELIMINARY CONSIDERATIONS.

The diagnosis of disease of the nervous system demands an accurate knowledge of the anatomy and physiology of the structures involved and of the pathological processes to which they are liable. It is essential to determine not only the location but also when possible the nature of the lesion.

The nervous system, by which the organism is brought into relation with its environment and by which its functions are made manifest and controlled, is essentially composed of morphological units having a similar structure—the neurons—and held together and supported by a special tissue—the neuroglia.

The Neuron.—Each neuron consists of (a) a nucleated protoplasmic mass—the cell-body—which presides over the nutrition of the neuron and is the seat of origin of nervous impulse, and (b) processes which form outgrowths from the cell-body and constitute the elements along which impulses are conveyed. These processes are of two kinds, (a) branched protoplasmic outgrowths, *dendrites,* which may be multiple and form arborescent interlacing ramifications with similar processes from other neurons, and (b) the single elongated process, *axon,* commonly prolonged to form the axis-cylinder process.

The dendrites, uneven in contour and relatively thick as they arise from the cell-body, rapidly become more slender in consequence of their repeated branching until they terminate in delicate end branches with terminal bead-like thickenings.

The axons are slender thread-like extensions of uniform diameter and variable length, sometimes reaching only to adjacent neurons, sometimes extending to distant neurons within the cerebrospinal axis, as from the cerebral cortex to the lower part of the spinal cord, sometimes beyond as from the lower part of the cord to the muscles of the foot. The axons, like the dendrites, finally terminate in end arborizations—*telodendria.* Most of them, shortly after leaving the cell-body, give off processes termed collaterals which after a variable course terminate in end arborizations which interlace with the processes of other and sometimes distant neurons. Less frequently short axons arise which are not continued as axis-cylinders but at once terminate in complex branching end brushes within the substance of the gray matter.

Histologists are not of accord as to whether the relation between the neurons is that of continuity or simple contiguity. The weight of opinion is at present in favor of the view that the neurons are separate and distinct morphological units, their processes interlaced to form paths of conduction but probably never actually continuous in the anatomical sense. The axis-cylinders, usually supplied with a medullary sheath, are described as *nerve-fibres.* Collected into bundles they form the *nerve-trunks* which ramify to the various muscles and other organs.

Divisions of the Nervous System.—*Central Portion.*—In vertebrates there is an axial accumulation of the cell-bodies in the cerebrospinal axis from and to which the processes pass. This includes the brain and spinal cord and contains the principal axial collections of neurons.

Peripheral Portion.—This division embraces the nerve-cells of the sensory ganglia and is chiefly made up of the nerve-fibres which pass to and from the end organs.

Sympathetic Nervous System.—This division is intimately correlated with the peripheral nervous system, but possesses a certain degree of physiological independence and supplies the unstriped muscular and the glandular tissues of the body and the muscle of the heart.

Nerve Terminations.—The terminal end arborizations of the peripheral nerves constitute the mechanism by which the various structures of the body are combined in consistent and harmonious relation with the nervous system. Certain of these terminations transmit impulses which give rise to muscular contraction; others originate impulses which cause various sensations of pain, temperature, pressure, or the special senses. The nerve terminations may therefore be divided according to their function into *motor* and *sensory.*

Motor Nerve Endings.—These include three groups: (a) The terminal arborization of the axons of neurons in the motor nuclei of the spinal cord and brain stem that pass to voluntary muscle; (b) those of sympathetic neurons that pass to involuntary muscle; (c) the muscle of the heart.

Sensory Nerve Endings.—These are the peripheral terminal arborizations of the neurons, the cell-bodies of which are in the spinal and other sensory ganglia. They therefore constitute the point of departure of the paths which conduct sensory stimuli to the central nervous system.

The function of the neuron is to conduct nervous impulses. In its simplest form the nervous system consists of (a) the *sensory neuron*, which receives the external stimulus acting upon the integument and other sensory surfaces and by means of its process conducts it from the periphery to the cell-body which commonly lies in the cerebrospinal axis. Such a process constitutes functionally a centripetal or afferent fibre. The stimulus thus received is transmitted from the cell-body of the sensory neuron by means of its dendrites which interlace with those of the associated cell-body of (b) a *motor neuron* to the latter, in which a responsive impulse originates and is conveyed along its axis-cylinder process—nerve-fibre—to the muscle-cell and causes contraction. The latter process is therefore known as a centrifugal or efferent fibre. This elementary conception of the relation and functions of the sensory and motor neurons is greatly modified by the fact that the centripetal impulses are conveyed to the cell-bodies of other neurons not only in the immediate neighborhood but also at different and even distant levels. Neurons of the same function are usually grouped together, aggregations of cell-bodies forming nuclei, and collections of the fibres forming bundles, tracts, or systems. The former are situated in the gray matter of the brain and spinal cord, the latter run in the white substance of the brain and spinal cord and in the peripheral nerves. By this means the various parts of the central nervous system are connected with each other and with the muscles and viscera.

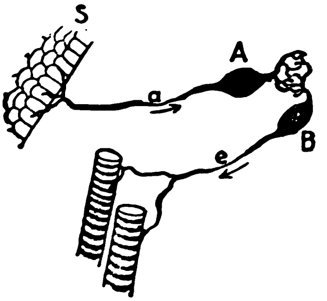


FIG. 125.—Diagram showing fundamental units of nervous system. *A*, sensory neuron, conducting afferent impulses by its process (*a*) from periphery (*S*); *B*, motor neuron sending efferent impulses by its process (*e*) to muscle.—Piersol.

Many of the tracts are highly complicated and obscure both as to their course and formation. Others are simpler and, as the result of studies of the degenerations caused by injury or disease, have been traced in their course through the cerebrospinal axis. Chief among the latter group is the pyramidal tract which transmits motor impulses from the cortex to the periphery.

The Motor System.—A muscular movement depends upon the combined functional activity of many associated neurons. It follows that the movements of the various parts of the body are represented in the central nervous system by localized aggregations of correlated neurons, or centres. Muscular movements are not only localized in the motor areas of the cerebral cortex but they are also localized in the different levels of the ventral horns of the spinal cord and the motor nuclei of the cerebral nerves. Voluntary motor impulses originating in the cortex of the brain pass through at least two neurons before they reach the muscles. For this reason the motor tract is divided into an upper and a lower segment.

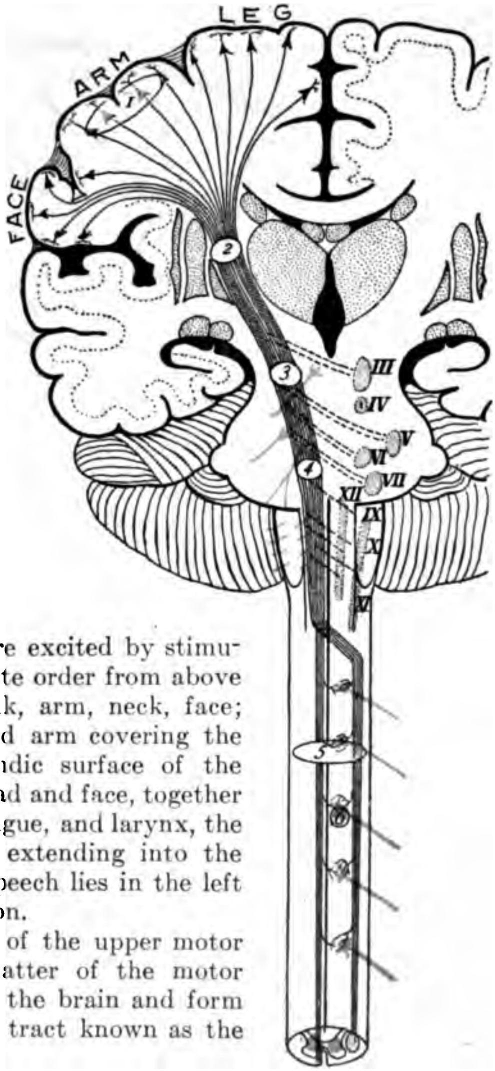


Fig. 126.—Diagram of motor path from right cortex. Upper segment black; lower red. A destructive lesion at 1 causes upper segment paralysis of the arm of the opposite side; at 2 upper segment paralysis of the opposite side—hemiplegia; at 3 upper segment paralysis of the face, arm, and leg of the opposite side and lower segment paralysis of the eye muscles of the same side—crossed paralysis; at 4 upper segment paralysis of arm and leg of the opposite side and lower segment paralysis of the face and external rectus of the same side—crossed paralysis; at 5 upper segment paralysis of all muscles below lesion and lower segment paralysis of muscles represented at level of lesion—spinal paraplegia; at 6 lower segment paralysis of muscles represented at level of lesion—anterior poliomyelitis.—VanGehuchten modified.

The Upper Motor Segment.—Clinical researches (Hughlings Jackson), experimental studies (Hitzig, Ferrier, Horsley and others), and the studies of tract myelination at progressive periods in the development of the cerebrum by Flechsig, have thrown much light upon the functions of many of the cortical regions of the brain and the sensory and motor tracts. The cell-bodies of the upper motor neurons are arranged in functionally allied groups in the cerebral cortex over the ascending frontal convolution and extending deeply into the fissure of Rolando. In this region the movements of the body are definitely represented. It has been demonstrated that motor impulses are excited by stimulation over these areas in a definite order from above downward, as follows: leg, trunk, arm, neck, face; the areas for the leg, trunk, and arm covering the upper half, including the Rolandic surface of the convolution, and those for the head and face, together with those for the jaws, lips, tongue, and larynx, the lower half, likewise the surface extending into the fissure. The centre for motor speech lies in the left third frontal, Broca's, convolution.

The axis-cylinder processes of the upper motor neurons pass from the gray matter of the motor cortex into the white matter of the brain and form part of the extensive converging tract known as the corona radiata. Collected into a compact bundle—the pyramidal tract—they pass between the basal ganglia in the internal capsule occupying the knee and the anterior two-thirds of the posterior limb. The movements of the opposite side of the body are represented at this level from before backward in the following order: eyes, head, tongue, mouth; shoulder, elbow, wrist, fingers, thumb; trunk; hip, ankle, knee, toes.

Emerging from the internal capsule the fibres of the pyramidal (corticospinal) tract pass into the crus. At this point some of them leave the tract and crossing the middle line end in arborizations among the ganglion cells in the nucleus of the third nerve upon the opposite side, and at successive levels fibres are given off which terminate in the nuclei of all the motor cerebral nerves of the opposite side, while a limited number of fibres are distributed to the corresponding nuclei of the same side. From the crus the pyramidal tract enters the pons and passes to the medulla oblongata forming its anterior area—the pyramid. At the lower limit of the medulla, after the fibres to the nuclei of the cerebral nerves have been given off, five to seven coarse strands pass obliquely across the anterior median fissure, interlacing with similar strands from the opposite side and thus constituting the decussation of the pyramids. In consequence of this arrangement the greater number of the fibres of the important motor paths pass to the opposite sides to reach the lateral columns of the cord in which they descend as the lateral or crossed pyramidal tracts. The fibres that remain upon the same side as the pyramid from which they emerge

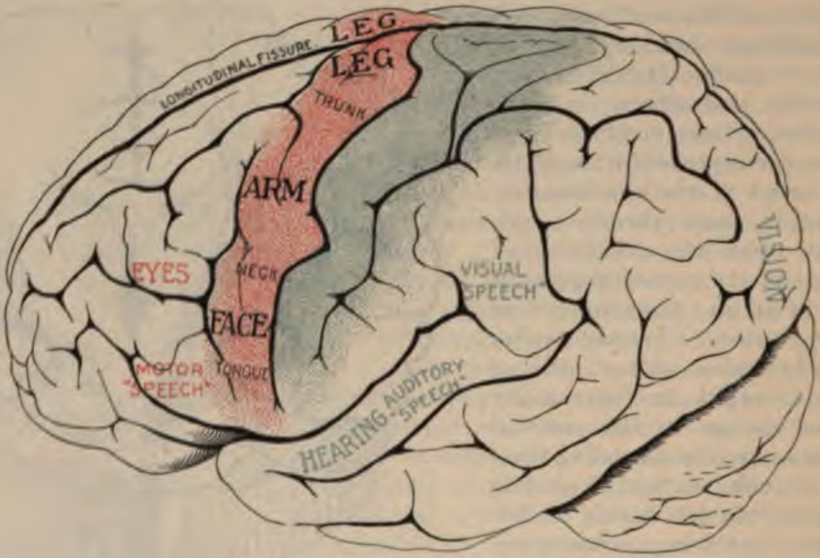


FIG. 127.—Diagram of cortical centres.

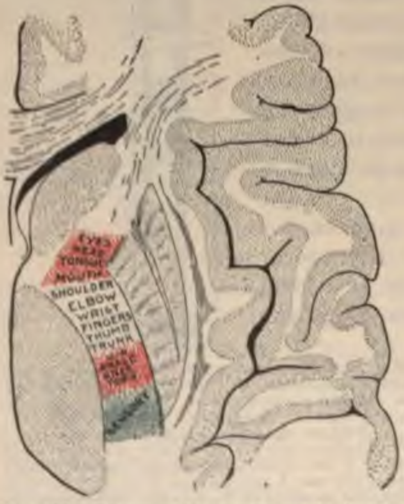


FIG. 128.—Diagram of internal capsule showing motor and sensory paths.

are collected in its lateral portion and descend in the ventral columns as the direct pyramidal tracts or Türck's columns. At every level of the spinal cord axis-cylinder processes emerge from the crossed pyramidal tract to enter the ventral horns and end in arborizations about the cell-bodies of the lower motor neurons. In consequence of this arrangement the tract diminishes in size as it descends in the cord. In a somewhat similar manner the fibres of the direct pyramidal tract cross at different levels in the ventral white commissure and end in arborizations about cell-bodies in the ventral horns on the opposite side. The direct pyramidal tract also diminishes in size as it descends and commonly ends about the middle of the thoracic portion of the cord.

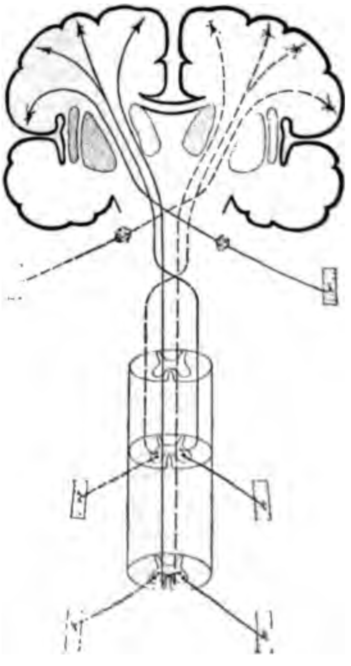


Fig. 120.—Diagram of motor path, showing the crossing of the path in the upper segment.

Motor impulses originating in the right cerebral cortex cause muscular contractions upon the left side of the body, while those starting from the left side of the brain cause contraction of the muscles upon the right side of the body. As a rule, to which there are few exceptions, the motor paths are crossed chiefly at the decussation of the pyramids and to a less extent at different levels of the cord by fibres given off from the direct pyramidal tracts to the cell-bodies of the opposite side. This crossing in either case is in the upper motor segment.

The Lower Motor Segment.—The cell-bodies and processes of the neurons of the lower motor segment lie in the nuclei of the cerebral motor nerves and in the various levels of the ventral horns of the spinal cord. The axis-cylinder processes of the neurons of this segment leave the spinal cord in the ventral roots and pass in the peripheral nerves to the muscles of the body, in which they end in brush-like arborizations in the motor end plates. These neurons, in contradistinction from the neurons of the upper motor segment, which are crossed, are direct, that is, the cell-bodies, their protoplasmic processes, and the muscles to which their axis-cylinders are distributed are upon the same side of the body.

The Segments of the Spinal Cord.—The spinal nerves are connected with the lateral surfaces of the cord by fan-shaped bundles of anterior and posterior roots which are collected into compact strands as they are assembled to form a common trunk. That portion of the cord to which the root fibres of a spinal nerve are attached constitutes its cord segment, the limits of which correspond to the interval which separates the extreme fibres of the nerve and those of the adjacent nerves. The spinal cord is thus seen to consist of a series of segments, each of which gives origin to the anterior or motor and receives the posterior or sensory root fibres of one pair of spinal nerves. These nerves, commonly numbering thirty-one pairs, are eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal. In the cervical region all the nerve-roots but the eighth emerge above the vertebra, while throughout the thoracic, lumbar, and sacral regions the roots for each segment of the cord leave the spinal canal below the vertebra of corresponding number. Owing to the fact that the vertebral column increases in length to a greater extent than the cord, there is a progressive disparity from above downwards between the cord segments and their respective vertebræ. In point of fact the segment corresponds to the nerve which is connected with it, and not to the level of the vertebra opposite to it. The position of a lesion involving a particular spinal segment is therefore, except in the upper cervical region, some distance above the vertebra of corresponding number. Ziehen has formulated the following rule to determine the levels of origin of the cervical and thoracic nerve-roots: For the cervical nerves subtract one from the number of the nerve, and the remainder will indicate the corresponding spinous process; for the upper thoracic nerves (I–V) subtract two; for the lower thoracic nerves (V–XII) subtract three. Axis-cylinder processes from more than one segment of the cord may enter into the formation of a peripheral nerve and the greater number of the long striped muscles are supplied with nerve-fibres from more than one segment.

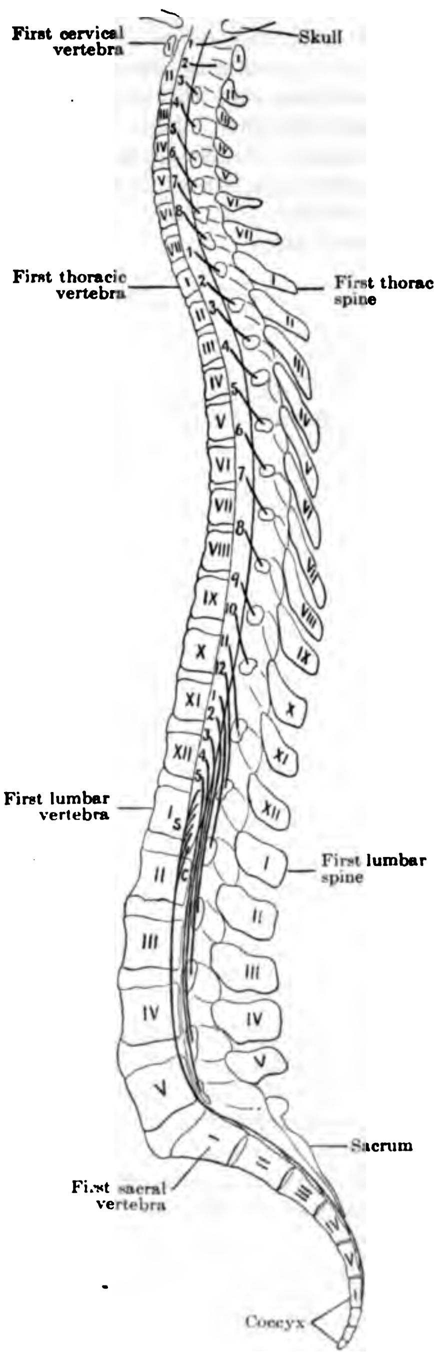


FIG. 130.—Diagram showing relations of bodies and spines of vertebræ to levels at which spinal nerves escape from vertebral canal.—Piersol.

The cutaneous distribution of the peripheral nerves has been accurately worked out and is of diagnostic value in lesions of the main trunks and their ramifications. The segmental areas which correspond to the dorsal roots, though less definitely determined, are sufficiently known to be of great service in the segmental localization of lesions of the dorsal roots and the cord. These skin-fields or dermatomes have been mapped out as the result of observations by Henry Head in the distribution of the cutaneous lesions of herpes zoster and the areas of referred pain and tenderness corresponding to certain visceral lesions, and in cases of gross lesions of the cord by Starr, Kocher and others; as the result of studies of anæsthesia under similar conditions; and of morphological investigations, anatomical dissections, and experimental physiological researches. The skin areas upon the trunk form irregularly parallel zones, somewhat horizontal in the erect posture, and even more irregularly distributed elongated tracts

THE LOCALIZATION OF THE FUNCTIONS IN THE SEGMENTS OF THE SPINAL CORD.

Based upon the studies of Starr, Edinger, Wuhmann, and others.

Segment.	Muscles.	Reflex.
I, II, and III Cervical	Splenius capitis, trapezius, hyoid muscles, diaphragm (C. III-V), sternomastoid, levator scapulæ (C. III-V)	Diaphragmatic.
IV Cervical..	Trapezius, scaleni (C. IV-T. I), rhomboid, diaphragm, teres minor, levator scapulæ, supraspinatus	Dilatation of the pupil (C. IV-VII).
V Cervical ...	Diaphragm, rhomboid, biceps, supinator brevis (C. V-VII), teres minor, subscapularis, brachialis anticus, pectoralis (clavicular part), supra- and infraspinatus (C. V-VI), deltoid, supinator longus (C. V-VII), serratus magnus	Scapular (C. V-T. I), supinator longus (C. V), and biceps (C. V-VI).
VI Cervical ..	Teres minor and major, biceps, supinator brevis, coraco-brachialis, extensors of wrist (C. VI-VIII), infraspinatus, brachialis anticus, pectoralis (clavicular part), pronator teres, deltoid, supinator longus, serratus magnus (C. V-VIII), triceps (outer and long heads)	Triceps and posterior wrist (C. VI-VIII).
VII Cervical..	Teres major, pectoralis major (costal part), pronators of wrist, flexors of wrist, subscapularis, pectoralis minor, triceps, latissimus dorsi (C. VI-VIII), deltoid (posterior part), serratus magnus, extensors of wrist and fingers	Scapulohumeral and anterior wrist (C. VII-VIII).
VIII Cervical	Pectoralis major (costal part), latissimus, pronator quadratus, radial lumbricales and interossei, flexors of wrist and fingers	Palmar (C. VII-T. I).
I Thoracic ...	Lumbricales and interossei, thenar and hypothenar eminences (C. VII-T. I)	
II to XII Thoracic	Muscles of back and abdomen, rectus abdominis (T. V-T. XII), transversalis (T. VII-L. I), erectores spinæ (T. I-L. V), external oblique (T. V-XII), intercostals (T. I-T. XII), internal oblique (T. VII-L. I)	Epigastric (T. IV-VII), abdominal (T. VII-XII).
I Lumbar	Lower part of external and internal oblique and transversalis, psoas major and minor (?), quadratus lumborum (L. I-II), cremaster	Cremasteric (L. I-III).
II Lumbar ...	Psoas major and minor, sartorius (lower part), iliacus, flexors of knee (Remak), pectineus, adductor longus and brevis	
III Lumbar ..	Sartorius (lower part), inner rotators of thigh, adductors of thigh, abductors of thigh, quadriceps femoris (L. II-L. IV)	Patellar tendon (L. II-IV).
IV Lumbar ..	Flexors of knee (Ferrier), abductors of thigh, quadriceps femoris, extensors of ankle (tibialis anticus), adductors of thigh, glutei (medius and minor)	Gluteal (L. IV-V).
V Lumbar ...	Flexors of knee (hamstring muscles) (L. IV-S. II), flexors of ankle (gastrocnemius and soleus) (L. IV-S. II), outward rotators of thigh, extensors of toes (L. IV-S. I), glutei, peronæi	
I to II Sacral.	Flexors of ankle (L. V-S. II), intrinsic muscles of foot, long flexor of toes (L. V-S. II), peronæi	Foot reflex (S. I-II), plantar (S. II-III).
III to V Sacral	Perineal muscles, levator and sphincter ani (S. I-III)..........	Vesical (L. IV-V) and anal (S. I-III).

upon the extremities. The technic consists in the use of a blunt instrument, as the head of an ordinary toilet pin, in determining the presence of areas of abnormal sensation and defining their boundaries.

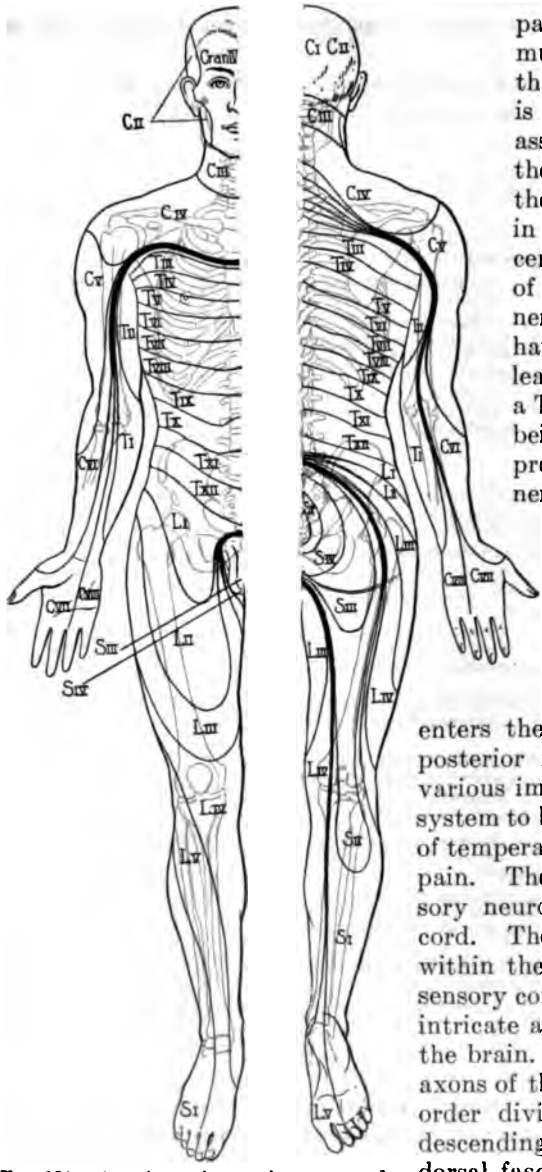


FIG. 131.—Anterior and posterior segmental skin-fields.

The Sensory System.—The path for sensory conduction is much more complicated than that for motor conduction and is composed of three or more associated neurons, one above the other. The cell-bodies of the lowest neurons are situated in the ganglia of the sensory cerebral nerves and the ganglia of the dorsal roots of the spinal nerves. The latter ganglia cells have a single process which, after leaving the cell-body, undergoes a T-shaped division, one portion being the peripherally directed process or dendrite (sensory nerve) which conducts impulses from the integument, mucous membranes, muscles, tendons, and joints of parts of the body with which it may be related; the other the axon or axis-cylinder process which enters the spinal cord by way of the posterior root fibre and conveys the various impulses to the central nervous system to be transformed into sensations of temperature, touch, muscle-sense, and pain. The larger number of the sensory neurons lie outside of the spinal cord. The portions of those neurons within the cord constitute the paths of sensory conduction, which become more intricate as the various tracts approach the brain. Upon entering the cord the axons of the sensory neurons of the first order divide into an ascending and a descending branch which run in the dorsal fasciculi. The short descending branch, after giving off a number of collaterals, terminates in the gray matter of the cord. The ascending branch is of variable length. It may soon terminate in the gray matter or may reach to the nuclei of the medulla. The lower sensory neuron does not cross the middle line. The cell-bodies about which the axis-cylinders of the neurons of the first order and their collaterals end are the sensory neurons of the second order. The axis-cylinder processes of many of those cells cross to the opposite side of the cord and run in the ventrolateral ascending column of Gowers and the ground bundles. The lemniscus is probably the principal sensory tract in the medulla, pons, and cerebral peduncles. The fibres are not, however, continued directly to the cerebral cortex but terminate about cells in the ventrolateral portion of the optic thalamus, from which point the path of sensory conduction is continued by a higher order of neurons, the processes of which terminate in the postcentral and parietal convolutions of the cortex. Other but less direct sensory paths lie in series of neurons in the gray matter of the cord and in the direct cerebellar tract and the tract of Gowers, and pass onward through the cerebellum. Some of the axis-cylinder processes of the sensory neurons of the first order and their collaterals terminate in arborizations about the cell-bodies of the lower motor neurons and thus complete the path for reflexes.

It is probable that the conduction paths for cutaneous sensory impulses reach the opposite side soon after entering the cord, and that the paths for muscular sense lie upon the same side of the cord in the tracts of Goll, crossing by way of the axons of the second order in the medulla.

Fibre Tracts of the White Matter of the Cord.—Of these there are three sets: (1) Those which enter the cord from the periphery, viscera, and other parts of the body; (2) those which enter it from the brain; (3) those which have their origin in the cell-bodies of the neurons which lie within the cord. The fibres which arise from the same group of nerve-cells or nucleus have the same function and a similar destination and proceed together in the same course, thus constituting a tract, column, or fasciculus. Some of these fibres are the pathways for the transmission of impulses from lower to higher levels, and the strands which these form constitute ascending tracts, while others which convey impulses from above downward enter into the formation of descending tracts. These tracts are not sharply defined, nor do their boundaries and areas, since they are subject to increase and diminution by the continual accession or departure of nerve-fibres, remain the same at different levels of the cord. In fact the borders of those tracts often overlap. Their anatomical differentiation has been accomplished partly by the study of degenerative processes caused by experimental methods—Wallerian degeneration—and partly by researches in myelination at progressive periods of development—embryological method. Pathologically they are differentiated in a corresponding manner by the degenerations which follow traumatism of the cord and the definite and constant reaction of certain tracts to pathogenic influences as in tabes and other diseases.

Relation of Diseases of the Cord to Lesions of the Fibre Tracts.—In tabes and Friedreich's ataxia the posterior columns are principally involved; in combined sclerosis the posterior columns and lateral pyramidal tracts; in lateral sclerosis the lateral pyramidal tracts; in amyotrophic lateral sclerosis the lateral pyramidal tracts and the anterior horns, and in anterior poliomyelitis and progressive muscular atrophy the anterior horns.

Sensory Areas of the Cerebral Cortex.—The cortical representation of sensory stimuli is less definite than that of motion. It lies posterior to the fissure of Rolando and is extensively distributed over the post-central and parietal convolutions.

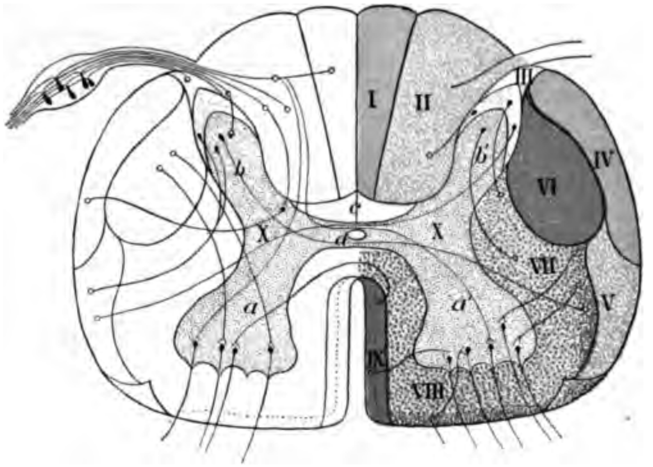


Fig. 132.—Diagram of spinal cord, showing the relation of the principal tracts.

I. Goll's or postero-internal column—fasciculus gracilus. *Termination.*—Fibres end around neurons of gray matter of cord or in nuclei of medulla. *Function* —Sensory impulses from muscles, tendons and joints of same side. Degeneration followed by ataxia and loss of muscle sense.

II. Burdach's or posterolateral column—fasciculus cuneatus. *Termination.*—Nucleus cuneatus in the medulla; Clark's column. Collaterals to neurons of posterior horn. The root fibres passing to Clark's column traverse the middle and median part of this tract. *Function.*—Tactile impulses from opposite side. Various afferent impressions of muscle sense, heat, cold and pain. Degeneration causes pain, anæsthesia, ataxia, and loss of reflexes.

III. Lissauer's tract or marginal zone. This fasciculus is situated immediately dorsal to the inner side of the posterior horn. Composed of some of the more external root fibres which do not enter Burdach's column. Fibres of small size and short course. They penetrate the substantia Rolandi and end in arborizations about its cells and those of the caput cornu.

IV. Direct cerebellar tract—fasciculus cerebellospinalis. *Termination.*—Ascending path of the second order conveying impulses from Clark's cells to the cerebellum. *Function.*—Impulses from viscera, which probably influence maintenance of equilibrium.

V. Gowers's tract—fasciculus anterolateralis superficialis. *Termination.*—Sensory pathway of second order connecting cord with cerebellum and probably with cerebrum. Fibres are chiefly axons of neurons in the posterior horn, partly upon the same and partly upon the opposite side. Boundaries not well defined. *Function.*—The conveyance of sensory impulses—tactile pain and temperature—from opposite side by way of the anterior commissure.

VI. Lateral or crossed pyramidal tract—fasciculus cerebrospinalis lateralis. *Termination.*—Fibres are axons of cortical motor neurons. They extend from superficial gray matter of cerebrum to various levels of cord, undergoing decussation at lower part of medulla. *Function.*—Conveyance of motor impulses of brain.

VII. Lateral ground bundle—fasciculus lateralis proprius. *Terminations.*—Composition very complex. Long descending paths; one long ascending strand and many short strands both ascending and descending. *Functions.*—Both motor and sensory. Connects, by means of its intersegmental association fibres, different levels of the cord and forms a direct sensory link between cord and higher centres—medulla and cerebrum.

VIII. Anterior ground bundle—fasciculus anterior proprius. Constitutes with lateral ground bundle, with which it is continuous, a single anterolateral tract or fundamental column. Its composition and functions are the same as those of the lateral ground bundle.

IX. Anterior or direct pyramidal tract—fasciculus cerebrospinalis anterior. *Termination.*—Composed of pyramidal fibres which do not undergo decussation in medulla oblongata. Made up of 15 to 20 per cent. of pyramidal fibres. Almost all fibres cross in anterior white commissure at successive levels to terminate in arborizations about root cells of anterior horn of opposite side. *Function.*—Motor tract from cerebral cortex.

X. Gray matter of the cord. *a*, *a'*, anterior horns; emergences of anterior motor root fibres; *b*, *b'*, posterior horns; entrance of posterior root fibres; *c*, posterior commissure; *d*, anterior commissure. *Function.*—Anterior horns motor; posterior sensory. Cells of anterior horns trophic; those in angle of posterior commissure probably influence automatic movements while those near by are trophic vasomotor, and secretory.

Of the foregoing, I, II, and III comprise the fibre tracts of the posterior column; IV, V, VI, and VII the fibre tracts of the lateral column, and VIII and IX the fibre tracts of the anterior column of the cord.

The Cortical Areas for the Special Senses.—The individual sensory paths terminate in circumscribed regions which are as a rule widely removed from one another. As mapped out by myelination these areas correspond to regions of the cortex which pathological lesions have shown to be related to the various special forms of sensation. According to Flechsig olfactory fibres end mainly in the uncinate gyrus; visual fibres have been traced to the occipital lobe in the region of the calcarine fissure, while auditory fibres run to the temporal lobe.

It is in accordance with these observations that the cuneus and calcarine fissure together constitute the primary or lower cortical visual centre in which are represented the opposite visual half fields, while the outer surface of the occipital lobe contains centres for higher visual processes in which the vision of the eye of the opposite side is represented. Mind blindness results from a destructive lesion of the lateral lobe in the left hemisphere if both occipital lobes are involved. A lesion of the cuneo-calcarine cortex results in lateral homonymous hemianopsia. The centre for memory of the meaning of printed words, letters, figures, and objects seen is probably in the left angular gyrus. A destructive lesion in this area is attended by inability to read or comprehend written language although ordinary vision is not impaired. This area is known as the visual speech centre. The auditory centre is in the upper temporal convolution and transverse temporal gyri and it is in this region upon the left side that the memories of the meaning of heard words and sounds are stored. A special centre for musical memories lies anterior to the auditory centre. It is probable that the centre of each side is connected with both auditory nerves. The olfactory centre probably comprises a portion of the base of the frontal lobe and the uncinate gyrus. The gustatory centre has been thought to be in the anterior portion of the gyrus fornicatus near the centre for smell. Our knowledge in regard to these two centres is not definite.

The centres for the "higher psychical functions" are generally assumed to lie in the prefrontal lobes, particularly upon the left side. Extensive unilateral lesions of the anterior portion of the frontal lobe may be present without causing marked symptoms of any kind. Atrophy of this portion of the brain is often marked in various forms of dementia.

Symptoms due to derangements of the motor tracts constitute the most important group of localizing phenomena. They are objective on the one hand and are upon the other caused by lesions of conduction paths that are comparatively well understood. Lesions involving the motor path are irritative or destructive. The greater number of the lesions of the motor cortex are at the same time destructive and irritative. They destroy the nerve-cells and their processes in a particular centre and by their presence and advance stimulate those of adjacent centres into morbid or disordered activity. The clinical manifestation of a destructive lesion of a motor centre is loss of function—paralysis; that of an irritative lesion abnormal muscular contraction. Important differences in the paralysis or abnormal contraction are dependent upon the position of the lesion as regards the motor segments. These differences are due first to anatomical relations and second to secondary degenerations.

The cortical motor centres are more or less widely separated from one another, and a circumscribed destructive lesion of the motor area may therefore give rise to a limited paralysis involving a limb or a group of muscles in a limb—cerebral monoplegia. As the axis-cylinder processes converge to form the pyramidal tract in the internal capsule, a lesion of limited extent causes paralysis of most of the muscles upon the opposite side of the body—hemiplegia. A lesion in the pyramidal tract as it descends, giving off fibres to the motor nuclei at various levels, causes paralysis of the muscles having their spinal centres below the seat of the lesion. It follows from the decussation of the pyramids that when the lesion is above the crossing the paralysis is upon the opposite side of the body, and when it is below it, upon the same side.

The cell-body and particularly its nucleus maintain the nutrition of all parts of the neuron. If the cell-body be destroyed its processes undergo degeneration, or if any process be separated from its cell-body it likewise undergoes degenerative changes throughout its whole extent—secondary degeneration. Degeneration of the axons of the upper motor segment ceases, however, at the lower motor segment. The muscles are paralyzed but do not undergo degenerative atrophy; they are spastic; their reflexes are exaggerated and they do not show qualitative changes in their electrical reactions.

In complete transverse lesion of the cord—complete spinal paraplegia—the muscles upon both sides are paralyzed below the lesion, but they are flaccid; the deep reflexes are abolished; the muscles undergo rapid atrophy with loss of faradic excitability.

Irritative lesions of the upper motor segment involving the motor cortex give rise to the convulsive phenomena known as cortical or Jacksonian epilepsy.

Destructive lesions of the lower motor segment cause degeneration alike of the axis-cylinder processes in the peripheral nerves and of the muscle-fibres with which they are connected. The anatomical distribution of the cell-bodies of the segment gives rise to special peculiarities in the distribution of the paralysis which are strongly in contrast to that resulting from lesions of the upper motor segment and which have important bearings upon the localization of the lesion. These cell-bodies are collected in groups or nuclei from the peduncles of the brain throughout the entire extent of the spinal cord and send axis-cylinder processes to all the muscles of the body. Certain groups of the neurons which make up the lower segment are therefore widely separated, and a circumscribed lesion may result in paralysis of a limited number of muscles or a group of muscles instead of one-half of the body as in upper segment paralysis—hemiplegia. A lesion causing lower segment paralysis may be situated either in the cord or in the peripheral nerve. If in the cord or its nerve-roots the paralyzed muscles are not supplied by a single nerve but are represented in adjacent cord segments and the accompanying sensory derangements involve the skin fields related to those segments; if on the contrary the lesion is in the nerve, the paralyzed muscles and the anæsthetic area are those supplied by that particular nerve and its branches. The neurons of the lower motor segment maintain not only the nutrition of their axis-cylinder processes which make up the peripheral nerves but also that of the muscle-fibres in which their processes terminate. The degeneration which results from injury of the cell-bodies or their processes involves the muscles to which they are distributed. In lower motor segment paralysis the affected muscles are the seat of degenerative atrophy, manifest in diminished tension, abolition of their reflexes and reaction of degeneration—flaccid paralysis.

Irritative lesions of the lower motor segment cause fibrillary contractions which may be due to stimulation either of the cell-bodies or of their axis-cylinder process in the peripheral nerves; or they may give rise to spasmodic contractions when the lesion affects the motor nerve-roots as they emerge from the cord.

Symptoms due to derangements of sensory paths are of far less localizing value than motor symptoms. This is partly due to the greater complexity of the sensory tracts, partly to less exact knowledge concerning them. If sensory symptoms are limited to the distribution of a peripheral nerve it is evident that the lesion is in the nerve-trunk or its branches; if restricted to the fields corresponding to one or more spinal segments the cord is at fault; if they chiefly affect one side of the body, the brain. The nature of the sensory phenomena has little value. Intense pain, for example, may be symptomatic of peripheral nerve disease as in some forms of neuritis, or of a degenerative process within the cerebrospinal axis as in tabes.

Irritative lesions cause disordered subjective sensations of heat, cold, formication, and the like—the paræsthesias—and pain of every variety as to kind and degree.

Destructive lesions, if they completely interrupt the sensory path, wholly abolish sensation in the parts of the body involved. A lesion of a peripheral sensory neuron in the course of the nerve gives rise to anæsthesia in the area of distribution of the nerve; a complete transverse lesion of the spinal cord gives rise to total loss of sensation of all parts below its level. Destructive lesions of the central nervous system do not however usually interrupt all the sensory conduction paths, and sensation may not be wholly abolished even in extensive disease. Sensation may be diminished or lost in all its phases as in complete transverse lesions of the cord, or there may be dissociation sensory paralysis as in certain diseases of the cord in which pain-sense and temperature-sense are abolished while tactile sensation remains unimpaired, or in some lesions of the cerebral cortex in which there may be a loss of the muscular sense and astereognosis—the loss of the ability to recognize an object placed in the hand—while other phases of sensation are fully preserved.

EXAMINATION OF THE PATIENT.

The Anamnesis.—An accurate history of the case is of the highest importance in disease of the nervous system. This must include in many cases the facts relating to the antecedents of the patient, which bear upon hereditary predisposition, as the occurrence of nervous or mental disease in the parents, children, or collateral members of his family. Peculiarities, idiosyncrasies, and psychoses are especially to be ascertained, often a matter of no little difficulty. A history of gout, alcoholism, or syphilis in a parent, when it can be obtained, may give the key to the situation.

The investigation of the personal history must bear upon any previous serious illness and its nature, whether nervous or not, and especially whether or not such an illness was of a similar nature to that from which the patient is suffering.

It may be necessary to follow in our investigation a chronological order, ascertaining whether or not nervous symptoms have occurred in infancy and childhood, such as convulsions, enuresis, night terrors. The period of school life is to be studied in obscure cases. The neurasthenic may have been bright and successful at school, but shy, retiring, and not disposed to make friends; the sufferer from *petit mal*, sometimes confused and forgetful; the hysterical girl, especially at puberty, nervous and emotional. The occupation is next to be considered. Is it one that involves continuous monotony, mental strain, extreme responsibility? Have there been prolonged or cumulative depressing emotions, disappointment, fear, sorrow, or grief? Wounds and injuries, alcoholism, and abnormal sexual matters, especially syphilis, are of etiological importance in many neurological cases. Severe infectious processes, particularly enteric fever, may have been the point of departure for visceral and vascular changes which after a time manifest themselves in the guise of nervous disease. Of special importance are such maladies in their relation to postinfective psychoses and neurasthenia. The part played by obscure toxæmias due to chronic gastro-intestinal or other visceral diseases in the etiology of certain spinal cord degenerations is not to be disregarded. Notwithstanding the number of points to be considered the value of the history cannot be measured by its length. On the contrary it is most important to briefly record only the facts which are pertinent and significant.

Status Præsens.—While investigation on every side is necessary for a full understanding of many nervous cases, yet there are certain special paths of approach which experience has taught us lead most directly to a diagnosis in the average case; in other words certain distinctly *neurological methods* of investigation. These methods may be grouped according to the character of the symptoms and signs that each brings into view, the most important being, (1) motor and (2) sensory symptoms; (3) cerebral symptoms, of which, on account of comprehensive and special characters, (4) aphasia requires separate consideration; (5) spinal symptoms in so far as they connect segments of the cord with particular regions of the body; (6) the reflexes; (7) electrical phenomena; (8) trophic disturbances; (9) pain and temperature; (10) muscular sense.

1. Motor Symptoms.

Paralysis.—Motor paralysis signifies impairment of some portion of the motor pathway. When partial it is to be distinguished from *akinesia*, common in states of mental stupor, and from incoördination, often mistaken by the patient and his friends for true weakness. The practical tests for muscular weakness consist, for the hand and forearm, in estimating the patient's "grip" as he squeezes the hand of the examiner, especially in comparing the grip of an affected hand with the other, which may be normal or less affected. Of mechanical devices the dynamometer of Mathieu is most commonly used. The power of arms and legs is tested by having the patient make various movements while the examiner, grasping the part, offers resistance.

A general surmise as to the location of the lesion (cerebral or spinal) causing the impairment of the motor path is made by observing whether the affected part is flaccid or spastic. Flaccidity nearly always denotes a lesion of lower motor neurons (ganglion cells of ventral gray horns, peripheral nerves with their terminals) as seen in poliomyelitis and neuritis, while spasticity signifies a lesion of central or upper motor neurons (cell-bodies of motor cortex, fibre tracts through subcortex, internal capsule, pons, medulla, ventral and lateral pyramidal tracts of spinal cord), as in old brain hemorrhage.

A notable exception to this broad rule is that in lesion of the spinal cord, complete or nearly complete transversely, especially one high up in the cord, the effect is as if all motor neurons below it were destroyed, *i.e.*, there is total flaccid paralysis below the level of the lesion. The explanations of this phenomenon are numerous but unsatisfactory. Another exception to this rule is readily correlated with it by bearing in mind that the superior motor neurons of the pyramidal tract are not wholly cerebral but have a spinal portion which is mostly contained in the lateral tract; hence it follows that a spinal palsy is spastic if the lateral tracts are involved.

To decide whether a member be flaccid or spastic, all the patient's active movements, including gait, are to be studied, as well as various passive motions which may suggest themselves to the examiner. His opinion will be rather one of judgment than of definite methods.

MONOPLEGIA is a paralysis restricted to one member, whether this be disabled entirely or only in one group of muscles. HEMIPLEGIA, or paralysis of one side of the body, is nearly always due to a brain lesion, and, when so, the upper face will be found unaffected or slightly affected, except in recent cases where the paralysis in the upper distribution of the facial nerve may be very distinct for a time. The slight implication of the upper face is characteristic of a long-standing cerebral hemiplegia. DIPLEGIA—double hemiplegia—occurs particularly in childhood. PARAPLEGIA is a symmetrical paralysis involving the upper or lower limbs, but when the term is used without qualification it refers to paralysis of the lower limbs. The term *brachial paraplegia* is employed to denote paralysis of the upper limbs; *crural paraplegia* that of the lower. It is generally a spinal palsy.

Contracture. — In paralysis of long duration contractures appear which are generally characteristic. Those which arise in spastic paralyses depend upon shortening of the paralyzed muscles, the stronger muscles contracting more than the weaker, and produce such postures of the limbs as are seen in hemiplegia (flexion of elbow, wrist and fingers, adduction of arm to chest, extension of the leg on the thigh, adduction of the knees, extension of the foot and inversion with plantar flexion of toes). According to some investigators the contractures of cerebral hemiplegia are the result of the greater paralysis in certain groups of muscles. The contractures in flaccid paralyses depend upon the unbalanced action of the opposing sound muscles, as seen in the accentuated wrist-drop and foot-drop of old peripheral neuritis, or depend upon the contraction of the paralyzed muscles themselves.

Convulsions and spasm (see Part III, p. 588).

Jacksonian epilepsy (see p. 589).

Athetosis or mobile spasm consists of irregular writhing movements, especially of the fingers but also of the arms and other parts. It is almost pathognomonic of the cerebral palsies of childhood, in which affections the symptom may mislead by being more prominent than the hemiplegia or diplegia which underlies it. Occurring in adult hemiplegics these movements are sometimes called *posthemiplegic chorea,* but are less prominent than the weakness and rigidity of the limb. Athetosis is usually aggravated by voluntary movements, as when the patient attempts to pick up a small object.

Tremor (see p. 592).

Fibrillary tremor or fibrillary twitching (see p. 593).

Tics.—Twitching simultaneous over a large area, inducing a purposive movement at intervals and habitually, is called a "tic." It is not a sign of any known lesion but is functional (a neurosis).

Ataxia.—In the course of investigation of motor signs the examiner may observe irregularity and uncertainty in various acts which require a degree of precision. Ataxia results from inharmonious action of muscle-groups even when disorder of motility, either excess or deficiency, is not present. The defect is largely in the muscular sense, which is discussed in its relation to astereognosis. Yet the practical tests for the symptom are motor. In the arm ataxia is discovered by directing the patient to close his eyes and then with his index finger to touch the tip of his nose, or to meet the tip of the other index finger in sweeping the arms around horizontally in front; in the leg, by having him attempt to touch one knee with the heel of the other foot. If there be considerable ataxia the patient touches wide of the mark. Ataxia of the legs is better revealed in the patient's manner of walking, which is considered in connection with other disorders of gait.

2. Sensory Symptoms.

Studies of sensation involve a subjective element which makes them at best uncertain. Scientific methods aim to diminish this uncertainty by magnifying the objective element through the use of technical procedures which render the examiner less dependent upon the patient's statements. In children, and in stuporous and demented patients, the objective element alone is considered—a start, a vocal sound, or the withdrawal of a member when the patient is touched, pricked, etc.

Paræsthesia.—"Numbness and tingling," "pins and needles," "crawling sensations"—formication—and burning sensations are symptoms of sensory irritation. They are prominent in neuritis, and in spinal diseases which implicate the posterior nerve-roots (see also p. 582).

Delayed Sensation.—Recognition of any artificial sensation is, for the purposes of the clinician, instantaneous; if an interval occurs between the application of a stimulus and the patient's response to it, we speak of "delayed sensation," which is common especially in tabes dorsalis.

Pain.—Pain is a prominent symptom of many nervous diseases.

The objective study of sensation comprises the testing of the senses of touch—common sensibility—of pain, and of temperature. The muscular sense is of interest clinically in relation to astereognosis, and also in relation to ataxia of movement, which may arise from defect of the muscular sense.

In testing the sense of touch it is well to blindfold the patient, to take care that the surface examined shall not be chilled by exposure, and to touch the part with light pressure and without causing pain. The instrument most commonly used for this purpose is Carroll's æsthesiometer, but a tooth-pick or a feather will serve. The patient is directed to say "now" when the touch is felt; or to count successive touches a short distance apart, "one, two, three," etc., and the failure to note one or more touches will mark the boundary of an area of anæsthesia. Sensibility to touch is more acute on the back than on the front of the body. Loss of tactile sensibility, either total—*anæsthesia*—or partial—*hypæsthesia*—may be functional and a sign of hysteria, in which case it commonly affects half the body—hysterical hemianæsthesia—or a segment of one limb, or all of one extremity up to a certain level—"glove-anæsthesia" and "stocking-anæsthesia;" or anæsthesia and hypæsthesia may constitute a sign of organic nervous disease which is destructive in character or is at an advanced stage. In the case of hemihypæsthesia or hemianæsthesia, the hemorrhage or other destructive lesion may be in the posterior part of the internal capsule—where according to some anatomists sensory fibres are collected into a bundle (*carrefour sensitif*)—in the tegmentum of the pons, or in the spinal cord, provided one lateral half of the pons or cord be severed. In any of the cases mentioned the lesion is situated on the side opposite to that of the anæsthesia.

In testing the pain sense, a needle-point or one of the sharp points of the æsthesiometer is employed, and the skin is "pricked," not scored, with the instrument. Remind the patient that actual pain, not the mere sense of being touched, is to call forth his response; or instruct him to say "touch," or "pain," according as the one or the other sensation is excited by the sharp point.

The temperature sense is well studied by the use of two test-tubes of water, one heated to about 100° F. or above, the other cooled to 60° F. or lower, the tubes being applied alternately, and each being held in contact with the skin-surface for several moments, since recognition of heat or of cold is commonly less prompt than that of touch and of pain. The heat of the one tube should not be sufficient to burn, as that would introduce the factor of pain; yet practically this distinction is of little consequence, because the thermic sense and the pain sense, being conducted in adjacent tracts of the cord, are commonly abolished together. Ordinarily when tactile anæsthesia has been demonstrated in a certain area, we may expect to find thermo-anæsthesia and analgesia associated with it. But the converse of this does not always hold true; for over surfaces which betray no tactile anæsthesia, or at most only hypæsthesia, we may find areas of analgesia and thermo-anæsthesia. This is that *dissociated* sensory loss which is most common in syringomyelia, though other lesions of the central part of the gray matter of the cord may cause the phenomenon.

3. Regional Diagnosis of Cerebral Disease.

General Symptoms.—The general symptoms of intracranial disease—vomiting, headache, and optic neuritis—have little value in cerebral localization. Headache is more likely to be frontal in lesions of the fore-brain and occipital in those in or about the cerebellum, but this is not constant. Dense tumors of some size, well above the base of the skull, may yield a shadow on the X-ray plate.

Predominant mental symptoms are suggestive of lesion of the prefrontal lobes, particularly the left; but it must be remembered that after head injuries delirium, confusion, or stupor may ensue from shock, without reference to severity or site of the trauma, and moreover that demonstrable brain lesions are comparatively rare causes of insanity.

Paralysis.—Of motor signs indicating lesion of the precentral convolution, anterior to the fissure of Rolando, paralysis has the greatest localizing value. Paralyses in the distribution of cranial nerves, especially of several, commonly indicate lesion at the base of the brain. If a single cranial nerve is implicated, the lesion is probably outside of the central nervous system; if one arm or leg is paralyzed, a cortical lesion should be suspected, and this is rendered probable if the paralyzed part is the seat of clonic spasm. Paralysis of the face indicates lesion in the lower third of the Rolandic cortex; paralysis of an arm or leg, lesion of the middle or upper third respectively.

Astereognosis.—Pure motor phenomena point to a lesion anterior to the fissure of Rolando; if the lesion be posterior to this fissure (postcentral) the motor signs are likely to be associated with the phenomenon called astereognosis, which becomes more prominent as the parietal lobe is encroached upon. By study of the "stereognostic sense" which is the physiologic process by which solid objects are recognized by contact, neurologic diagnosis has made a distinct advance. Astereognosis, or want of this sense, may be diagnostic of lesion of the superior parietal lobule. To test for this phenomenon it is well to study separately the several processes by which normally the hand recognizes the shape and size of objects, especially the "spacing sense," the sense of position, and the pressure sense, the last two of which are the chief components of the muscular sense.

The **"spacing sense"** is tested by touching the skin at two points simultaneously, as with the two arms of the æsthesiometer, and observing how near together they may be while still recognized as two points. The examiner compares his results with those obtained in a normal subject.

The **sense of position** is studied by asking the patient (blindfolded) to tell where his hand or foot is, after the examiner has quietly placed it in a particular attitude, or to imitate with one limb an attitude given to the other by the examiner.

The **pressure sense** is tested by blindfolding the patient, placing his hand supine upon a table, and laying in his palm, one after another, small objects identical save in their weight, which is graded in a series. For this purpose cartridges filled with layers of cotton and regulated numbers of buckshot may be used. The main test, which reveals astereognosis directly if it be at all pronounced, consists in handing the patient various common objects,—watch, spool, block of wood, penknife,—each of which he essays to name or to describe.

Deafness, in the absence of disease of the external, middle or internal ear, may be due to lesion of the first or second temporal convolution, particularly that of the left side.

Blindness without demonstrable cause in the eye may be due to lesion anywhere in the course of the optic nerves, tracts or "radiations" as far as the cunei lobes, which face one another across the great longitudinal fissure in the occipital lobe. Unilateral blindness of both eyes and in the same side of each eye (lateral homonymous hemianopsia) indicates that the lesion is unilateral, that it is back of the optic chiasm and is on the side opposite to that on which the patient's vision has failed—that is, on the same side as the blind half-retina. To determine how far back of the chiasm such a lesion is we must rely on signs and symptoms arising from involvement of contiguous structures, especially (in lesions at the base of the brain) the cranial nerves, which are spared in lesion of the optic radiations, or of the cuneus—subcortical or cortical lesions. A theoretically positive means of distinguishing basal from cortical lesions causing hemianopsia is WERNICKE'S PUPILLARY-INACTION SIGN, which consists in the absence of the light reflex of the iris when only the blind half of the retina is illuminated. The finding of this condition points to a basal lesion, *i.e.*, at or below the optic thalamus and external geniculate body, for contraction of the iris is a function of the third nerve, and no part of this nerve extends above the "primary optic centres," which are at the base of the brain.

Partial loss of vision, not accounted for by eye disease, may be due to lesion of the angular gyrus, visual acuity—macular vision—being impaired; or it may be due to lesion in front of the optic chiasm, in the angle between the optic nerves, where by interfering with the internal fibres of each nerve it causes blindness of each inner (nasal) half-retina, a condition called (from the blind half-fields) temporal hemianopsia, which is pathognomonic of lesion in the situation described.

Symptoms of Cerebellar Disease.—The cerebellum is to the clinician chiefly an organ of coördination, and this function resides mainly in the middle lobe. The cardinal signs of cerebellar disease are *nystagmus* and a peculiar ataxia which gives a staggering character, or *titubation,* to the patient's gait. This ataxia disappears when the patient lies down, and the knee-jerks are often preserved. Neoplasms beneath the middle lobe of the cerebellum are likely to cause this form of ataxia together with external ocular palsies from pressure upon the nuclei of the third and fourth nerves beneath the quadrigeminal bodies. A tumor arising from these bodies can hardly be distinguished from cerebellar tumor implicating the vermis.

The Internal Capsule.—Of the great interior structures of the brain only the posterior limb of the internal capsule has functions so definite that certain symptoms may be referred to it. Sudden hemiplegia, with hemianæsthesia and hemianopsia, is generally indicative of lesion in the internal capsule, since this complex of symptoms from cortical or even subcortical lesion could be induced only by uncommonly extensive damage. The symptoms referable to single minute destructive foci in the posterior limb of the capsule, from the "knee" backwards, are, so far as is known, (1) paralysis of the face from above downwards, (2) of the arm and (3) of the leg, also from above downwards, (4) anæsthesia of varying extent up to hemianæsthesia, which probably indicates destruction of the posterior third of the posterior limb, (5) hemianopsia.

Cerebral Ganglia.—Of the great cerebral ganglia none has an independent symptomatology. Lesions affecting the corpus striatum cause predominant motor signs because of pressure upon the motor bundles of the capsule, while affections of the optic thalamus commonly cause hemianæsthesia from pressure upon the posterior fibres of the capsule—*carrefour sensitif*—or destruction of sensory fibres within the thalamus and often hemianopsia from involvement of the optic radiations, which are collected into a bundle posterior to the capsule and enter the optic thalamus. Mobile spasm or athetosis, associated with these paralyses, is in favor of thalamic lesion. Weakness of the articulatory muscles resembling bulbar paralysis, but not due to lesion of the medulla oblongata, is called pseudobulbar paralysis. It is most often due to multiple hemorrhages or softening in the outer part of the lenticula.

Lesions of the corpus callosum are revealed by disturbance of the functions of surrounding parts, notably of the motor zone,—as shown by early epileptic seizures, by paralyses, and symptoms referable to the prefrontal region. From the latter arise the pseudoparetic mental states which are characteristic of callosal lesion.

4. Aphasia and Other Defects of Speech.

Though endowed with a normal brain, the individual born deaf and blind becomes an imbecile by deprivation of the sense-impressions out of which knowledge grows, unless he be trained like Laura Bridgman through the touch-sense. The cochlea, the retina, etc., begin the transformation, from mere contact with the external world, into the higher special sense-impressions. These, carried by their separate paths to the cortex, are elaborated in the special-sense centres into perceptions of things. Roughly speaking, each cortical centre is opposite the organ of that sense. Taking one sense, vision, rays of light from an object, for example a cow, received by the retina are carried through the visual system to the cuneus as sensations of form, color, etc. Thence passing still higher, in the angular gyrus is formed a visual image of a cow—object-seeing—and this is associated with an image of the word cow written or printed—word-seeing. Lesion of angular gyrus then does not cause ordinary blindness—as lesion of the cuneus does in one half-field—but loss of these visual images, so that the patient seeing a cow can hardly tell it from a horse—object mind-blindness; and seeing the word cow fails to get the meaning from it, as if it were a foreign word—word-blindness. In like manner, close to the auditory centre is a higher centre for the formation of auditory images, by which a peculiar sound is identified, for example, as the lowing of a cow—object-hearing—and by which the spoken word cow is recognized as the name of that animal—word-hearing. With a lesion then in the first temporal convolution, sparing the main auditory centre, so that the patient is still capable of hearing noises, there may be loss of these auditory images with consequent object-deafness and word-deafness; sounds and words heard are meaningless.

To speak of a cow it is necessary to recall the word cow. Many persons are likely to recall a word as it sounds; some as it appears written; but most revive it in both ways, so that impairment of either the auditory or the visual word-image interfering with the recollection of words causes aphasia, in the one case, from lesion of the angular gyrus—visual or optic aphasia; in the other, from lesion of the first temporal convolution—auditory aphasia. In either case due to a lesion of a sensory centre it is spoken of as sensory aphasia; and because the essential defect is inability to recollect words (verbal amnesia) both are included under the term amnesic aphasia. The act of speaking involves several groups of muscles, and is interfered with, therefore, in various forms of paralysis. In bulbar paralysis, the lips, tongue, etc., becoming atrophied and paretic, there is defect of articulation, incidentally; and in the similar paralysis from cerebral lesion (pseudobulbar paralysis) the patient may be inarticulate. In like manner lesion in the cortical centres for the lips, tongue, etc., at the foot of the motor zone, cripples the speech just as lesion in the leg centre causes limping; so here on the emissive side of the speech-process there is set apart a higher centre for the fine adjustment of movements in uttering words, and for the memory of these movements. This is Broca's centre, in the posterior part of the third frontal convolution. By lesion here, the muscles of articulation still intact, the patient loses his motor memories and his power to utter words. This is motor aphasia—or aphemia.

Parallel to these defects of articulation are defects in the act of writing which has its higher centre in the second frontal convolution, related to the arm centre as Broca's is to the centres for the tongue, lips, etc. Lesion in the writing centre causes motor agraphia, even though the arm be still useful otherwise. In lesion of the angular gyrus, as the appearance of words is forgotten, writing is imperfect; there is sensory agraphia. In reading aloud, the image of the printed word must be conducted from the angular gyrus to Broca's centre, there to be matched with the motor image used in uttering the word; and the utterance must be guided, too, by the auditory image conducted from its centre. For this purpose Broca's centre is connected with the others by tracts of fibres which being damaged, particularly in the insula—island of Reil, there is interference with the conduction referred to, and hence, with reading aloud and with similar uses of speech, conduction-aphasia. For perfect speech all the centres must act in unison through conduction-paths connecting each centre with the rest, and considering such multiple connections it is evident that aphasia of some kind may result from lesion at any point within a wide area. This "zone of language" is nearly coextensive with the distribution of the middle cerebral artery, and aphasia is generally a consequence of apoplexy from this vessel, commonly in association with hemiplegia. This same region, acting as a unit, forms a complete image not alone of the word but of the object also, as it looks, sounds, feels, smells, tastes—in short, a concept of the object; so that this is a concept area (Mills).

Aphasia being a curtailment of the power to comprehend as well as of the power to emit language, spoken, written or by signs—pantomime—care and system in testing for it are very important. "Impediments" of speech, mechanical imperfections of the vocal organs, are first to be eliminated by examination of the mouth, throat, and nasal cavities. In cleft palate, hypertrophic rhinitis, and in tongue-tie, the difficulty is mainly in the enunciation of consonants, such as m, n, b, etc.

Dysarthrias, from paralysis or defective innervation of the muscles of articulation, are to be recognized partly by finding additional signs of cerebral paralysis or other organic nervous disease, and partly by special characters of the speech in certain affections. Somewhat suggesting mechanical impediment is the speech of bulbar paralysis, marked as it is by labored pronunciation of consonant-sounds.

Elision of syllables by running words together, may be observed in hereditary ataxia, in which disease speech is at the same time monotonous. These two characters belong also to the speech of general paresis, forming with the difficulty of enunciating the r's and l's, as in "artillery," and with its tremulous, measured drawl, the peculiar "paretic speech" which is one of the cardinal signs of this disease. The measured character of such speech exists in purer form—scanning—in disseminated sclerosis.

Ordinary stuttering is a pure neurosis. It manifests itself by spasmodic halting in attempts to utter certain words, usually those beginning with consonants.

In differentiating aphasia from other speech defects the greatest difficulty arises in the case of actual mental loss—dementia—which indeed may coincide with aphasia, as in hemiplegia and senility, or may have aphasia for an episodic manifestation, as in paresis.

The stubborn speechlessness frequently met with in paranoia and melancholia is nearly always accompanied by other signs of negativism, as refusal of food and resistance to the attentions of the nurse. Hysterical aphasia is intermittent and its victim exhibits the stigmata of the neurosis.

In testing an apparent aphasic it is well to begin on the sensory side, ascertaining whether the centres for word-hearing and word-seeing are impaired. A number of common objects may be placed before the patient who endeavors to pick out those named in turn by the examiner and then to select from a list of names on paper, that of the object selected by the examiner.

As the purpose is to determine the clearness of word-images, these simple tests are essential; but the examiner may progress to words and sentences of any complexity. Rarely being complete, aphasia is often betrayed by persistent, helpless misapplication of words, the patient saying or writing for instance "dog" when a hat is showed to him and its uses demonstrated by him. Paraphasia and paragraphia are forms of aphasia rather characteristic of sensory aphasia.

To test a patient's emissive power of language, that is, to discover motor aphasia and agraphia, objects are shown to him, and he endeavors to utter and write their names. Simple acts performed in the patient's presence are described by him both orally and in writing. In motor aphasia "recurring utterances" are common, a patient repeating "any one any" or other meaningless phrase on all occasions when attempting to talk.

Even after demonstrating that a patient hears, reads, utters, and writes words correctly, and thus that the widely separated cortical speech-areas in the first and second left temporal convolutions, the angular gyrus, the third frontal and the second frontal convolutions are probably intact, together with the subcortical region corresponding to each centre, we may still find that the patient is much crippled in the use of language. In such a case we conclude that some of the fibres connecting these cortical centres with one another are cut off, and that we are dealing with a case of "conduction aphasia" or "transcortical aphasia." The prominent features of this form are paraphasia, paralexia, etc., so extreme that the jumbling of words and syllables is spoken of as "jargon-speech."

So entangled in these "association-systems" are all the cortical centres that speech-defect from cortical lesion always betrays some features of conduction aphasia. If our tests reveal uncomplicated word-deafness or word-blindness, or simple loss of the power of utterance, we recognize that the lesion is deep in the brain, beneath the level of association-systems, where the fibres radiating to or from the particular centre are bundled together. This is called "subcortical" or "pure" aphasia, and yet some evidence exists that this form of aphasia may result from cortical lesions.

As an auxiliary test for this form, the study of pantomime is of value. In pure motor aphasia, for instance, the patient though speechless as regards utterance, may when asked how old he is, open and shut his hand the proper number of times. In ordinary motor aphasia this is impossible.

5. Spinal Localization.

One of the consequences of modern clinico-pathological study is the tendency to interpret nervous symptoms and signs in relation to anatomical structure, rather than in relation to empirical disease forms. This tendency in the field of brain disease has created cerebral localization; and it has affected our conceptions of spinal disease to the extent that we speak less of "locomotor ataxia," of "spastic paraplegia" or of "progressive muscular atrophy" as disease entities than as dominant symptoms of various lesions affecting certain structures of the spinal cord. Assuming in this connection that the symptoms in a given case are of spinal origin, we infer from "ataxia" of a limb that the dorsal column of the cord is affected; from spasticity with increased reflexes, the pyramidal tract; from atrophy, the ventral horns of the gray matter; from anæsthesia, the dorsolateral column again; from loss of pain- and temperature-sense without anæsthesia—dissociation of sensation—the central part of the gray matter; from pain, the dorsal roots. Then we endeavor to determine the lesion which has caused the particular symptom-complex which confronts us by bringing to bear our knowledge of the natural history of nervous disease and by collating the spinal symptoms with any cerebral manifestations which may be present. By this method we may find that our "locomotor ataxia" case is really one of combined degeneration of the cord or one of paresis, and that "progressive muscular atrophy" is symptomatic of syringomyelia or of tumor.

Spinal localization in the ordinary sense, however, relates to diagnosis of the level of a lesion in the cord. It is based upon our accumulated knowledge of the motor sensory reflex and sympathetic control exercised by each segment of the spinal cord over a corresponding segment of the body.

Injury to the spinal cord at any point involving the motor tracts—unless it be completely severed—causes paralysis, with increase of reflexes, below that point; but at the level of the lesion we are likely to find the reflexes abolished. We commonly find also anæsthesia covering the body below this level if the lesion is grave, and the upper limit of anæsthesia, with the zone of absent reflexes coinciding, is the best index to the level of the spinal lesion. If the lesion affects one lateral half of the cord the above principles still apply, but the disturbance of sensation, except of the sense of position, is found on the side opposite to that of the lesion and to that of the motor symptoms—Brown-Séquard's paralysis, although even in this form tactile sensation is often preserved.

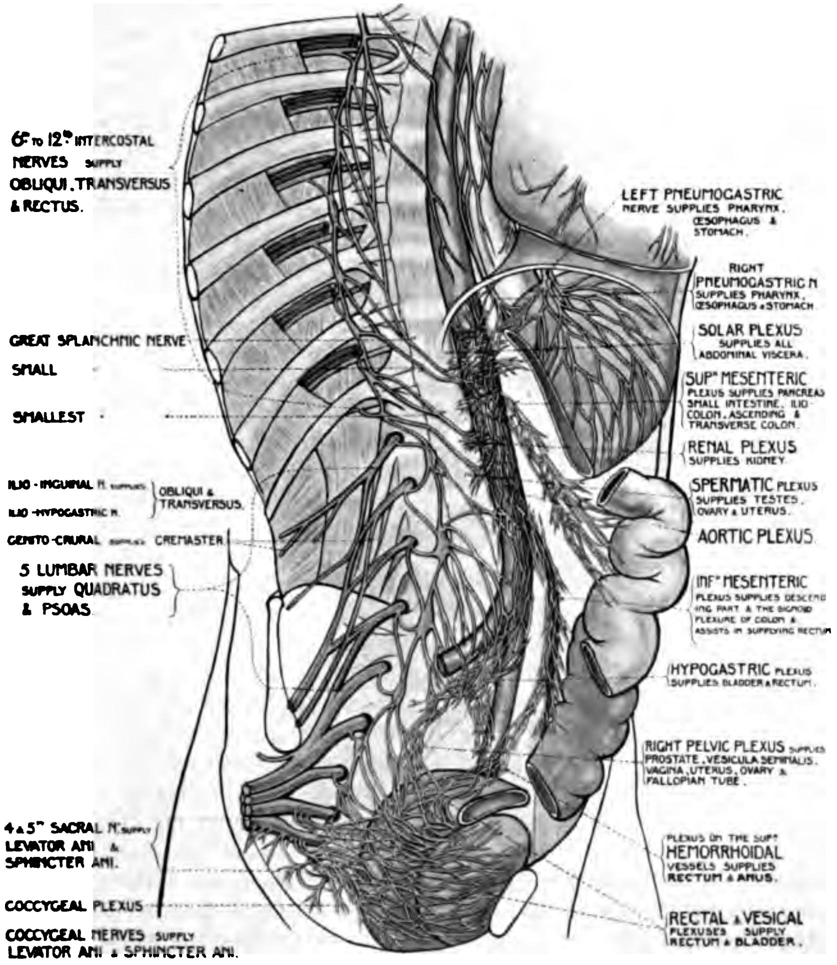
The level thus ascertained marks the relative position of the lesion, but its actual position in the spinal column will be found above this, generally a distance of about three spinal segments. A narrow zone of anæsthesia is usually present in Brown-Séquard's paralysis on the side of the lesion, and at its level and above this may be a narrow zone of hyperæsthesia. Such an anæsthetic zone occurring independently points to a lesion outside the cord substance and involving spinal roots of at least two segments. Sensory loss from injury to the cord proper or the posterior roots is distributed in horizontal bands about the trunk and longitudinal bands in the limbs, irrespective of the distribution of the nerves—segmental anæsthesia.

The clinician should be able to conclude off-hand from atrophy of the shoulder, or loss of reflexes in that region, that the upper cervical region is affected when the symptoms are of spinal origin; from such symptoms affecting the forearm and hand, that the lesion is lower down in the cervical swelling; from loss of knee-jerk, that it is in the lumbar, and from loss of control of sphincters, in the sacral region; but for finer deductions it is well to record the findings in a particular case, and then interpret them by reference to the tables and diagrams upon pp. 312, 313, and 314.

Combined Degenerations.—"Typical cases" are as narrow summits in the great ranges of disease. From each summit the symptomatology and pathology form a downward slope, by which that disease merges with one or more of its neighbors. Of lateral sclerosis very few absolutely pure cases have been reported. On the one hand, in cases that seem like pure lateral sclerosis, there is nearly always insidious degeneration in the ventral gray horns—chronic poliomyelitis; or the latter disease after a course of years may take on spastic symptoms because the pyramidal tracts are invaded, that is, degeneration beginning in either motor neuron tends to progress to the other.

In some cases the affection of superior and inferior motor neurons is simultaneous, progressive muscular atrophy and spastic paraplegia developing *pari passu*. Such cases constitute amyotrophic lateral sclerosis. In them the bulbar part is prominent and degeneration may extend even to the cortex, mapping out the motor zone, for amyotrophic lateral sclerosis is a disease of the whole motor system.

PLATE VII.


6th TO 12th INTERCOSTAL NERVES SUPPLY OBLIQUI. TRANSVERSUS & RECTUS.
GREAT SPLANCHNIC NERVE
SMALL
SMALLEST
ILIO-INGUINAL N. SUPPLIES OBLIQUI & TRANSVERSUS.
ILIO-HYPOGASTRIC N.
GENITO-CRURAL SUPPLIES CREMASTER
5 LUMBAR NERVES SUPPLY QUADRATUS & PSOAS.
4 & 5th SACRAL N. SUPPLY LEVATOR ANI & SPHINCTER ANI.
COCCYGEAL PLEXUS
COCCYGEAL NERVES SUPPLY LEVATOR ANI & SPHINCTER ANI.
LEFT PNEUMOGASTRIC NERVE SUPPLIES PHARYNX, ŒSOPHAGUS & STOMACH.
RIGHT PNEUMOGASTRIC N. SUPPLIES PHARYNX, ŒSOPHAGUS & STOMACH.
SOLAR PLEXUS SUPPLIES ALL ABDOMINAL VISCERA.
SUPR MESENTERIC PLEXUS SUPPLIES PANCREAS SMALL INTESTINE, ILIO-COLON, ASCENDING & TRANSVERSE COLON.
RENAL PLEXUS SUPPLIES KIDNEY.
SPERMATIC PLEXUS SUPPLIES TESTES, OVARY & UTERUS.
AORTIC PLEXUS.
INFR MESENTERIC PLEXUS SUPPLIES DESCENDING PART & THE SIGMOID FLEXURE OF COLON & ASSISTS IN SUPPLYING RECTUM
HYPOGASTRIC PLEXUS SUPPLIES BLADDER & RECTUM.
RIGHT PELVIC PLEXUS SUPPLIES PROSTATE, VESICULA SEMINALIS, VAGINA, UTERUS, OVARY & FALLOPIAN TUBE.
PLEXUS ON THE SUPR HEMORRHOIDAL VESSELS SUPPLIES RECTUM & ANUS.
RECTAL & VESICAL PLEXUSES SUPPLY RECTUM & BLADDER.

Connection between sympathetic nerves supplying viscera and spinal nerves supplying muscles of abdominal walls.

On the other hand degeneration in the lateral columns may be accompanied by dorsal degeneration. Such implication of *superior motor* neurons with *inferior sensory* neurons in combined degeneration suggests a local, extraneous cause, acting upon the lateral and posterior columns simultaneously. The mechanism for this could be the marginal system of arteries carrying some toxin into these portions of the white matter, and the lesion might be expected to spread around the arterioles from the very margin of the cord. These conditions are clearly present in some cases of ergotism, pernicious anæmia, etc. An acute diffuse dorsolateral degeneration may be found at autopsy, though commonly no symptoms have been observed in life. In various undetermined toxæmias (Putnam and E. W. Taylor) a subacute degeneration is established, partly diffuse, but also partly systematic, as in time it enters the course of ascending and descending tracts. This subacute combined degeneration is clinically distinct from the other types (Russell, Batten and Collier). Possibly similar in origin but appearing as a pure combined system disease (Strümpell), the chronic form, posterolateral sclerosis, has been recognized longer. It is manifested clinically by paraplegia from the pyramidal tract lesion, with ataxia from the lesion in the dorsal columns—ataxic paraplegia of Gowers—but without lightning pains or other sensory phenomena and without eye-symptoms, because the sensory root-zones and roots—including the optic nerves—are spared. For this last reason, too, the reflexes are preserved in posterolateral sclerosis; by the degeneration of the pyramidal tracts they are usually increased and the legs made spastic. At a late stage the root-zones may be invaded and reflexes impaired until the case appears like one of simple tabes dorsalis, only an autopsy revealing the combined lesion.

A combined sclerosis is the commonest spinal lesion of paresis.

6. The Reflexes.

Every segment of the spinal cord contains not only centres for certain groups of muscles but also for reflex movements. The reflex starts in an impulse arising from the stimulation of a sensory nerve. It is transmitted to a centre in the cord and passes by way of the processes of the sensory cell-bodies to the neurons of the corresponding motor centre, in which it originates a motor impulse which in turn passes by way of the motor nerve to the muscle-fibres supplied by the nerve. This complete path, made up of centripetal or sensory fibres with their cell-bodies and correlated cell-bodies with their centrifugal or motor fibres, is called a reflex arc. The sensory impulse may be transmitted to centres at higher or lower levels and excite several motor impulses, thus producing a complicated reflex arc. The cord segments are connected with fibres from the cerebrum having the function of inhibiting the reflex. If these fibres are irritated the reflexes are impaired from abnormal inhibition; if they are destroyed the reflexes are exaggerated. If the arc is interrupted either in its afferent or efferent limb or in the centre the reflex is lost.

Involuntary contraction of muscles aroused by a sensory impression upon related parts is a reflex in the ordinary sense. For fine deductions the muscles themselves must be observed. The quadriceps cruris, for example, may be seen to contract on tapping the patellar tendon, even when no motion of the leg occurs, and under such circumstances the "knee-jerk" cannot be said to be abolished; but ordinarily we recognize reflex response in muscles by a characteristic motion imparted to a member, as the kicking movement of the leg which is regarded as a measure of the knee-reflex.

Absence of the usual motor response, of the knee-jerk for example, or its diminution or exaggeration, are the matters to be attended to in the study of most reflexes, particularly the "tendon-reflexes." This is true also of most of the superficial—skin—reflexes, though in certain of them the character of the motion elicited is significant; thus with the plantar reflex, flexion of the toes is normal, while extension—Babinski reflex—indicates lesion of the pyramidal tract of the corresponding side, extension of the toes being equivalent to exaggeration of other reflexes. In a third group, the so-called periosteal reflexes, any motion of the member establishes the presence of the reflex, as in the case of the scapulohumeral, the motion of which may be external or internal rotation, and ad- or abduction of the upper arm, according as to which of the muscles attached to the scapula are most actively excited when this bone is jarred by tapping at a spot where it is bare save of periosteum and skin.

A reflex must be fairly constant and discernible in the normal subject to give much significance to its alterations, particularly to its absence. Many reflexes are of minor clinical importance because they are present in only a small percentage of normal subjects and then are not pronounced, the ulnar for instance. Reflexes of the lower extremity are on the whole more important than those of the upper, and the knee-jerk is preëminent in this respect.

The reflexes of the upper extremity being inconstant, absence of any one of them signifies little; exaggeration of one has a certain value; and even the marked presence of a number of them in a patient has something of the import of exaggeration of other reflexes.

Knee-jerk or Patellar Tendon Reflex.—To elicit the knee-jerk the leg is rendered passive by crossing the knee over its fellow, or by supporting it on the examiner's forearm passed under the patient's knee and braced by the hand placed upon the other knee, or by having the patient while recumbent draw up his knee into an easy position with all muscular tension on his part withdrawn; the patellar tendon well below the knee-cap is then struck a firm, quick blow with the ulnar edge of the hand or with a percussion hammer.

The knee-jerk should never be declared absent until Jendrassik's method of reinforcement has confirmed the result. This is applied by directing the patient to hook his hands together and to keep them so while tugging at them as if to pull them apart. It is customary for the examiner to count "one, two, three" after instructing the patient to "pull hard" at "three," the tap on the tendon being made at about "four."

The signs + for increased and — for diminished knee-jerks are commonly employed; and, in writing, "kj" for the reflex itself is allowable.

The knee-jerk being due to contraction of the quadriceps cruris muscle, the essential phenomenon may be induced by tapping the muscle itself just above the patella, especially if the latter is pressed downward by a finger laid along the upper edge of the bone and this finger is then tapped with the hammer.

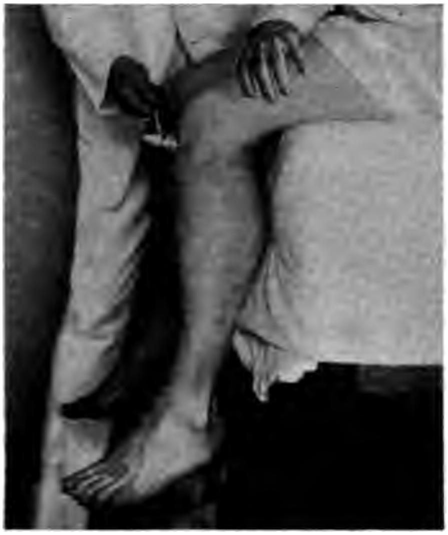


FIG. 133.—Method of testing patellar reflex.

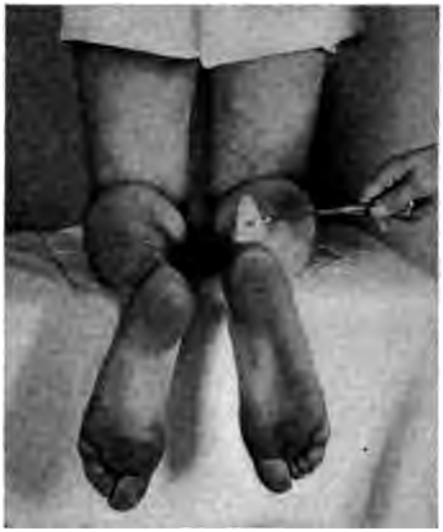


FIG. 134.—Achilles tendon reflex.

Babinski Reflex.—In testing for the Babinski reflex the examiner supports the patient's ankle with his left hand and strokes the sole of the foot with any object which makes a distinct sensory impression—a somewhat sharp point being necessary when the skin is thick—at the same time noting the movement of the toes, which in all normal persons past the age

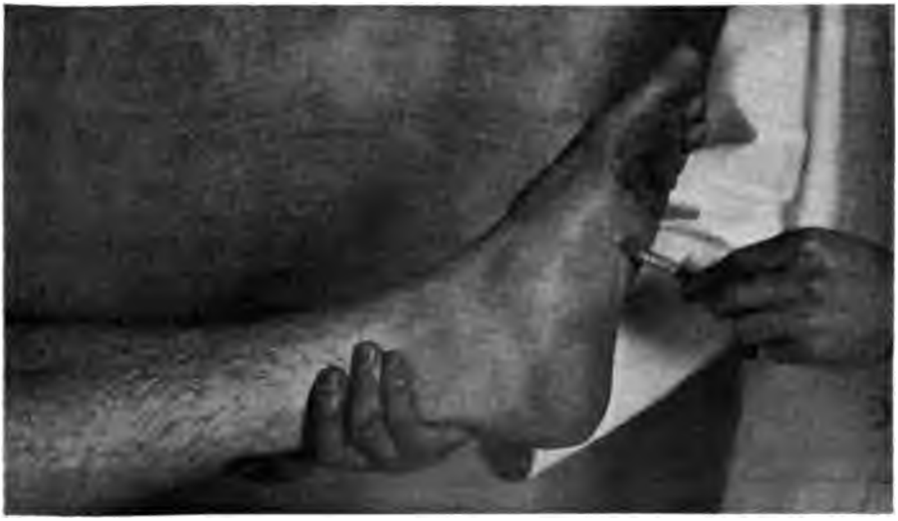


FIG. 135.—Plantar flexion.

of infancy is plantar flexion. Extension (dorsiflexion) of the toes, particularly of the big toe, elicited in this way constitutes the Babinski reflex, which is a most important sign of involvement of the pyramidal tracts.

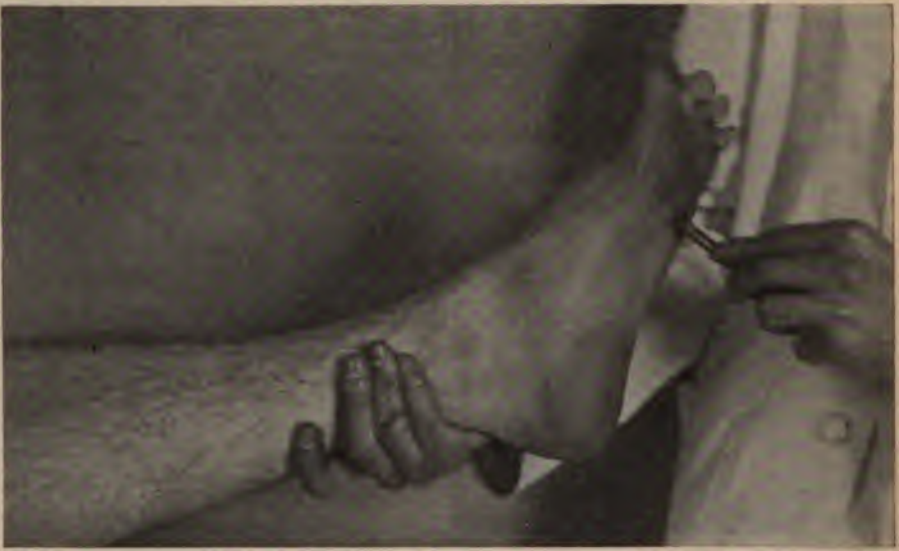


FIG. 136.—Babinski reflex (dorsiflexion of the toes).

Ankle Clonus.—This phenomenon usually accompanies a considerably increased knee-jerk, and has a similar significance. To test for it, the whole leg should be relaxed—best by having the patient supine. The examiner's left hand supports the leg, and his right, clasping the patient's foot, presses it upward, when, if clonus is present, the foot is pushed back against the hand in a series of jerks which are due to clonic spasm of the (soleus) muscle.

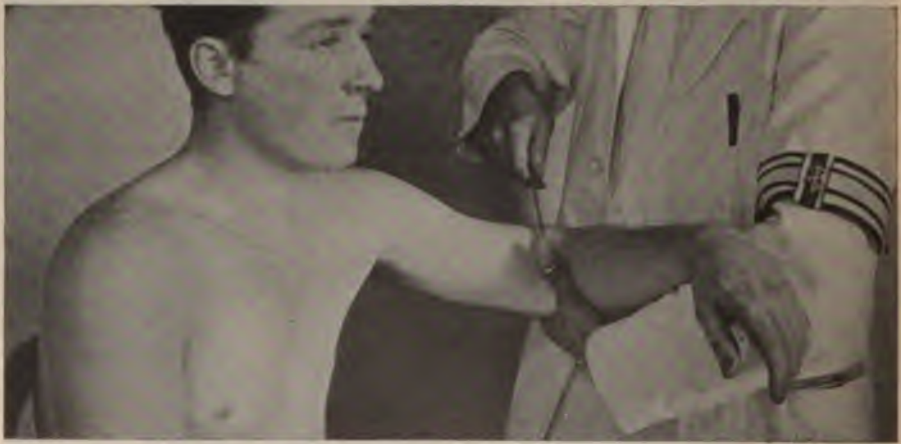


FIG. 137.—Biceps reflex.

Achilles Jerk.—A single reflex-contraction of the calf muscle may be induced by tapping the tendon, which the examiner has rendered tense by pressing the foot upward. This reflex is called the Achilles jerk and is an index of the condition of the sciatic nerve and corresponding segments of the spinal cord. It is best obtained by having the patient kneel upon a chair while the examiner taps the Achilles tendon.

The Abdominal Reflex.—This reflex, quite a constant one, is elicited by stroking the side of the abdomen. The ensuing contraction is widespread over this region.

The Cremasteric or Inguinal Reflex consists in a drawing-up of the scrotum and testicle on stroking the inside of the thigh.

The Epigastric Reflex.—On stroking along the costal margin the muscles over the pit of the stomach contract.

Among the reflexes of the upper extremity, the **Radial**—a periosteal reflex—is elicited by tapping above the styloid process of the radius, and consists mainly in flexion at the elbow-joint. The nearly identical motion of the **Biceps Reflex** arises when the tendon of this muscle is tapped at the bend of the elbow. In testing the last two reflexes the examiner places his forearm under that of the patient, in order to relax the latter.

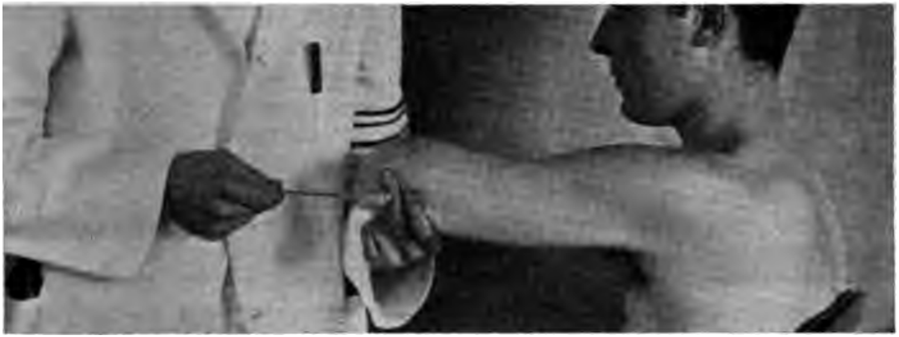


FIG. 138.—Triceps reflex.

To elicit the **Triceps Reflex** the patient's upper arm is given a fixed support on the examiner's wrist or on a chair-back, when tapping above the olecranon causes an outward jerk of the forearm.

Plantar Reflex.—Produced by tickling the sole of the foot. It consists, when fully developed, of sudden withdrawal of the foot by flexion at the hip and knee, dorsal flexion of the ankle and plantar flexion of the toes. The movement in undeveloped cases may consist of sudden plantar flexion of the toes. It occurs in normal conditions, but in varying degrees. There are those who have the power to voluntarily prevent it. This reflex is exaggerated in neurasthenia, hysteria and other functional diseases of the nervous system, and may be associated in extreme cases with general convulsive movements or may be crossed,—that is, it may occur on the opposite side, as well as on the side tickled. It is also increased, but usually to a moderate extent only, in organic disease of the central nervous system. It is as a rule abolished in the affected side in hemiplegia and invariably absent in destructive lesions involving the sensory nerves of the legs.

Other reflexes of minor clinical importance are:

The Supra-orbital Reflex.—Produced by a sharp tap upon the trunk of the supra-orbital nerve, it consists of slight, momentary contractions of the orbicularis palpebrarum, especially in its external half. It is absent in destructive lesions of the supra-orbital nerve and in peripheral facial palsy.

The Malar Reflex.—Not usually present in normal conditions, but caused in recent facial paralysis of peripheral origin by percussion over the malar bone. It consists of contraction of the elevator of the angle of the mouth and movements of the ala nasi.

The Chin Reflex.—This phenomenon is elicited by tapping upon a small flat object, as an ivory paper cutter or a tongue depressor, laid upon the lower front teeth, or the finger laid upon the protuberance of the chin when the mouth is open and the jaw relaxed and drooping. The response consists in a sharp upward movement of the jaw. It may be present in nervous conditions, as hysteria, and in cachectic states.

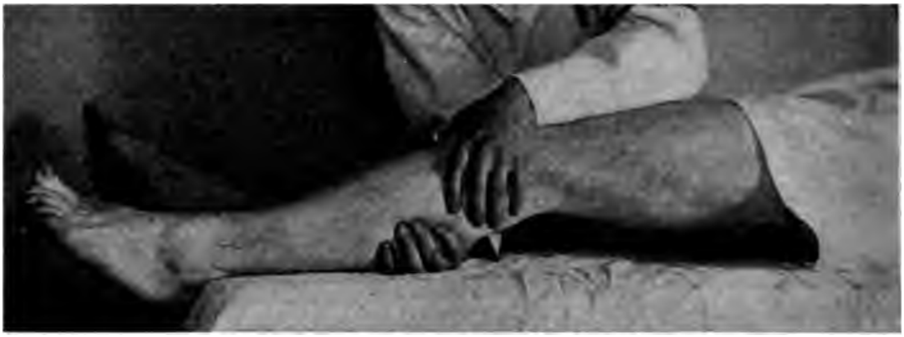


FIG. 139.—Paradoxical reflex.

The Femoral Reflex.—Not present in health. It is produced in transverse lesions of the spinal cord above the level of the eighth dorsal segment by irritation of the anterior surface of the upper part of the thigh, and consists in plantar flexion of the toes and extension of the foot.

Sinkler's Toe Reflex.—Produced by sudden forcible flexion of the great toe. It consists in forcible flexion of the knee and hip and is met with in spastic conditions arising in spinal disease, as spastic paraplegia.

Gowers's Front Tap.—The leg being slightly flexed, a blow is struck upon the tibialis anticus muscle. Plantar flexion of the toes occurs in a considerable proportion of normal persons, many neurasthenic and hysterical individuals, and not at all in tabes.

Paradoxical Reflex.—Caused by sudden shortening of the tendon; elicited by deep pressure upon the calf muscles, and consisting in extension—dorsiflexion—of the toes, especially the great toe. It is regarded as a sign of irritation or early organic affection of the motor pathway.

Oppenheim's Reflex.—Dorsiflexion of the toes and foot upon forcibly stroking the skin along the inner border of the tibia.

Pflüger's Laws.—1. The reflex occurs upon the same side of the body as that to which the irritant is applied and in muscles whose motor nerves arise from the same segments of the cord. 2. If the reflex occurs on the opposite side, only the corresponding muscles contract. 3. If the reflexes are unequal on the two sides, the stronger are on the side upon which the irritation has been applied. 4. When the reflex extends to other segments the direction of the extension is toward the medulla. 5. All the muscles of the body may yield reflexes.

Kernig's Sign.—Not a true reflex but conveniently described in this connection. Normally the leg may passively be fully extended on the thigh, when the latter is at right angles to the long axis of the trunk, as when the patient sits upon the edge of the bed with his legs hanging down, or has the thighs flexed when in the recumbent posture. The extending force must be moderate and gradual. Resistance and pain are developed at an angle between 95° and 135°. This sign occurs in acute meningitis, especially cerebrospinal fever, when collapse symptoms are absent, but is not constant in tuberculous meningitis. It has been variously ascribed to irritation of the meninges and posterior nerve roots, irritative lesions of the pyramidal tract, intraventricular pressure, and cerebellar irritation. It occurs in various acute diseases in young children and very rarely in adults in enteric fever. It may be simulated in old age, disuse of the lower limbs, arthritis, sciatica, and contractures.

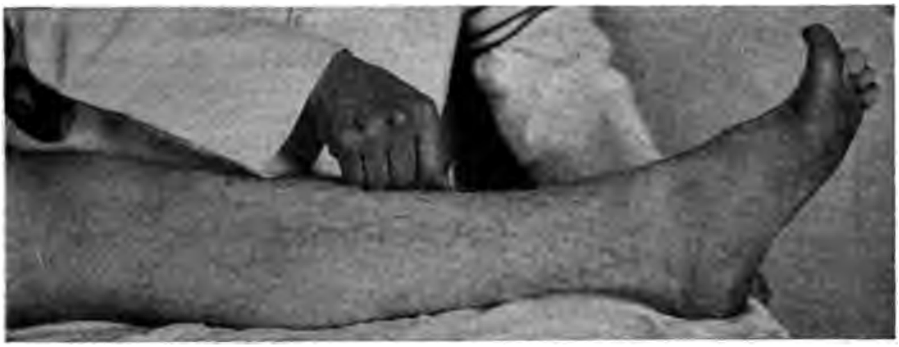


Fig. 140.—Oppenheim's reflex.

Brudzinski's Sign.—If the head is flexed upon the chest, flexion of the legs at the hips and knees occurs and flexion of one thigh upon the trunk causes a movement of the same kind on the other side.

7. Electrodiagnosis.

For diagnostic purposes the galvanic battery is more important than the faradic; but each gives considerable information as to the cause and character of motor paralysis or the variety of muscular atrophy present, and the prognosis in paralysis and atrophy of certain kinds.

The electrodes, covered with absorbent cotton and wetted, are placed upon the patient's bare skin, one at some "indifferent" point, as the back of the neck, the other upon the part to be examined—motor point of the muscles or the nerve-trunks. With a faradic current thus applied, on opening the circuit a quick contraction of the muscles ensues in the region of the distal electrode, whether this be positive (the anode) or negative (the cathode); but if the interruptions are rapidly repeated the muscle is thrown into a tetanic state. If these muscles be the seat of paralysis from lesion of the inferior motor neuron—poliomyelitis, neuritis, etc.—or if they be atrophied, their response to the faradic current is diminished in a degree which, after some experience, can be estimated by the examiner.

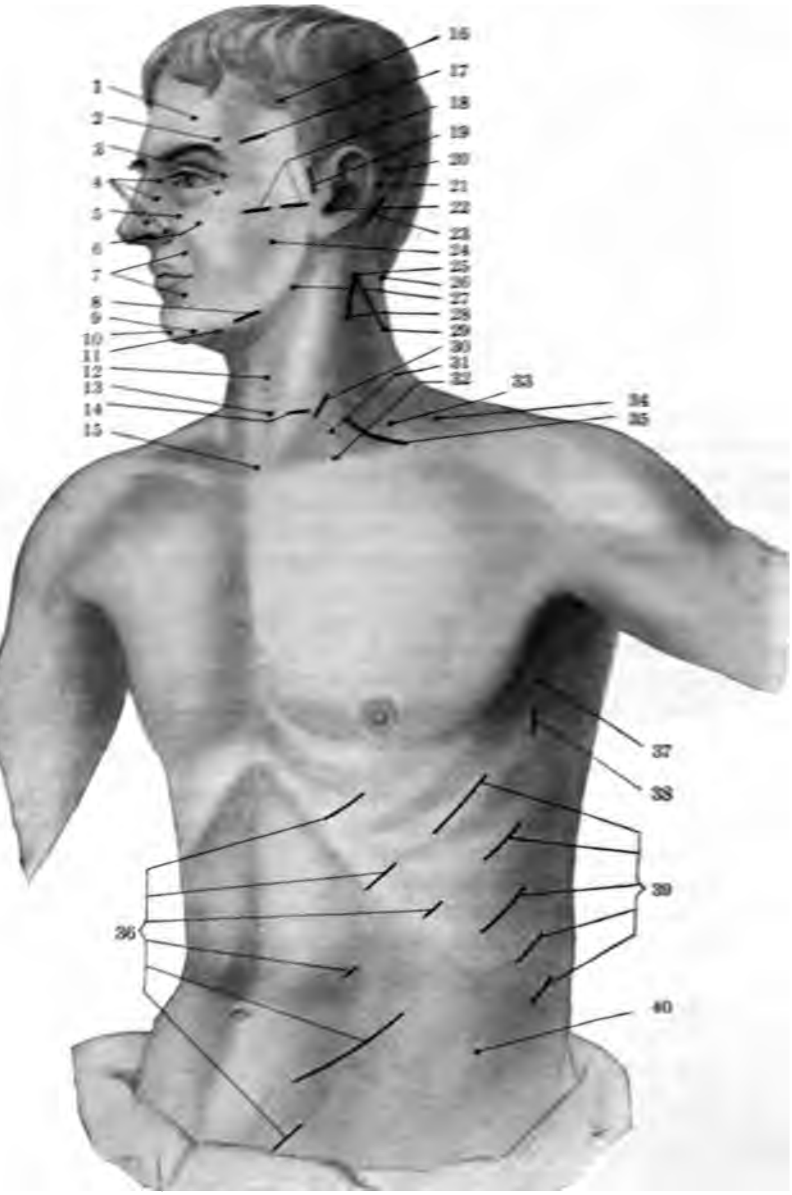


Fig. 141.—Motor points. 1, frontalis; 2, corrugator supercilii; 3, orbicularis palpebrarum; 4, nasal muscles; 5, levator labii superioris; 6, zygomaticus major; 7, orbicularis oris; 8, lower branch of facial; 9, depressor labii inferioris; 10, levator labii inferioris; 11, depressor anguli oris; 12, platysma; 13, sternohyoid; 14, omohyoid; 15, sternothyroid; 16, temporalis; 17, facial nerve, upper branch; 18, facial nerve, middle branch; 19, facial nerve, lower branch; 20, occipitalis; 21, retrahens aurem; 22, facial trunk; 23, posterior auricular nerve; 24, masseter; 25, spinal accessory nerve; 26, splenius; 27, hypoglossal nerve; 28, sternocleidomastoid; 29, trapezius; 30, phrenic nerve; 31, Erb's point (deltoid, biceps, brachialis anticus, supinator longus); 32, anterior thoracic nerve (pectoralis major); 33, circumflex nerve (deltoid); 34, long thoracic nerve (serratus magnus); 35, brachial plexus; 36, rectus abdominis (nervi intercostales abdominales); 37, serratus magnus; 38, latissimus dorsi; 39, obliquus abdominis externus (nervi intercostales abdominales); 40, transversus abdominis.

This diminution of faradic contractility serves as an early index of the extent of paralysis and atrophy which is likely to appear in acute anterior poliomyelitis, in Bell's palsy, or other disease inducing rapid degeneration of muscles; but at the end of two weeks from the onset in these affections there is commonly no response whatever to faradism.

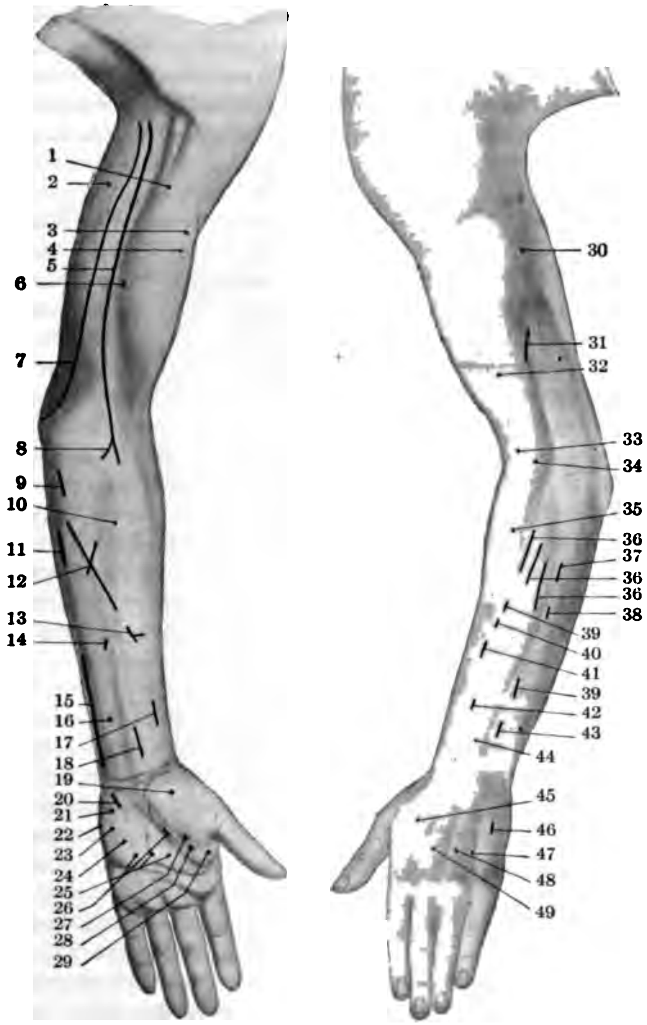


FIG. 142.—Motor points. 1, musculocutaneus; 2, caput internus m. tricipitis; 3, n. musculocutaneus; 4, biceps; 5, medianus; 6, brachialis internus; 7, n. ulnaris; 8, rami n. mediani pro m. pronatore radii terete; 9, palmaris longus; 10, radialis internus; 11, ulnaris internus; 12, flexor digitorum profundus; 13, flexor digitorum sublimis; 14, flexor digitorum sublimis (digitt. II et III); 15, n. ulnaris; 16, flexor digitorum sublimis (digitt. indicis et minim.); 17, flexor pollicis longus; 18, medianus; 19, abductor pollicis brevis; 20, rami volar. prof. nervi ulnaris; 21, palmaris brevis; 22, abductor digiti minimi; 23, flexor digiti minimi; 24, opponens digiti minimi; 25, lumbricales II, III et IV; 26, opponens pollicis; 27, flexor pollicis brevis; 28, adductor pollicis; 29, lumbricalis I; 30, caput externus m. tricipitis; 31, n. radialis; 32, brachialis internus; 33, supinator longus; 34, radialis externus longus; 35, radialis externus brevis; 36, extensor digitorum communis; 37, ulnaris internus; 38, extensor digiti minimi proprius; 39, extensor indicis proprius; 40, extensor indicis prop. et abductor pollicis longus; 41, abductor pollicis longus; 42, extensor pollicis brevis; 43, extensor pollicis longus; 44, flexor pollicis longus; 45, interosseus dorsalis I; 46, abductor digiti minimi; 47, interosseus dorsalis IV; 48, interosseus dorsalis III; 49, interosseus dorsalis II.

On the other hand, if the galvanic current be applied as described above over paralyzed or atrophied muscles the contractility is found to be at first increased; that is, galvanic hyperexcitability is a sign of muscle

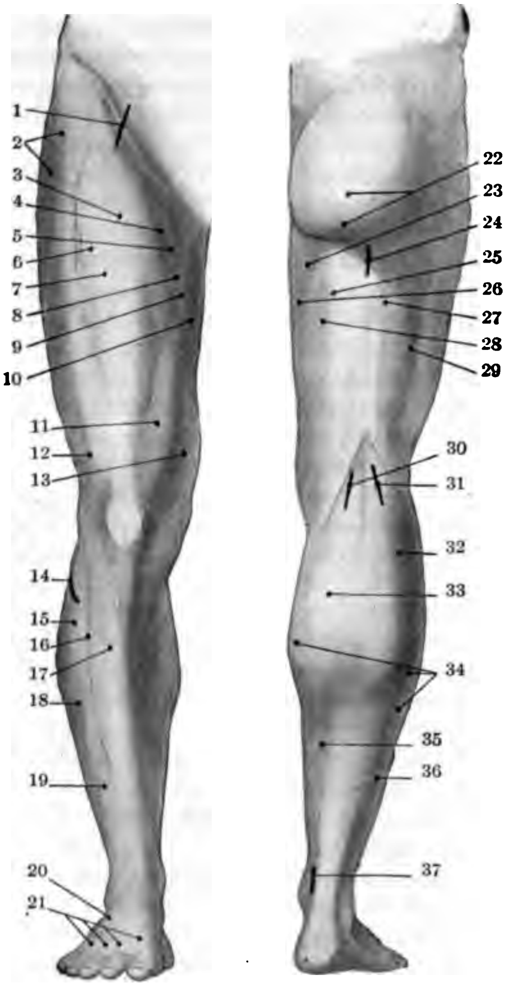


FIG. 143.—Motor points. 1, anterior crural nerve; 2, tensor fasciæ latæ; 3, sartorius; 4, obturator nerve; 5, pectineus; 6, quadriceps (common point); 7, rectus femoris; 8, adductor longus; 9, adductor magnus; 10, gracilis; 11, crureus; 12, vastus externus; 13, vastus internus; 14, external popliteal nerve; 15, peroneus longus; 16, extensor longus digitorum; 17, tibialis anticus; 18, peroneus brevis; 19, extensor hallucis longus; 20, extensor brevis digitorum; 21, dorsal interossei; 22, gluteus maximus; 23, adductor magnus; 24, sciatic nerve; 25, semitendinosus; 26, gracilis; 27, biceps (long head); 28, semimembranosus; 29, biceps (short head); 30, internal popliteal nerve; 31, external popliteal nerve; 32, gastrocnemius (outer head); 33, gastrocnemius (inner head); 34, soleus; 35, flexor longus digitorum; 36, flexor longus hallucis; 37, posterior tibial nerve.

degeneration. Later it diminishes. Contraction of the muscles under the galvanic current is only momentary, appearing both on closing and on opening the circuit. The various responses of the normal muscle are as follows: To the negative pole, or cathode, the first or most active response is on closing the circuit, which is expressed thus, CCC. On opening the circuit there is no response, C O C. To the positive pole, or anode, a response not so active as to the cathode is obtained on closing the circuit, A C C , and occasionally a response is also obtained on opening the circuit, especially if the pole is held on the trunk of a motor nerve, A O C. These two responses to the anode may be about equal, but usually the response at closure is greater than at opening, and neither is as active as the response to the closure of the cathode. Thus the normal formula stands as follows:

C C C > A C C > or = A O C > C O C.

This formula represents what we find practically at the bedside. There are some distinctions between the responses to nerve-tissue on the one hand and muscle-tissue on the other, as observed in laboratory experiments on animals; but these need not detain and confuse us here.

Reaction of Degeneration—R. D.—When a muscle is degenerating—for instance, when it is cut off from its nerve supply either by injury or disease (nerve injuries, neuritis, acute anterior poliomyelitis)—the reactions to galvanism are altered. The anodal closure contraction becomes greater than the cathodal closure contraction, A C C > C C C , although both are diminished as compared with those of the normal muscle. At the same time the anodal opening contraction (never very conspicuous) disappears, and very rarely the cathodal opening contraction is seen. Thus the typical reaction of degeneration is as follows: A C C > C C C (C O C sometimes seen, A O C disappearing). The response of degenerating muscle is sluggish, not quick and active.

8. Trophic Disturbances.

In a broad sense all disease is nutritional disorder; but there are some diseases which directly attack the nervous structures presiding over nutrition of related parts of the body, and these are properly "**trophic diseases.**" The nutritional disorder may be the principal manifestation of the disease, as is indeed recognized in the very name of the group of muscular atrophies. Whether or not there be separate trophic nerve-fibres, we know that for the muscles the trophic impulses traverse the motor nerves chiefly. If motor palsy is accompanied by rapid wasting, the lesion is probably in the gray matter (of the cord, oblongata, etc.) or in the peripheral nerves, since they, comprising the lower motor neurons, preside over nutrition most directly. But slow wasting may affect parts paralyzed by cerebral disease (upper motor neurons), the affected side in old hemiplegia being commonly much atrophied. This is ascribed to involvement of trophic centres in the cortex. While the spastic spinal palsies arise from disease of superior motor neurons—pyramidal tracts—they often manifest atrophy which may be similar in all respects to that of chronic poliomyelitis—ordinary progressive muscular atrophy. In such cases there is no physiological paradox: the atrophy is referable to implication of the gray matter of the cord. Primary lateral sclerosis is practically always accompanied by atrophy, distributed as in poliomyelitis, which implies that the two motor neurons—superior and inferior—are perhaps independently, though simultaneously, involved, and bulbar palsy is frequently included in the clinical picture. It is well, therefore, to conceive of chronic poliomyelitis, lateral sclerosis, amyotrophic lateral sclerosis and bulbar palsy as constituting one disease, of which a particular symptom—atrophy, etc.—is dominant in each of the types named.

The distribution of muscular atrophy has considerable significance, especially the region of the body in which it first appears. Atrophy beginning in the small muscles of the hand, or in the shoulder, is generally progressive—spinal—muscular atrophy.

In the "family type" of spinal atrophy appearing in infancy, the muscles of the legs and back are the first to show wasting. The myopathies or muscular dystrophies are likely to appear first in the pelvic girdle (leg type), in the shoulder girdle (arm type), or in the face (face type). When atrophy occurs in the foot and outer lower leg—peroneal muscles—the so-called primary neuritic atrophy is to be considered.

The cardinal tests of spinal, as distinguished from idiomuscular, atrophies are the electrical reaction of degeneration and fibrillary twitching, both present in the former, and absent in the latter or myopathies.

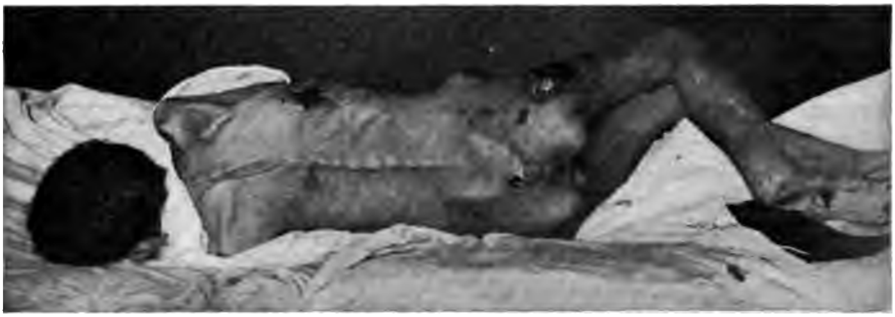


Fig. 144.—Bed-sores.—German Hospital.

The muscles above or below a diseased joint often waste. This is called "arthritic atrophy," and is explained as a reflex phenomenon set up by irritation of sensory nerves supplying the joint.

The clinician must discriminate between the atrophy from disuse or from joint disease, and that which is the essential manifestation of certain grave nervous diseases; and he does so mainly by considering the correlated symptoms and signs.

Certain diseases are presumably, though not manifestly, trophic in origin; as arthritis deformans. Others, such as acromegaly, myxœdema, and adiposis dolorosa, result from disease of ductless glands—pituitary body, thyroid—through the medium of the trophic nervous apparatus, which is affected by the absence or derangement of the secretions of these glands. These diseases—characterized respectively by gross enlargement of hands, feet, and face; by thickened, doughy skin; by great masses of painful fat—illustrate trophic excess, hypertrophy, and hyperplasia.

Still another group of affections, pathologically obscure and clinically indefinite, illustrate trophic disease arising through the medium of vasomotor derangement. These comprise angioneurotic œdema, acroparæsthesia, Raynaud's disease, erythromelalgia, and perhaps other diseases.

A variety of local affections occur however as incidents, more or less ıportant, in the course of organic nervous diseases, and constitute trophic anifestations of these diseases, just as paralysis and anæsthesia constitute

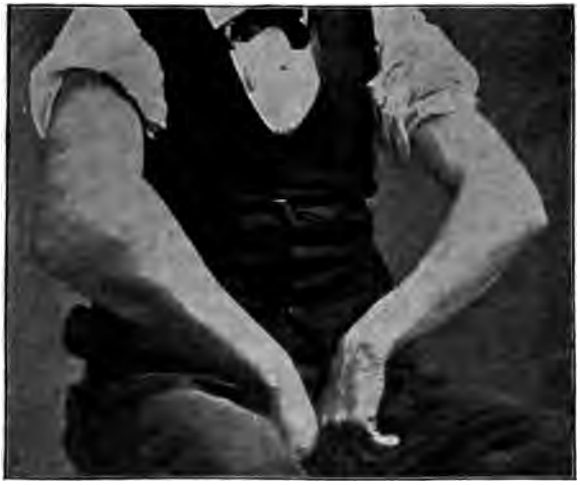


Fig. 145.—Ataxic elbow-joint.—Young.

eir motor and sensory manifestations. These trophic disturbances somenes resemble independent affections and the nervous diseases underlying em may thus be overlooked, for instance, bed-sores in emaciation or rains in Charcot joints. A joint affection, especially if it be subacute or

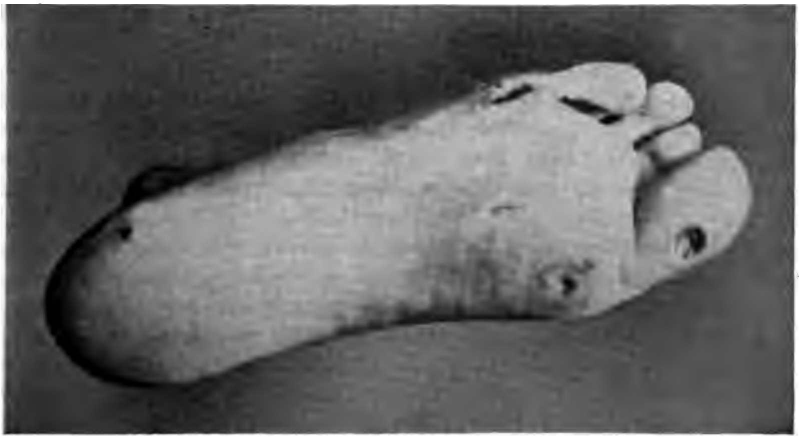


Fig. 146.—Perforating ulcers of the foot.—German Hospital.

ronic and confined to one or two joints, is often of nervous origin. In ute myelitis inflammatory arthropathies, resembling rheumatism, may ise. In old hemiplegia—on the paralyzed side—and in various chronic rd-diseases, an osteo-arthritis is not uncommon. The classic form is e Charcot joint, which occurs most frequently early in the course of tabes dorsalis. The arthropathy of syringomyelia often affects the spine, inducing scoliosis. Painless whitlows of fingers or toes call for study of sensation in these parts, for if they betray loss of temperature and pain sensibility, we are dealing with "Morvan's disease," a trophic manifestation of syringomyelia. Painless perforating ulcer of an extremity, often on the ball of the foot or great toe, belongs commonly to tabes dorsalis or to syringomyelia. Bed-sores form usually at spots injured, especially over the sacrum from pressure of the bed, but the extent of the ulceration is ordinarily out of proportion to the apparent cause. Moreover, sloughs do form without external cause, from purely trophic defect. The skin shows changes in various nervous diseases, as do the nails, hair and other structures histologically allied to the skin. "Glossy skin,"—shiny, thin, dry epidermis on the extremities,—results from neuritis of somewhat long duration. The vesicles of herpes zoster are a trophic manifestation of neuritis, most frequently intercostal.

9. Pain and Temperature.

In ordinary anæsthesia, as that of neuritis or of tabes dorsalis, loss of sensibility to pain, and to heat and cold, is associated with the loss of touch sense. The nerves and dorsal roots, the seat of these diseases, contain the fibres for all forms of sensation.

There is a remarkable condition, however, in which a patient, though feeling himself touched by an object—touch sensation preserved—cannot tell whether it is hot or cold—temperature sense lost—or whether it is sharp or dull—pain sense lost. This separate sensory loss is called *dissociated anæsthesia.* It is in the root-zone that the pain and temperature fibres part company with all others to enter the gray matter and sweep across by way of the commissure to the opposite margin of the cord.

In the neighborhood of the central canal—central gray matter—the pain and temperature fibres from one side decussate with those from the opposite side in a narrow space, and a small lesion at this point, sparing the dorsal columns, may cause dissociate anæsthesia. The lesion that most often occurs here is a peculiar tumor that forms by proliferation of neuroglia just back of the central canal.

When proliferated rapidly neuroglia forms a soft mass. In the brain where its commonest seat is deep in the cerebellum, it meets equal pressure on all sides and so becomes globular—glioma; but in the cord the line of least resistance is up and down, and the gliomatous tissue forms a rod along the centre of the cord. Neuroglia tumors tend to break down centrally. Glioma of the brain is thus commonly cystic, and gliosis of the cord when advanced is characterized by cavity formation within it, by which the cord is finally converted into a tube. From this circumstance the entire disease-process gets its name *syringomyelia.*

Dissociated anæsthesia may result from tumor, hemorrhage in the central gray matter, but it is so early and so constant in gliomatosis that it is commonly spoken of as *syringomyelic* dissociation.

As the neuroglia mass spreads it causes various symptoms, most commonly those of progressive muscular atrophy because the ventral gray horns are slowly invaded. In chronic poliomyelitis it is usually the hands and arms that are atrophied, the cervical and upper thoracic part of the cord being the commonest seat of the gliosis. The pain and temperature fibres from each root-zone, having reached the opposite margin of the cord, turn upward to form the column of Gowers. This column is bounded in front by the motor root-zone, behind by an imaginary line passing transversely through the central canal. A lesion such as tumor severing the column of Gowers will cause dissociated anæsthesia below it on the opposite side of the body. Lateral trauma of the spine is likely to sever this column, with a similar result; but practically such a trauma always implicates also the crossed pyramidal tract, causing motor paralysis on the side of the lesion. The combination of motor paralysis on one side of the body and sensory paralysis on the opposite side, "Brown–Séquard's paralysis," is pathognomonic of unilateral cord lesion. In Brown-Séquard's paralysis touch sense is usually preserved on both sides of the body, the dorsal columns of the cord escaping.

10. Muscular Sense.

Normal coördination depends upon several factors, any one of which being defective, incoördination or ataxia may result. In walking, under normal circumstances, the sensations imparted by the surface control to some extent the movements, and the absence of this control, as in the anæsthesia of tabes, constitutes an element of ataxia. Subconscious sensations from the joints, muscles, skin, fasciæ, together with appreciations of weight and balance, enter into the special kind of perception designated the muscular sense—"sixth sense" of Sir Charles Bell—and defect of this sense is an important factor in most forms of ataxia. It is suppressed at its very source when the nerve-termini in joints and muscles are implicated in a peripheral neuritis, and this causes so marked an ataxia that such cases have been designated peripheral pseudotabes. A part of the ataxia in such cases of peripheral neuritis may be due to anæsthesia of the skin. The ataxia of true tabes has, to some extent, this same peripheral origin, since neuritis is a part of the disease, but it has a more important spinal origin. In the cord many muscular-sense axons pass up the dorsal columns in company with the touch-sense axons, and here they are implicated in tabetic degeneration. Ataxia, by loss of muscular sense and by anæsthesia combined, is a constant symptom of lesion of the dorsal columns.

Muscular sense is represented in the cerebral cortex posterior to the motor area, being associated with touch sense here as in the cord. These two senses are involved when the hand, unaided, recognizes an object held in it (stereognosis); they are especially combined for this purpose in the superior parietal lobule, and loss of this perceptive power—*astereognosis*—is most commonly due to lesion in that area.

Muscular sense guides the cerebellum in its chief function, the maintenance of equilibrium. Fibres delegated to this function from the root-zone enter the base of the dorsal gray horn and connect with the cell-bodies of Clarke and Stilling which are found in that situation throughout the cord (Gordinier). These cell-bodies are the beginning of superior muscular-sense neurons; their axons sweep outward to the margin of the cord and turn upward in the direct cerebellar tract, the terminus of which is the middle lobe—vermis—of the cerebellum, which it reaches by way of the inferior cerebellar peduncle—restiform body. Lesion of this neuron-system, in the cord or in the cerebellum (Barker), causes the defect of equilibration called cerebellar ataxia.

Assistance in coördination is derived from all the senses, consciously, as when the tabetic watches the ground in walking, and unconsciously, through impulses collected in the cerebellum from the eye, cutaneous sensations, the joint and muscle surfaces and the internal ear. Disturbance in one of these sensory organs may cause vertigo.

The internal ear is virtually two organs, having distinct functions, and the eighth nerve is double accordingly. The semicircular canals of the vestibule are water-levels telling the position of the head, as muscular sense does that of the limbs, and the part of the eighth nerve arising thence called the vestibular nerve is concerned not with hearing but with equilibration. It connects with its superior neurons in the dorsomesal nucleus to pass to the cerebellum.

Lesion of any part of the vestibular tract from the internal ear to the cerebellum may cause vertigo, as in Ménière's disease.

The eighth nerve's division into two is clear as it enters the pons, the two parts being separated by the inferior cerebellar peduncle. The outer or cochlear division is the true nerve of hearing. It enters the ventrolateral nucleus to be continued by fibres that cross the middle line of the pons, forming the trapezoid body,—acoustic decussation (M. Allen Starr),—then pass upward in the lateral fillet, and by way of the postgeminum and postgeniculum reach the auditory centre in the first temporal convolution.

The Stigmata of Degeneration.

Degeneration, degeneracy, deviation are terms used to denote in individuals a decline from the average normal condition in physical or moral qualities. This decline varies in degree from deviations from the normal scarcely to be recognized upon the most careful study, to the possession of physical and moral defects which render the subject unfit for the ordinary duties and responsibilities of life, and are obvious to the casual observer. It is accompanied by physical, physiological and neuropsychic anomalies known as the "stigmata of degeneration." An undue importance has doubtless been ascribed to these anomalies and their combinations, especially to those of minor degree, by Lombroso and his followers; nevertheless their consideration is of practical value in the study of diseases of the nervous system and has an important bearing upon the diagnosis and prognosis of individual cases of this group of affections. Every sign of deviation from the average normal is not necessarily a stigma of degeneration, and Walton has suggested that it is desirable "to name the phenomena signs of deviation, and call their possessors deviates or a deviate as the case may be, limiting the term degeneration only to such deviations as obviously imply deterioration."

Etiological Classification.—Walton has grouped the causes of the so-called stigmata of degeneration, including the signs of deviation only, as follows:

I. The potential variations from the average normal contained in the parent germ, including the results (a) of atavism, (b) of parental similarity, and (c) of selective tendency on the part of the ancestry.

II. Intra-uterine infection.

III. Mechanical injury during intra-uterine life.

IV. The absence or peculiarity in the germ of certain elements, or their disappearance or anomalous development, without traceable inherited tendency or other known explanation.

V. Mechanical influence exerted during infancy.

VI. Deleterious influences and habits in the ancestry, productive of enfeeblement, undersize, and lessened resistance in the progeny but not altering the essential potential characteristics transmitted by the parent germ.

VII. Absence or hypertrophy of certain glands, pituitary, thyroid, which have a nutritional influence.

VIII. Arrest of development, such as is seen in harelip and similar defects.

List of Stigmata.—The following list, compiled from Dana, Church and Peterson, Walton, and others, includes the more important deviations and stigmata. Those which are of minor significance, either alone or in association with others, as indicating actual degeneracy, are placed in middle single columns; those generally recognized as stigmata of degeneration, in double columns at the sides.

Anatomical Stigmata.

ANOMALIES OF THE CRANIUM.

Cranial asymmetry.		Macrocephalus.
Microcephalus.		Platycephalus.
Leptocephalus.		Oxycephalus.
Plagiocephalus.		Scaphocephalus.
Trigonocephalus.		Short parietal arc.
Short frontal arc.		High prominent forehead.

ANOMALIES OF THE FACE.

Heavy jaws.		Prognathism.
Lemurian hypophysis.		Opisthognathism or retrognathism.
Orthognathism.		Crania progenæa (lower teeth projecting beyond upper, and inferior maxillary angle obtuse).
Large frontal sinuses, small orbit.		
Great or unequal prominence of malar bones.		

ANOMALIES OF THE EYE.

Narrow palpebral fissure.		Microphthalmus.
	Flecks on the iris.	
Albinism.	Chromatic asymmetry of the iris.	Congenital cataracts.
Pigmentary retinitis.		Muscular insufficiency, strabismus.
	Hypermetropia.	
Myopia.		Astigmatism.

DEFORMITIES OF THE PALATE.

High and narrow.		Horseshoe.
Torus palatinus.		Gothic arch.
[illegible]		Flat-roofed.
Hypsiloid.		Asymmetrical.

DENTAL ANOMALIES.

Badly set and badly [illegible].		Small or **peg-shaped lateral incisors.**
	Double rows.	
	Adventitious teeth.	
	Double crown.	
	Macrodontism.	
Microdontism.		Projecting teeth.
Badly placed or misplaced teeth.		Striated **transversely.**
Hutchinson's teeth.		

ANOMALIES OF THE NOSE.

	Deviation of nose.	
Defective development of cartilage and tissue of alæ.		Absent cartilages.
		Atresia of **nasal fossa.**
		Defective **osseous** development.

ANOMALIES OF THE TONGUE AND LIPS.

	Macroglossus.	
	Microglossus.	
	Bifidity of point.	
	Harelip.	
	Cleft palate.	

ANOMALIES OF THE EAR.

	Excessively long.	
	Excessively prominent.	
	Set too close to the head.	
Set too far back.		Set too low.
Absence of helix, antihelix, or lobule.		Obliteration of **markings.**
Absence of fissura intertragica.	Excessively large (absolutely or relatively).	Too conchoidal **(antihelix, crura, etc., too little marked and helix like rim of funnel).**
Too small.		Lack of **uniformity in width.**
Asymmetry of the two ears, general anomaly of left (Blainville ear).		
	Prominence of antihelix.	
	Adherent lobules.	

ANOMALIES OF THE LIMBS.

	Left arm and leg longer than right.	
	Excessive length of arms.	
	Long fingers.	
	Polydactyly.	
Symphysodactyly or schistodactylus (joining of fingers).		Syndactyly **(web fingers).**
Ectrodactyly (fingers wanting).		Amelus or **ectromelus (limb** wanting).

Anomalies of the Limbs.

Phocomelus (segment of limb wanting).		
	Megalomelus. Megalodactyly.	
Oligomelus (excessive gracility).		Oligodactyly.

Anomalies of the Trunk and General Structure.

Hernias, when congenital.		Malformation of breasts and thorax.
Dwarfism.		Gigantism.
Infantilism.		
Masculinism in women.		Femininism in men.
	Spina bifida.	
Feebleness of construction.		Lordosis.
Scoliosis.		Kyphosis (Fere).
Malformed coccyx.		Thoracic asymmetry.
Mammary development in males.		Mammary absence or reduplication in females (polymastia).

Anomalies of the Genital Organs.

Small or deformed genitalia.		Hermaphrodism.
Cryptorchismus.		Hypospadias.
Epispadias.		Defect, or great volume of prepuce.
Torsion of prepuce. .		Imperforate meatus.
Labia too large or too small.		Clitoris large.
Labia minora hypertrophied.		
	Folds between labia majora and minora.	
	Labia minora pigmented, particularly in brunettes.	
Imperforate vulva.		Atresia of vagina.
Double vagina.		Uterus bicornis.
Atrophic uterus.		

Muscular Anomalies.

Dystrophies.		Unequal innervation of facial muscles on the two sides.
Depression above glabella due to overaction of corrugators.		

Anomalies of the Skin.

	Polysarcia.	
	Hypertrichosis (superfluous hair).	
	Premature grayness.	
	Precocious and abnormal hairy development.	
	Rudimentary tail.	
Glabrous chin (no beard).		Vitiligo.
Absence of nails or fetal state of nails.		Melanism of skin.
	Pigmented or vascular nævi.	
Molluscum.		Ichthyosis.
	Pigmented spots.	

Physiological Stigmata.

Anomalies of Motor Function.

Lefthandedness. **Retardation of learning to walk and talk.** Nystagmus (congenital).		Tremors. Epilepsy. Tics.—Facial spasm, habit chorea, tic convulsif.

Anomalies of Sensory Function.

Deaf-mutism.		
	Neuralgia. Migraine. Constitutional headaches.	
Hyperæsthesia. Blindness.		Anæsthesia.
	Daltonism (color-blindness, achromatopsia). Hemeralopia (night-blindness).	
Nyctalopia (day-blindness).		Concentric limitation of visual field.

Anomalies of Speech.

Mutism.	Stammering. Stuttering.	Defective speech.

Anomalies of Genito-urinary Function.

Sexual irritability. Sterility. Amenorrhœa.		Impotence. Urinary incontinence.

Anomalies of Instinct or Appetite.

Gluttony. Rumination.		Merycism. Uncontrollable appetites (narcotics).

Retardation of Puberty.

Deficient Vital Activity of Organic Functions.

	Weak heart. Low arterial tension. Coldness of extremities. Flushing of extremities. General chills and flushes. Weak digestion. Constipation.	

Psychic and Psychoneurotic Stigmata.

Dementia præcox. Mania depressive. Hysteria. Phobia. Invalid habit. Feeble-mindedness.		Compulsive insanity. Melancholia. Hypochondria. Psychopathic endowment. Idiocy.
	Eccentricity.	
Moral delinquency. Precocity.		Sexual perversion. Over-development of certain aptitudes.
Paranoia.		Ideo-obsessive constitution.

X.

THE EXAMINATION OF THE EYE.

General Considerations.

The close relationship existing between the eye and the nervous system, the opportunities furnished by the fundus of the eye to study changes in the *general* circulatory system, and the knowledge that disturbances of ocular function are not infrequently the underlying cause of systemic affections, render a study of the ocular apparatus of extreme importance in the diagnosis of general disease.

It must be remembered that, while the eye is the organ of sight, with its own special function, it is also a part of the general organism, is influenced by the status of the whole body, is disturbed with the disturbance of other structures, and exhibits in a marked degree affections of other organs by which its function is interfered with.

The importance of the thorough analysis of the ocular complications in all diseases, particularly in affections of the brain and spinal cord, is well recognized. An examination of this character, to be effective, must be systematized in order to determine the actual conditions underlying an affection of which the eyes furnish the chief manifestation; in what respect, if any, the ocular functions are abnormal; and finally, the true inference to be drawn from these disturbed functions in the diagnosis of systemic affections.

1. INSPECTION: The position of the eyeballs in relation to the orbital bones is observed to determine any undue prominence or recession of one or of both eyes; any abnormality of the eyelids as evidenced by tumors, general swelling, drooping, inability to close the lids, inverted margins, size of the commissure, and the presence of crusts or secretions on the margins; congestion of the blood-vessels, or granulations or new growths on the conjunctiva; the size, response to light stimuli, and equality or inequality of the pupils, and variations in the color of the irides; deviation of the visual axes, or involuntary movement of the eyeballs; the sensibility of the cornea or its loss of transparency; and the depth of the anterior chamber and any turbidity of its fluid contents.

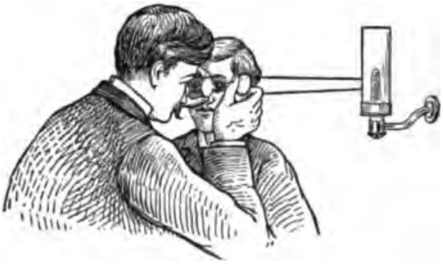


FIG. 147.—Oblique or focal illumination.—From Hansell and Sweet.

The anterior segment of the eyeball is most satisfactorily studied by *oblique illumination.* The patient is placed about two feet from the source of illumination. The examiner focusses the light upon the cornea with a convex lens of 2-inch or 3-inch focus held between the thumb and forefinger of the right hand, and studies the illuminated area through another lens of similar strength held between the thumb and forefinger of the left hand, the second finger raises the upper lid, and the little finger resting upon the forehead steadies the hand. The distance of the second lens from the eye is varied slightly to bring into focus the cornea, iris, and crystalline lens. Opacities of the cornea or lens, as seen by oblique illumination, appear as gray or white spots upon the black background of the pupil.

2. VISION: Decrease in the normal acuteness of vision of each eye as measured by test letters for near and far is to be noted; the history of the decline, and its association with pain or inflammation of the external structures; any departure from the normal field of vision must be recorded; contraction of the peripheral limits for form and color, areas of deficient or lost perception, and reversal in the order of the color fields.

3. OPHTHALMOSCOPIC EXAMINATION: Two methods are employed in the examination of the deeper structures of the eye by the ophthalmoscope—the Direct Method, which gives an upright image of the eyeground, and the Indirect, in which the image is inverted. In both the patient is seated in a darkened room with his back to the source of illumination, and the observer is to the side to be examined. By the direct method the examiner approaches close to the side of the patient's head, using his eye corresponding to the eye under examination, and reflects the light by means of the ophthalmoscopic mirror into the eye. The rays from the fundus are reflected back, and, passing through the opening in the mirror enter the observer's eye, giving an upright image of the eyeground. The optic nerve is best seen when the patient looks at a distant object to the side and beyond the observer's head. The foveal region is brought into view when the patient's gaze is directed into the aperture of the mirror. By the indirect method the observer, about 15 to 20 inches in front and to the side of the patient, reflects the light through a convex lens of about 2-inch focus held at its focal length from the eye, and secures an aërial image focussed by the strong glass. In case the details of the fundus are not at first plainly seen the object lens is slightly advanced or withdrawn from the eye. Strain on the examiner's accommodation is relieved by a +4 D. lens rotated before the sight-hole of the ophthalmoscope.

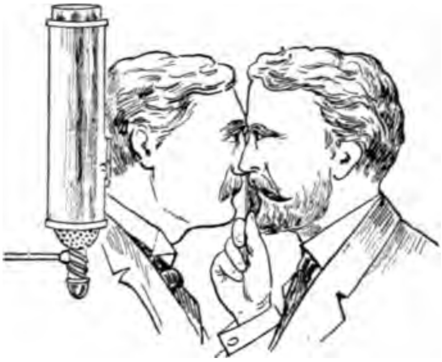
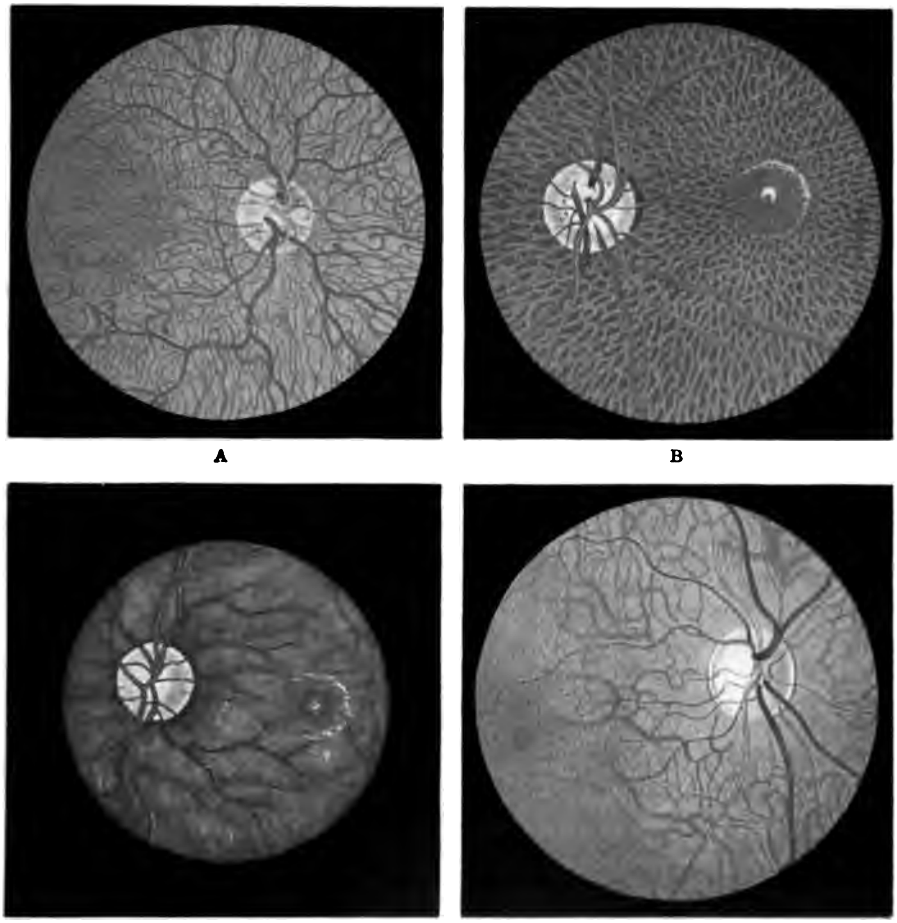


FIG. 148.—Ophthalmoscopic examination by direct method.—From Hansell and Sweet.

The normal eye presents many variations from the typically pictured fundus, and extended experience is necessary to distinguish the variations in health from the changes wrought by disease. The color of the fundus reflex is a bright pink or red, due to the reflected light from the choroidal vessels and the pigment of the retina and choroid. In the negro the reflex is grayish, because of the absorption of the light rays by the abundant pigment. The optic disk, or nerve head, lies to the nasal side of the posterior pole, and is round or oval, with clear cut edges, often fringed with choroidal pigment. The nerve is often cupped in the centre, at which point the central artery and vein

PLATE VIII.



A

B

C

D

[Varieties of the Normal Fundus.—After Würdemann in Posey and Spiller.

A, albinotic fundus, albino and light blonde (after Greef, modified by Würdemann). B, the tessellated fundus; brunette (after Greef, modified by Würdemann). C, the negroid fundus; negro (Würdemann). D, the yellow fundus; Chinese (after Oeller, modified by Würdemann).—D.]

pass. The artery and vein divide into two main branches, and these subdivide into the numerous smaller vessels. The fovea, with its central yellow spot, is the most sensitive part of the retina. It is about 3 mm. to the temporal side of the nerve, and is darker than the rest of the retina. In this region no blood-vessels are to be seen by the ophthalmoscope.

4. PAIN: The character of the pain should be known, its situation, its dependence on the use of the eyes, and its association with tenderness in the region of the orbit, particularly at the points of exit of the supra-orbital or infra-orbital nerves.

5. HEADACHE is one of the most prominent symptoms of eyestrain. It is dull and heavy, usually bilateral, increased by application to close work, riding in cars and shopping, and sometimes accompanied by pain in the eyeballs. It is to be distinguished from the sharp periodic attacks of pain characteristic of neuralgia of the first and second divisions of the 5th nerve.

Affections of the nasal tissues, as deflections of the septum and purulent collections in the frontal sinus, cause headache which resembles that of eyestrain. The diagnosis of nasal and sinus headache is made by its longer duration, its association with manifest symptoms of nasal trouble, and its independence of use of the eyes.

Asthenopia from general muscle weakness is present during convalescence from acute fevers or prolonged illnesses, and attempts at reading are often followed by headache, blurring of sight, and pain in the eyes and head. DeSchweinitz refers to a peculiar form of asthenopia seen after the presbyopic age, most frequently in women, which is not relieved by glasses or treatment of muscular anomalies. These patients present the ordinary symptoms of neurasthenia, doubtless the outcome of beginning arteriosclerosis, and proper tests usually show high arterial tension, which, if reduced by appropriate dietetic and medicinal measures, will cause a disappearance of the asthenopia.

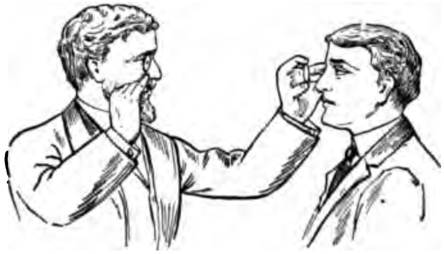


FIG. 149.—Ophthalmoscopic examination by indirect method.—From Hansell and Sweet.

6. PHOTOPHOBIA is a symptom of affections of the cornea and iris, of a few diseases of the retina, and in many cases of uncorrected refractive errors and muscular anomalies. As an isolated symptom it possesses little importance in arriving at a differential diagnosis of ocular affections.

7. EPIPHORA: Increase in the flow of tears is seen in exophthalmic goitre, in certain affections of the central nervous system (locomotor ataxia), and in obstruction of the lachrymal duct.

The Eyeball and Orbit.

Protrusion of the eyeball—exophthalmos, proptosis—may be caused by tumors, aneurisms, hemorrhage, exostoses, and growths or inflammations originating in or extending to the orbit from the adjacent sinuses; by orbital cellulitis and paralysis of the ocular muscles.

Bilateral exophthalmos, varying from a slight prominence of the eyeballs to a protrusion that prevents the closure of the eyelids, is found in exophthalmic goitre. Widening of the palpebral fissure from nervous affections, with undue exposure of the sclera, will give the impression of exophthalmos.

Proptosis associated with deep-seated pain upon attempts to move the eyeball, limited or complete immobility of the globe, and swelling and œdema of the eyelids, which may be so great as to prevent opening of the lids, is found in orbital cellulitis, facial erysipelas, meningitis, in general septicæmia, or, in less degree, after scarlatina, typhoid fever, and influenza. Purulent inflammation of the adjacent sinuses may lead to the same symptoms.

Sudden exophthalmos in infants, with the eyeballs turned down, points to the possibility of the existence of scurvy. The protrusion may be moderate at first and increase during 24 hours and be associated at its height with thickening and ecchymosis of the upper lid. The subperiosteal hemorrhage to which the affection is due may affect both orbits, but unequally. The eyeball is freely movable.

Pulsating exophthalmos, usually unilateral, most frequently follows traumatism, and is found in arteriovenous aneurism of the internal carotid and cavernous sinus or aneurism of the ophthalmic artery.

Abscess of the frontal sinus may cause displacement of the eyeball downwards and outwards, with diplopia. In purulent disease of the frontal and ethmoidal sinuses a small fluctuating swelling may appear at the upper and inner angle of the orbit, which breaks and discharges pus. Gradual displacement of the eyeball forward may be the result of an orbital tumor within the cone of muscles.

Exophthalmic Goitre.—One of the earlier signs is lagging of the upper lid when the eyes are slowly rotated downwards (Graefe's sign). There is also imperfect power of winking (Stellwag's sign); retraction of the upper lid and widening of the palpebral fissure (Dalrymple's sign), and imperfect power of convergence of the eyes.

Involuntary resistance to eversion of the upper eyelids is believed by Gifford to be one of the earliest symptoms of Graves's disease. It tends to disappear with the development of the disease, and is explained by hyperexcitability of Müller's muscle through the sympathetic. Gifford also attaches importance to swelling of the tissues between the eyebrow and eyelid as an early diagnostic sign of the disease.

Retraction of Eyeball.—Enophthalmus, or sinking of the eye into the orbit, occurs in some instances in extreme emaciation from absorption of orbital fat, in paralysis of the sympathetic, in facial hemiatrophy, and from traumatism. Since the amount of exposed sclera determines the apparent recession of the globe, the examiner may be misled into mistaking the widened commissure of tabes or other nerve disease for enophthalmus. Traumatism in the vicinity of the orbit is sometimes followed by an actual enophthalmus, which may be immediate, or be delayed for several weeks or months.

Nystagmus is a series of involuntary, regular, and rapid oscillations of the eyes. These movements may be horizontal, vertical, or rotary, or a combination of all three. Unilateral nystagmus is rare. The lateral oscillation is the most common. Congenital nystagmus is found in children with congenital cataract, dense central corneal opacity, or imperfectly developed eyeballs, and in albinism. Miner's nystagmus is an acquired form, which is probably due to the prolonged upward inclination of the eyes in semi-darkness. Nystagmus may be a symptom of irritation or diseases of the inner ear, of tumors of the cerebellum, multiple sclerosis, hereditary ataxia, and of syringomyelia.

In multiple sclerosis and in hereditary ataxia the nystagmus occurs only when the eyes are turned in the direction of a moving object, and particularly as the eyes reach the limit of their rotation in the lateral plane. A slight nystagmus is occasionally found in hysteria.

Tension.—In the normal eyeball the tension of the globe, as measured by the pressure of the two index fingers upon the sclera through the closed lid, presents a uniform resistance. Increase in the intra-ocular tension occurs in acute glaucoma, in some forms of iridocyclitis, and occasionally after traumatism. Lowered tension may result from degeneration of the ciliary body and choroid, in rupture of the globe, detachment of the retina and after operations.

The Eyelids.

Marginal Inflammation.—Red, moderately swollen lid margins, associated with heat, burning and photophobia, are seen in persons exposed to cold winds and dust, in children affected with nasopharyngeal inflammation following measles, and as a result of the strain of uncorrected refractive errors. In severe types of the disease the lid margins are covered with hardened, yellowish crusts which, upon removal, expose ulcers extending deep into the lid border.

Redness and itching of the lid margins in children may be due to the presence of the pediculus pubis in the eyelashes. Close examination will show the eggs upon the cilia, and the parasite partly buried in the hair follicle.

Inversion of the lashes or of the lid border is most commonly caused by chronic inflammation of the bulbar conjunctiva. The irritation of the misplaced cilia ultimately leads to inflammation and haziness of the cornea. Eversion of the lid may follow burns or wounds, with subsequent cicatricial contraction of the skin; appears as a senile condition, from loss of muscle power; or accompanies facial palsy. The lower lid is most frequently affected.

Œdema of the lids accompanies severe inflammation of the conjunctiva, purulent disease of the eyeball, orbit or frontal or ethmoidal sinuses, and general affections like nephritis and gout.

Localized swelling of the eyelids and conjunctiva, with or without vascular changes of the eyeball, is seen in *neurotic œdema,* urticaria, and disease of the antrum, or may be due to errors of diet. The swelling may be sufficient to completely close the eye, and is accompanied by itching and burning. In a few days the parts return to the normal.

A localized painful swelling of the lid and discoloration of the skin may be either a stye (*hordeolum*) or an abscess of the lid (*phlegmon*). The pain is severe and is frequently accompanied by swelling of the entire lid and œdema of the conjunctiva. Styes are situated in the connective tissue near the lid margin and are apt to recur in persons with deranged bodily functions, and in those who suffer from uncorrected refractive errors.

Swelling of the lid, appearing suddenly after injury, and increasing upon blowing the nose, the soft mass crackling on pressure, is caused by the presence of air which has escaped into the cellular tissue through a fracture of the orbital wall (emphysema).

Inflammation of the tarsus (tarsitis), usually monocular, may be syphilitic, gouty or tubercular. The lid is swollen and drooping and cannot be raised by the levator palpebrarum muscle.

Sebaceous cysts occur both on the eyelids and in the eyebrow. An accumulation of secretion in the sebaceous glands of the lids appears as small yellowish elevations which develop about the age of puberty, and are due to improper care of the skin or to gastro-intestinal disorders. Molluscum contagiosum is a disease of the sebaceous glands which occurs among ill-nourished children. It is characterized by waxy-colored, rounded papules, the size of a pea.

Erysipelas attacks the lids by extension from the adjoining skin of the face. The typical shiny, dusky swelling, with the subsequent development of small vesicles, serves to distinguish it from other affections. Severe attacks may affect the orbital tissues and cause optic nerve atrophy and fatal meningitis.

Syphilis may appear either as a primary sore, or as a secondary disease, and is usually located at the lid border. The ulceration and induration present the typical features of a chancre. It may be mistaken for an epithelioma, but the improvement under antisyphilitic treatment clears up the diagnosis.

Herpes Zoster.—Violent paroxysmal pain, associated with a vesicular eruption on the skin of the lid, forehead and occasionally the side of the nose, is indicative of *herpes zoster ophthalmicus.* The vesicles vary in size, have an inflamed base, and are situated over the region supplied by the first and second division of the fifth nerve. Corneal ulcers and iritis show ocular involvement. Depressed nutrition is a common cause.

Xanthelasma.—Yellowish irregular shaped patches of connective tissue, located in the upper eyelids near the internal canthus, are termed *xanthelasma.* Rarely they form on the lower lids. The growth causes no trouble, and is probably due to local ill nutrition.

Chalazion.—A small round elevation of either the upper or lower lid, of slow growth, with the skin freely movable over the hardened tumor, and a purplish discoloration of the surface of the conjunctiva immediately beneath, is termed a *chalazion.* It is due to inflammation of a Meibomian gland, with retention of the secretion. Inflammation of the lid margins and the strain of ametropia may be causative factors.

Inflammation of the Lachrymal Sac.—A small tumor near the inner surface of the lower lid over the lachrymal sac, which disappears upon pressure, with the escape of a viscid mucus through the puncta, is due to catarrhal inflammation of the sac. The secretion may become purulent, with severe pain, and intense swelling and redness of the skin somewhat resembling erysipelas. In both the chronic and acute forms the tears flow over the cheek. A swelling at the upper and inner angle of orbit, due to sinus disease, has been mistaken for lachrymal abscess.

Ptosis.—Drooping of the eyelid, partially or completely covering the cornea, may be either congenital, or due to injury to the levator muscle, to thickening of the tissues of the lid, or to paralysis of the third nerve. A form of hysterical ptosis due to spasm of the orbicularis muscle has been described.

Lagophthalmos, or inability to close the eyelid, may be congenital, but is usually an accompaniment of paralysis of the facial nerve. Marked protrusion of the eyeball, mechanically preventing closure of the lid, is seen in orbital tumors, exophthalmic goitre, and in corneal staphyloma.

Blepharospasm, or an involuntary contraction of the eyelids, may vary in degree from a slight twitching of a few of the fibres of the orbicularis to a tonic spasm. In its simplest form it is due to uncorrected refractive errors, inflammation of the lid borders, and corneal and conjunctival irritation. Obstinate cases of cramp of the orbicularis arise from reflex irritation of the fifth nerve, through some remote cause that it is often difficult to determine. It is occasionally a hysterical manifestation.

Conjunctiva and Sclera.

The white of the conjunctiva changes to a dull yellow in so-called biliousness, and to a pronounced brownish-yellow color in icterus. In anæmia, tuberculosis, and nephritis the conjunctiva may become pearly white.

Inflammation.—The normal conjunctiva is coursed by a few small blood-vessels which arise from the deep furrow where the membrane is reflected to the under surface of the lids. Redness is the result of a marked increase in the number of blood-vessels from inflammation of the conjunctiva, or, as this membrane covering the eyeball is transparent, to congestion of the deep sclera beneath.

Hyperæmia of the conjunctiva is seen in measles, scarlet fever, hay fever, influenza, nasal catarrh, the strain of uncorrected refractive errors and from exposure to wind, dust, and bright light and heat. The conjunctiva is often inflamed in facial paralysis, owing to the inability of the lids to protect the globe from external irritants. The presence of a foreign body on the conjunctiva or cornea causes many of the characteristic symptoms of an acute catarrhal conjunctivitis.

The ordinary conjunctival inflammations are unattended with severe pain, but are accompanied with a mucous or mucopurulent discharge. In inflammation of the sclera, the affected area is seen to be beneath the loose conjunctiva, while in disease of the iris, ciliary body or cornea, a ring of fine straight vessels surrounds the corneal border. In these latter affections, pain is often quite severe. Since the conjunctiva may be also inflamed in disease of these deeper structures, a diagnosis cannot be made by the appearance of congestion only.

A type of contagious conjunctivitis (acute contagious conjunctivitis) is due to the Koch-Weeks bacillus or to the pneumococcus. A subacute form of conjunctival inflammation, which may occur in epidemic form, is caused by the diplobacillus of Morax and Axenfeld. Severe inflammation, with swelling of lids, infiltration of the conjunctiva, and a purulent discharge, occurs from the entrance of infection, usually the gonococci, into the eyes of the child from the birth canal (ophthalmia neonatorum). A similar form of inflammation follows the entrance of gonorrhœal pus into the conjunctival sac of the adult (gonorrhœal conjunctivitis).

Diphtheria of the conjunctiva is rare. A membrane forms on the surface of the conjunctiva, presenting the same characteristics as that found in the throat. A pseudomembranous conjunctivitis may be due to the pneumococcus or to streptococcus infection, and to some of the other micro-organisms found in the ordinary types of inflammation.

Roughness or elevation of the conjunctiva of the lids may indicate trachoma or vernal catarrh. Distended and tortuous vessels in the conjunctiva may be due to constipation, auto-intoxication, chronic alcoholism or lithæmia.

Single or multiple blebs appear on the conjunctiva in badly nourished children, often after measles. Eczema of the nares and disease of the nasopharynx are usually coexistent.

Hemorrhage beneath the conjunctiva appears in injuries of the head, and also in severe compression of the abdomen. It is not uncommon in whooping-cough, after severe vomiting, and in obstinate constipation, the straining causing a rupture of one of the conjunctival vessels. Spontaneous hemorrhage in the aged, especially if recurrent, should direct attention to the possibility of disease of the blood-vessels and to nephritis.

Uric acid deposits are frequently found in the conjunctiva of the lids of gouty individuals.

Tumors and cysts of various kinds may appear in the conjunctiva of the eyeball. Small, yellowish elevations are found near the cornea, usually at the inner portion, but are of little significance. A fleshy fan-shaped growth is often seen in persons past 40 years whose eyes have been subjected to long exposure to wind, dust or sand. The usual situation is over the internal rectus muscle, the apex often extending upon the cornea. Most of the malignant growths appear at the junction of the sclera and cornea.

Inflammation of the sclera is found in association with the rheumatic and gouty diathesis, in scrofula, intestinal disorders and in syphilis. In the superficial form of inflammation (episcleritis) there is usually a circumscribed area of purplish discoloration beneath the conjunctiva, most frequently in the region of the external rectus muscle, and slightly raised above the healthy sclera. In disease of the true sclera (scleritis) the inflammation may affect the entire anterior portion, and extend to the cornea, iris, and ciliary body. Affections of sclera are distinguished from conjunctivitis by the engorgement of the deeper vessels, the purplish color, the severe pain, the absence of discharge, and the frequent relapses.

Cornea.

Keratitis.—The cornea is subject to both ulcerative and non-ulcerative affections.

Ulcerative Keratitis.—Loss of sensibility of the cornea, with subsequent ulceration and destruction, is found in affections of the trunk of the fifth nerve or of its ganglion, or after removal of the latter for trifacial neuralgia. The corneal affection is due to a trophic change in the membrane and to the irritation of foreign substances, which are not recognized by the insensitive cornea.

A severe type of corneal ulceration, which may progress to perforation, is found in association with herpes of the region about the eyes, particularly of the lachrymal branch of the trifacial. The disease is preceded by severe burning and neuralgic pain, in isolated spots, upon which are developed the characteristic vesicles.

Chronic malarial keratitis attacks the superficial layers of the cornea, is characterized by anæsthesia of the cornea and tenderness over the supracorneal notch. The ulcer is narrow, with offshoots resembling the veins of a leaf (dendritic keratitis). The disease is rare, however, even in malarious districts.

Small blebs, which later break down into ulcers, are located either on the cornea or at the junction of the cornea and the sclera in strumous children, and are associated with inflammatory diseases of the nasal passages, often following the exanthematous fevers. Eczema about the nares is usually coexistent. Abscess and ulceration may occur during convalescence from measles, smallpox, scarlet fever and other toxic diseases. Exophthalmic goitre may give rise to extensive ulceration owing to constant exposure of the cornea through inability to close the lids over the globe. An extensive ulceration of the centre of the cornea may follow the exhaustion of a prolonged diarrhœa or dysentery or similar debilitating illness in the aged.

INTERSTITIAL KERATITIS.—Inflammation of the deeper layers of the cornea, without ulceration, is frequently seen in children, between 5 and 15 years of age, who have inherited syphilis, and also in tubercular, scrofulous, and other poorly nourished individuals. In its earliest stage the congestion surrounding the cornea is of the deep vessels, there is dread of light, and close examination shows a fine dot-like infiltration of the interstitial layers of the cornea, which later coalesce into the typical bluish white haziness. The affection is bilateral, although months may elapse before the second eye is affected.

Arcus Senilis.—A circle of fatty degeneration close to the cornea, but with a clear ring separating it from the junction of the cornea and sclera is present in the eyes of persons of advanced years. It possesses no significance. A senile atrophy of the margin of the cornea has been described in association with arcus senilis.

Partial insensibility of the cornea is seen in exophthalmic goitre, and its presence probably explains a number of the other eye symptoms.

The Iris and Pupil.

Pigmentation.—Slight variation in the pigmentation of the irides is not uncommon in health, but difference in color is rare, except in disease. A yellow green color of one iris, while the other is blue or brown, is an early evidence of inflammation of the iris and ciliary body. Retained metallic foreign bodies often cause the iris to assume the brownish hue to which the term siderosis is given. Inflammation of the iris occurs in syphilis, rheumatism, gout, tuberculosis, diabetes, and from injuries, primarily in one eye, or in the fellow eye from sympathy. Inflammation is accompanied by irregularity and contraction of the pupil, injection of the pericorneal vessels, and frontal pains, usually worse at night. Swelling in the stroma of the iris is a sign of tertiary syphilis. Sarcoma, as a primary disease, is rare.

The Pupil.—Variations in the size of the pupil occur under the influence of light, and in convergence and accommodation. The average size of the pupils, in diffuse daylight, with the eyes fixed on a distant point, is 4 mm. Careful tests of changes in the pupils are of importance in the diagnosis of general affections, particularly of the nervous system.

The normal reactions are as follows:

1. Direct Reaction.—If one eye is excluded, and the patient directed to fix a distant object, the pupil of the exposed eye, when covered by the hand or card, will dilate. Upon removal of the cover it will contract to its previous size.

2. Indirect Reaction (Consensual Reflex).—If one eye is shaded, the other pupil will dilate equally with the shaded pupil, to again contract when the shade is removed. Normally the two pupils should be of equal size, whether one or both is covered or uncovered.

3. Associated Reaction (Reflex to Accommodation and Convergence).—The patient is directed to look into the distance and then converge the eyes on a point, such as a pencil, held about 5 inches from the eye. The pupils contract under the influence of the convergence and accommodation.

4. Sensory Reaction (Skin Reflex).—Stimulation of the sensory nerves of the skin, by pinching the skin of the neck, or by the passage of a faradic brush along the spine, causes slight dilatation of the pupils.

5. Orbicularis Pupillary Reaction (Lid-closure Reflex).—Contraction of the pupils occurs upon forcible efforts to close the lids.

6. Drug Reaction.—Dilatation of the pupil (mydriasis) follows the instillation of mydriatic drugs, and contraction of the pupil (myosis) the instillation of myotics.

7. Cerebral Cortex Pupillary Reflex.—Haab describes a reflex to which this term has been given. He found that if a patient seated in a dark room, with the eyes fixed at the black wall, and a light placed to shine laterally into the eyes, is requested to direct his attention to the light, without changing the position of the eyes, the pupils will contract. Since the accommodation remains suspended, and the light entering the eye is unchanged, the contraction of the pupil is in some manner connected with the power of attention, and Haab, therefore, believes the test should be made in every case of nervous disorder.

Myotic Pupillary Tract.—Stimulation of the centre for the third nerve, by the action of light passing along the optic nerve and optic tracts, causes an impulse to pass to the lenticular ganglion, and thence by the short ciliary nerves to the sphincter of the pupil, which contracts, lessening the size of the pupil.

Mydriatic Pupillary Tract.—The dilator muscle of the iris is innervated by the sympathetic. The impulse passes from the medulla into the cord, thence through the first three dorsal nerves to the superior cervical ganglion, to the plexus around the internal carotid, and through the long ciliary nerves to the ciliary muscle and iris. Stimulation of the centres of this tract causes dilatation of the pupil.

Abnormal Pupillary Reactions.—Failure of the pupil to react, either wholly or in part, is due to a lesion in the iris, in some part of the third nerve, in the centres of the brain, or in the light-conducting paths. Lesions in the iris may be swelling or atrophy, or old or recent attachments from inflammation. Immobility to light stimulus, with preservation of the reflex to accommodation, is one of the important abnormal pupillary changes.

REFLEX IMMOBILE PUPIL (ARGYLL-ROBERTSON PUPIL). — Loss of reaction of the pupil to direct light, with preservation of the contraction of the iris in accommodation and convergence, comprises the well-known Argyll-Robertson pupil, and is an early symptom of tabes. Although of great diagnostic value when present,—and in the majority of cases it exists in the incipient stages of the disease,—there are rare instances in which it has not been found, even when all other symptoms of the disease have existed for years. Associated with lost light reflex is frequently noticed alteration in the shape of the pupil. The pupil may be of normal size, but more often myosis is found, from implication of the cervical portions of the cord controlling the dilating centres. The Argyll-Robertson pupillary phenomenon is also seen in paretic dementia. The loss of the light reflex in aortic disease is due to the general syphilitic infection.

DILATATION OF THE PUPIL.—The pupil is dilated in glaucoma, in optic atrophy, in diseases of the orbit, in irritation of the cervical sympathetic, in acute mania, in cerebral softening, in extensive disease or injury of the cerebral centres, in complete paralysis of the third nerve, in paralysis of the sphincter of the iris by a blow upon the eyeball, in strong emotion, and when mydriatics have been used. In neurasthenia and hysteria, mydriasis is often present.

Dilatation of the pupil may be caused by an irritation of the dilator pupillary centre or tract (irritative mydriasis), or by a paralysis of the pupil-contracting centre or fibres (paralytic mydriasis).

Unilateral mydriasis, in which the pupil fails to react to direct light but contracts consensually with its fellow, is seen in complete optic atrophy, in which the conductivity of the one optic nerve is lost. The failure of one pupil to react to separate stimulation of either eye, but contracting upon convergence, while the other pupil reacts to light stimulus of either eye, is seen in tabes and in syphilis. Sudden unilateral mydriasis in which the instillation of a drug can be excluded is worthy of a careful study as a possible early symptom of latent sclerosis of the cord.

Corte claims that in any serious diphtheritic attack failure of the pupils to react to light indicates a fatal termination.

Complete blindness will cause bilateral mydriasis with failure of the pupils to react to light stimulus. A slight contraction of the pupils has been observed in the blind, who are entirely devoid of light perception, after the eyes have been exposed to bright daylight for several minutes.

In mydriasis from drugs, the accommodation is temporarily suspended.

CONTRACTION OF THE PUPIL.—Abnormal contraction of the pupils is due either to irritation of the pupil-contracting centre or fibres, or to paralysis of the sympathetic.

In disease of the central nervous system, the myosis may be due to irritation of the sphincter nucleus; but should mydriasis follow the myosis, it is an indication of the spread of the affection and destruction of the sphincter centre.

In irritative myosis the pupil rarely dilates under cover or in a bright light, but acts normally when a mydriatic or myotic drug is instilled. In the paralytic myosis the reaction to light and in convergence is preserved, but the pupils dilate imperfectly when shaded. Mydriatics act imperfectly, but the pupils contract further to myotics.

In old age the pupils are usually smaller than in middle life, although perfectly normal in reaction. Inflammations of the iris are always associated with small pupils, and the iris is likely to become attached to the lens capsule.

Myosis is seen in the early stages of inflammation of the brain and meninges, apoplexy, abscess, and in other affections which indicate irritation of the part; also in hysteria, toxæmia, and in epilepsy. Paralytic myosis occurs in tabes, general paralysis, spinal meningitis, and destructive lesions of the cord.

UNEQUAL PUPILS (anisocoria) may point to purely functional affections, such as hysteria and the psychoses, or to grave organic disease, as paresis, tabes, etc. The pupillary phenomena must be studied in connection with other symptoms to arrive at a correct diagnosis. Inequality of the pupils, although the reaction to light remains, is present in many cases of exophthalmic goitre. Bichelonne believes that unilateral mydriasis is an important sign in the early diagnosis of pulmonary tuberculosis.

Alternating mydriasis, in which the dilatation changes from one eye to the other, is occasionally present in general paralysis and in tabes, and has been described as a premonitory symptom of insanity.

HIPPUS.—An alternate contraction and dilatation of the pupil, occurring under a uniform stimulus of light, is a normal phenomenon, but may be excessive in hysteria, epilepsy, advanced paralysis, early stages of meningitis and mania, and in phthisis.

HEMIOPIC PUPILLARY INACTION.—The Wernicke pupil is described under hemianopsia.

Iritis.—Inflammation of the iris may accompany disease of or traumatism to other ocular structures, or be due to constitutional disorders. The principal signs are changes in the color of the iris, injection of the pericorneal vessels, myosis, and attachments of the iris to the lens capsule. The symptoms are severe brow pain, worse at night, and slowly failing vision.

Syphilis is the most common cause of iritis. In the secondary stage the iritis is plastic, and in the tertiary stage, plastic and gummatous.

The iritis of rheumatism is usually unilateral, although the second eye may later become affected. In chronic rheumatic subjects the iritis is of a severe and destructive type. The attacks usually recur during a relapse of the rheumatism, or they may be the only evidence of the rheumatic poison. The so-called idiopathic iritis, in which syphilis, gonorrhœa, and traumatism can be positively excluded, is probably due to the so-called gouty or rheumatic diathesis. In these cases the pain is usually of greater severity, the disease more slowly amenable to treatment, and the relapses frequent.

In severe inflammations of the iris, the ciliary body is involved, and the disease is referred to as iridocyclitis. Uveitis, or inflammation of the iris, ciliary body, and choroid, occurs in rheumatism and gout, diabetes, influenza, anæmia, syphilis, tuberculosis, and the specific fevers. The disease, which is probably the manifestation of some toxic process, is characterized by moderately deep anterior chamber, hazy cornea and aqueous, pupil not contracted, occasionally a slight increase in the tension of the eyeball, and the deposit in triangular form of small dots on the posterior surface of the cornea.

Iritis is often seen during the late stages of gonorrhœa, usually affects both eyes, and recurs with relapses of gonorrhœa and with the appearance of the gleety discharge.

Although tubercle, particularly in the miliary form, is occasionally found in the iris and choroid, it is of little value in general diagnosis, since the deposits in the choroid are seen at a time when the disease has shown itself in other regions so plainly that the diagnosis is easy.

Ocular Muscles.

Mobility of the Eyes.—Under normal conditions, the eyeballs move in perfect accord in all directions, with no manifest lagging movement in either eye in any of the several rotations. In equilibrium of the ocular muscles, every movement of one eye is accompanied by simultaneous and equal movement of the other, the image of the object upon which the eyes are fixed is formed on the fovea of each eye, and the effort required on the part of any one muscle or group of muscles in sustaining binocular single vision is equal in the two eyes.

Disturbance of equilibrium may be arranged in two groups:

1. Organic anomalies, in which there is double vision in attempts to rotate the eyes in the direction of the affected or paralyzed muscle or group of muscles.

2. Functional anomalies, in which there are:

(a) An *actual* deviation of the visual line of one eye from that of the other, persisting in all movements of the two eyes.

(b) A *tendency* to deviation, which is overcome by increased or decreased innervation to the muscle or group of muscles affected.

Organic Anomalies (Ocular Palsies).—Since binocular single vision can only be maintained if the image of the object falls upon the macula of each eye or upon corresponding points of each retina, any disturbance of the motor apparatus by palsy of one or more of the ocular muscles results in an impression of the object upon non-corresponding points of each retina. Two images are, therefore, transmitted to the brain, and double vision, or diplopia, results. The symptoms of ocular palsies are: (1) diplopia; (2) limitation of movement of one or both eyes in the direction of the paralyzed muscle; (3) actual deviation; (4) false projection; (5) vertigo; and (6) abnormal position of the head.

DIAGNOSIS OF OCULAR PALSIES.—Paralysis of an ocular muscle is to be suspected if the patient complains of seeing double or tilts the head to prevent diplopia, and complains of vertigo in attempts to fix an object in that portion of the field in which double vision exists. If the paralysis is complete, the eye with the affected muscle fails to rotate past the median line when the object fixed passes to the side to which the affected muscle ordinarily rotates the eye, and in fixation with the affected eye, the deviation of the sound eye (*secondary deviation*) is greater than is the deviation of the squinting eye when the sound eye fixes (*primary deviation*).

DIPLOPIA.—In partial paralysis the limitation of rotation may be so slight as to escape observation. It becomes manifest, however, even in slight degrees, upon the tests for diplopia. The patient, seated in a darkened room, with the head fixed in one position, is directed to follow with the eyes a lighted candle held at a distance of about 10 feet, and moved in all portions within the field of vision. If a piece of colored glass is held before one eye, the images of the two eyes are in this way differentiated. By this test the behavior of the two images in their relative height and distance from each other, and their separation and approximation, as the light is carried up and down, to the right and to the left of the patient, determines which of the muscles is palsied. Special skill and training are essential in the diagnosis of the more complex forms of palsies, and it is unnecessary in this connection to enter fully into details, but the following points will serve to indicate roughly the character of the affection:

1. Double images are seen only when the eyes are turned in the direction in which the paralyzed muscle or muscles normally rotate the eye; in all other directions there is single vision.

2. The image of the eye with the paralyzed muscle (false image) separates from the image of the sound eye (true image) as the object is carried into the field governed by the muscle affected; that is, the distance between the double images increases as the object fixed upon is moved in the direction toward which the paralyzed muscle should rotate the eye.

If the false image is on the same side as the affected eye the diplopia is homonymous; if the false image is projected to the side of the sound eye the diplopia is crossed, or heteronymous.

Homonymous diplopia, with images in the same horizontal plane, indicates paralysis of an external rectus, right externus if the images separate as the object fixed is carried to the right, and left externus if they separate as the object fixed is carried to the left.

Crossed diplopia in the horizontal plane indicates paralysis of an internus, right internus if the double images separate in looking to the left, and the left internus if they separate in looking to the right.

Vertical diplopia in upper field (that is, one image higher than the other) indicates a paralysis of the superior rectus or inferior oblique: if diplopia increases in looking up and to the right, and image of right eye is higher, paralysis of right superior rectus; if lower, left inferior oblique. Increase in diplopia in looking up and to the left, with image of right eye higher, paralysis of right inferior oblique; if lower, left superior rectus.

Vertical diplopia in lower field [illegible] rectus or superior oblique. Increase in [illegible] right, with image of right eye lower, indicates paralysis of right inferior rectus; if higher, left superior oblique. Diplopia increasing down and to the left, with image of right eye higher, shows paralysis of right superior oblique, if lower, left inferior rectus.

Special Palsies.—PARALYSIS OF THE SIXTH NERVE.—The long course of the sixth nerve at the base of the brain renders it particularly liable to pressure from inflammatory exudation, hemorrhage, and fracture. It is the most frequent of the ocular palsies, and it is indicated by convergence of the affected eye, homonymous diplopia, and inability of the eye to rotate outwards past the median line.

PARALYSIS OF THE THIRD NERVE is shown by ptosis, the pupil moderately dilated and unresponsive, the power of accommodation abolished, and crossed diplopia, with the eyeball turned outward and slightly downward from the action of the external rectus and superior oblique. In cycloplegia only that portion of the nerve controlling the ciliary muscle is affected. There may or may not be associated paralysis of the sphincter of the pupil (iridoplegia).

PARALYSIS OF THE FOURTH NERVE, which controls the superior oblique, is less frequent. There is vertical diplopia in the lower field, the image of the affected eye is the lower, and the distance between the images increases as the eye is rotated downwards and inwards.

OPHTHALMOPLEGIA EXTERNA is the term employed to designate paralysis of all the external ocular muscles. The affected eye is incapable of movement, and the lid droops and cannot be voluntarily raised. Paralysis of the iris and ciliary muscle is known as *ophthalmoplegia interna*.

CONJUGATE PALSY.—In this rare affection the individual muscles of each eye possess their normal power to turn the globe in any desired position, but there is inability to rotate the two eyes in associated action. It may affect convergence, so that the eyeballs cannot be converged, although individually capable of internal rotation; or it is shown in loss of associated lateral or vertical movements. In all cases the lesion is central, and involves the centres for conjugate movement, although spasmodic conjugate deviation is seen in hysteria.

Causes of Ocular Palsies—THE SEAT OF THE LESION in paralysis of the ocular muscles may be intracranial, orbital, or peripheral: it may include meningitis, tumors, hemorrhage, gumma, or vascular changes in the brain; orbital cellulitis, traumatism, and inflammation of the nerve in the muscle. The constitutional causes are syphilis, tuberculosis, diabetes, nephritis, influenza, tabes, rheumatism, and general paralysis of the insane, and toxic agents.

At least one-half of the ocular palsies are considered to be due directly to syphilitic gummatous deposits, syphilitic periostitis in the orbit or along the base, or to degeneration in or close to the nuclei of the nerves. These are exclusive of the indirect syphilitic affections, as manifested in tabes, general paresis, and diseases of the blood-vessels. Nuclear and peripheral palsies may be caused by rheumatism, diabetes, tonsillitis, influenza, ptomaine poisoning, and by lead, alcohol, tobacco, and other toxic [illegible]. In that variety of ptomaine poisoning known as botulismus, [illegible] are frequent. Basal palsies are seen in hemorrhage, meningitis, especially tubercular, abscess, and cavernous sinus disease. The paralyses associated with diabetes occur only in diabetes mellitus, develop suddenly, but usually are of short duration and most frequently affect the sixth nerve. Neuralgia of the region about the eye is often associated with the paralysis, so that pain in this situation in saccharine diabetes should direct attention to a possible disturbance of the motor apparatus on the same side.

OPHTHALMOPLEGIA INTERNA, or paralysis of the ciliary muscle and the sphincter of the pupil, is more frequently unilateral than bilateral, and is seen in syphilis, tabes, and intracranial disease. Either the sphincter or the ciliary muscle may be first affected, and later the external ocular muscles become implicated. It is also found after diphtheria. The lesion is probably nuclear.

PARALYSIS OF THE ACCOMMODATION, destroying the power of reading, is seen in about 5 per cent. of cases of diphtheria, usually affects both eyes, and only rarely is associated with palsy of the iris. Occasionally paralysis of the external rectus is associated with the loss of accommodation. The same palsies are also seen in severe cases of influenza, in multiple sclerosis, and in ptomaine poisoning.

INTERMITTENT PALSY of one or more muscles is frequently one of the early symptoms of tabes. One eye is generally affected, and the paralysis disappears in a few weeks to again recur. The same is found in syphilis, but the paralysis affects more than one muscle. The external rectus is probably the most frequently involved, and next the muscles supplied by the third nerve, either as a group or individually, while the parts supplied by the fourth nerve are rarely affected.

PALSIES OF SOME OF THE OCULAR MUSCLES, most frequently those supplied by the third nerve, are present in "ophthalmoplegic migraine" and follow the subsidence of pain. The attacks are usually recurrent, the palsy occurring on the same side as the pain. The disease is rare, and should be differentiated from brain tumor.

Functional Anomalies.—Both of the functional defects, the *tendency* to deviation (heterophoria) and the *actual* turning of one visual line from that of its fellow (heterotropia, or functional squint), are due in many instances to errors of refraction, and to disturbance of the relation between convergence and accommodation. There is no paralysis and no double vision.

LATENT DEVIATIONS (HETEROPHORIA, INSUFFICIENCY OF THE OCULAR MUSCLES).—If there is a lack of equilibrium in the action of the muscles of the two eyes in binocular vision, so that fixation of the eyes is only maintained through an excessive amount of nerve force expended in helping the weak muscle or set of muscles, there follows a train of symptoms which is usually included under the term *muscular asthenopia.* There is more or less constant dull headache, which may be general or localized in the frontal or occipital region, blurred vision, inability to use the eyes at near work, and photophobia. Sometimes there may be vertigo and nausea, confusion of ideas, insomnia, and a feeling of physical exhaustion while in a moving crowd, in attendance at the theatre, or after riding in the cars. Heterophoria is a most active causative factor in many of the reflex nervous disorders. Relief in many cases has undoubtedly followed the correction of the defects, but does not justify the extravagant claims made that epilepsy, chorea, melancholia, dyspepsia, and other affections are not only primarily due to heterophoria, but are cured after correction of the muscle anomaly.

Forms of Deviation.—The tendency of the visual lines to deviate from the normal parallelism is divided into *esophoria*, a tendency of the visual lines to turn inward; *exophoria*, a tendency of the visual lines to turn outward; and *hyperphoria*, a tendency of one visual line to deviate above that of its fellow. The inward tendency of the visual lines is of relatively less importance as a cause of reflex symptoms than is hyperphoria or exophoria.

To determine the existence of the muscle anomaly, the latent defect is made manifest by means of a prism of sufficient strength to cause diplopia, or by the use of a piece of cobalt glass or a rod of glass held before the eye. The line of light made by the rod is so dissimilar from the image of the other eye that the fusion impulse is abolished, and the eyes take the position of greatest rest. The prism that fuses the double images made by the prism or brings the line of light into the flame seen by the other eye is the measure of the defect. Correction of the refraction is essential to a cure.

MANIFEST DEVIATIONS (CONCOMITANT SQUINT, HETEROTROPIA).—In this affection there is an actual deviation of one visual line from that of the other, but the squinting eye is able to follow the movements of the fixing eye in all directions; there is no acknowledged diplopia, and the deviation is transferred from one eye to the other, and remains of the same degree upon alternately covering one eye and then the other. The absence of double vision, and the fact that the power of rotation of the eye is not limited, serve to distinguish the functional from the paralytic squint.

Functional squint may be either convergent, divergent, or vertical. The three principal causes of the strabismus are a disturbance in the normal relation between convergence and accommodation, brought into existence by errors of refraction; a weakness of opposing muscles, either through structural changes or disturbed innervation; and unequal vision of the two eyes, so that the normal desire for fusion is abolished. The strabismus may be monolateral, when one eye always fixes and the other always squints; or alternating, when either eye may be used for fixation, since the visual acuity is about the same in each. Squint is an affection of early childhood, often disappearing if proper treatment is instituted at this time.

Vision.

AFFECTIONS OF VISION.

Imperfect vision is due to errors of refraction; to opacities of the cornea, crystalline lens, or vitreous; to disease of the retina, choroid, optic nerve, or central nervous system; or to functional neuroses.

Central vision is tested by means of letters corresponding in size to a fixed standard. The patient, seated 20 feet from the test card, and one eye covered, is asked to read the smallest line of letters that can be deciphered. If the vision thus estimated does not conform to the standard, the various errors of refraction should be excluded before concluding that the reduced vision is the result of disease. The effect of faulty vision upon the health of patients is oftentimes overlooked. In a person given to any manner of indoor vocation, whose nervous system is at all delicately balanced, an uncorrected eye-strain may give rise to headache, drowsiness, transient vertigo, and sometimes to nausea, irritability of temper, and insomnia. These symptoms are probably more often found when vision is in excess of the normal standard, hence the state of the refraction must be learned in order to determine the extent to which the accommodative strain is responsible for the reflex manifestation.

Peripheral Vision.—In testing the perception of the outlying portions of the visual field, the examination is made of each eye separately, the oculist employing an instrument known as a perimeter, which consists of an arc of a circle, of about 12 inches radius. The eye to be examined is at the centre of the circle, and fixed steadfastly upon a white spot upon the arc. A white object 5 to 10 mm. in size is slowly moved along the arc, from its extremity towards the fixed spot, until it comes within the patient's range of vision, and the point recorded at which the object is first seen. The arc is moved to another position and this is continued until the whole circle has been tested. The record of the usual points so taken is recorded, as in Fig. 150. As will be seen, the outlines of the visual field are far from symmetrical. Its greatest extent is on the temporal side, usually about 90°, on the nasal side 55°, above 50°, below 65°. The perimeter is not absolutely necessary to make out gross lesions such as hemianopsia or extensive contraction of the field, since the finger carried from point to point, as the patient gazes into the examiner's eye, will indicate marked departure from the normal limits. Accurate examination requires the services of the ophthalmologist.

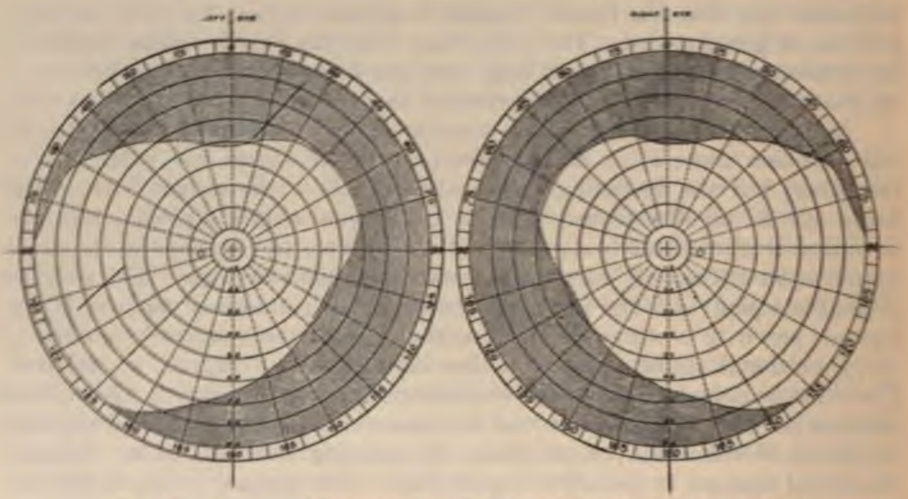


FIG. 150.—Diagram of perimetric charts of visual fields for white (form field).

Gradual failure of vision apart from refractive errors is seen in disease of the cornea, in cataract, non-inflammatory glaucoma, atrophy of the optic nerve, and various forms of intra-ocular disease. Rapid loss of sight occurs in acute glaucoma, retinal hemorrhages, embolism or thrombus of the central retinal vessels, œdema of the retina, cerebral effusions, metastatic disease of the eye, ptomaine poisoning, and after quinine, wood alcohol, and other toxic agents. In every instance of decrease in the normal acuity of vision, the oculist should be immediately consulted.

Cataract affects vision in proportion to the degree and situation of the opacity. It appears as a congenital or senile condition, in connection with disease of the eyes, in diabetes, in traumatism, and with many constitutional disorders that influence the nourishment of the lens through the nutrient vessels of the choroid and ciliary body. Cataract has been mistaken for non-inflammatory glaucoma, owing to the greenish reflex of the lens in the latter disease. The diagnosis is readily made with the ophthalmoscope.

Second Sight.—The ability of persons past middle life to lay aside their usual convex reading glasses and read the finest print (so-called second sight) indicates swelling of the lens, and is one of the first signs of cataract. Glycosuria is a frequent cause of cataract, and acquired myopia after 40 years of age, even with clear crystalline lens, should direct attention to the possible existence of diabetes.

Acute Glaucoma.—Recurring attacks of blurred vision, the obscuration lasting from a few minutes to an hour or more, when associated with halos about the light (iridescent vision), should direct attention in persons past middle life to the possibility of an oncoming attack of acute glaucoma. The "glaucomatous attack" usually occurs at night, is characterized by severe pain in the head, nausea and vomiting, and rapid loss of sight. The eyeball is intensely congested, the pupil dilated, the cornea anæsthetic and steamy, and the globe of stony hardness. The affection should not be mistaken for a "cold in the eye," iritis, or neuralgia. The rheumatic and gouty diathesis is a possible causative factor.

Alterations in the Visual Field.—Changes in the visual field, as evidenced by irregular or concentric narrowing of the normal limits for form and color, the presence of central or peripheral areas of lost perception (*scotoma*), or transpositions of the order of colors, is seen in disease of the retina, optic nerve, and central nervous system, or may be present in purely functional neuroses.

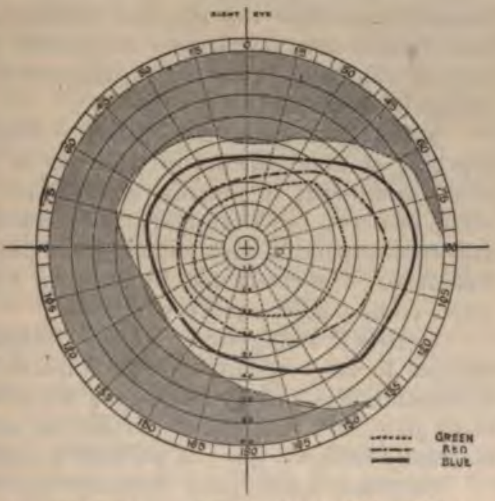


Fig. 151.—Diagram of form and color fields of right eye.

Amblyopia and **amaurosis** designate defective vision due either to functional disturbance or to actual disease of the visual apparatus, without gross ophthalmoscopic changes, although the latter restriction is not always adhered to. The affection of the sight may be limited to central vision, include the whole or only part of the visual field, or be only for form or for color. A number of congenital forms of amblyopia are recognized—for form, as in the poor vision of squint, or for color, as in color-blindness. Partial or complete loss of sight may be due to irritations affecting the fifth nerve, severe injuries of the head, autointoxication, the nephritis of the eruptive fevers, diabetes, malaria, rheumatism, action of certain drugs, and to hysterical manifestations.

Sudden transient failure of vision may mean merely the temporary giving out of eyes already weakened by general affections or too persistent use.

In the so-called "visual aura" of migraine, there is a decided blurring of the visual field, which has been designated as amblyopia, but is transitory, and is to be distinguished from the permanent functional impairment of sight included in the term.

Dercum regards a slight degree of amblyopia, with or without a diminution of the color sense, as an early and invaluable symptom of paresis, which may even antedate distinct and demonstrable anomalies of the pupils or changes in the eye-grounds.

Transient blindness, persisting for a few minutes to several hours or days, may be due to spasm of the retinal arteries. The diminution in the calibre of the vessels has been observed in epilepsy, migraine, cold stage of malarial fever, and in some toxic conditions.

In uræmia, particularly in the nephritis of scarlet fever and of pregnancy, the sudden loss of sight may be associated with convulsions, coma, and other cerebral symptoms. Although the blindness may be complete, the reactions of the pupils are usually preserved.

AMBLYOPIA FROM LOSS OF BLOOD.—Amblyopia, with subsequent complete atrophy of the optic nerve, may follow profuse spontaneous hemorrhages from the stomach, intestines, uterus, or nasal cavity. The loss of sight may not appear for a week or more after the bleeding, being due, as shown by Holden, to degeneration of the ganglionic cells of the retina from impaired nutrition.

METHYL-ALCOHOL AMBLYOPIA.—Rapid loss of sight may follow the drinking of wood alcohol in its crude or purified state, or when employed as an adulterant in the manufacture of Jamaica ginger, impure whiskey, cheap essences, bay rum, and other alcoholic beverages. The eye symptoms are often associated with vomiting and purging, severe headache, and intense weakness. The vision may improve for a few hours or days to again relapse, often ending in complete blindness.

QUININE AMBLYOPIA.—Total blindness may follow the taking of quinine in large quantities, the amount of the drug required varying in different individuals. The pupils are dilated and unresponsive, the optic disks pale, and the retinal circulation seriously restricted. Central vision is usually restored but the peripheral limits of the field remain contracted.

CENTRAL AMBLYOPIA (RETROBULBAR NEURITIS).—The orbital portion of the optic nerve is subject to interstitial inflammation in either an acute or chronic form. In both, the disease affects those portions of the nerve that supply the macular region. The early symptoms are dimness of vision, without marked ophthalmoscopic changes, and a weakness or loss of color perception in the central visual field.

In retrobulbar inflammations, as pointed out by Gowers, the visual acuity is less in very bright light, and exposure to excessive light may lead to deterioration of vision that may last for some time. This is due to the slowness with which the ill-nourished axis-cylinders are regenerated. The same author also shows the close relationship between retrobulbar disease and affections of the seventh nerve, since paralysis of the facial nerve may precede the optic-nerve inflammation.

In *acute retrobulbar neuritis* there is rapid failure of vision with central or paracentral scotoma, which is usually followed by recovery of vision, although the optic disk still shows pallor. The affection may, however, rapidly progress until the entire nerve is implicated, and vision is nearly if not completely lost. The disease may arise during the course of rheumatism, gout, diabetes, smallpox, and other general affections, in which the blood carries the toxæmic substance; or may follow orbital or sinus disease, menstrual suppression, alcohol or lead intoxication; and occasionally is found in insular sclerosis and myelitis.

In *chronic retrobulbar neuritis* there exists with dimness of vision a small central color scotoma, particularly for red and green, the horizontal oval area in the visual field extending from the fixing point to the blind spot. The affection is found principally in persons using large quantities of tobacco, especially when combined with the use of alcohol. It is most frequently noted between 40 and 50 years of age, and has also been found in alcoholics who are not users of tobacco, and from the toxæmia of lead, cannabis indica, stramonium, chloral, carbon bisulphide, iodoform, etc. The disturbance of vision is greater for near objects, and is more marked in bright light.

A form of retrobulbar neuritis similar to that of toxic origin appears as an hereditary affection, and is referred to as *hereditary optic neuritis*. It affects several members of a family, especially the males, and has been traced through several generations. The exciting cause is exposure to cold, syphilis, excessive venery, and the heavy consumption of tobacco.

Hemianopsia (hemianopia) is a loss of one-half of the visual field of one or both eyes, due to a lesion in the optic chiasm, along the optic tracts, or in the visual centres in the occipital lobe. It does not include defects in the field caused by disease within the eyeball. The line dividing the seeing from the blind field is horizontal or vertical, or nearly so, and may cut exactly through the fixing point, or circumscribe this point by a small zone of preserved vision. (See Fig. 387, p. 1309.)

The dividing line may have an oblique direction, but this is extremely rare, or only a sector, commonly a quadrant, of the field may be wanting.

Hemianopsia is classified according to the relative position of the blind portions of the two fields. It is *homonymous* if there is loss in the corresponding halves of each field; *bitemporal* if both temporal fields are blind, and *binasal* when the nasal halves are lost. When the dividing line between the lost and preserved field is vertical, the defect is known as *vertical hemianopsia,* and when the dividing line is horizontal, the hemianopsia is *horizontal or altitudinal.*

HOMONYMOUS HEMIANOPSIA is the commonest form, and reveals itself as a defect in the right or left half of each visual field. For instance, in

Fig. 152 the left half of each field is wanting, showing loss of function in the right half of each retina. If the right half of each field is lost the condition is *right lateral hemianopsia;* in loss of the left half of each field, *left lateral hemianopsia.* The seat of the lesion in homonymous lateral hemianopsia is in any part of the visual tract between the chiasm and the occipital lobe.

BITEMPORAL HEMIANOPSIA is a comparatively rare phenomenon, but one of great diagnostic moment when found. It manifests itself as a blindness of the outer, or temporal, halves of the visual fields, indicating suspended function of the nasal portions of each retina. It is caused by a lesion which destroys the function of the crossed fibres without affecting the uncrossed fasciculi. This may be a tumor, fracture, exostosis, aneurism, or disease of the blood-vessels. Loss of the two temporal fields is seen in acromegaly, although it is not a constant symptom, since the type of hemianopsia will depend upon the direction the pressure is exerted upon the chiasm and tracts.

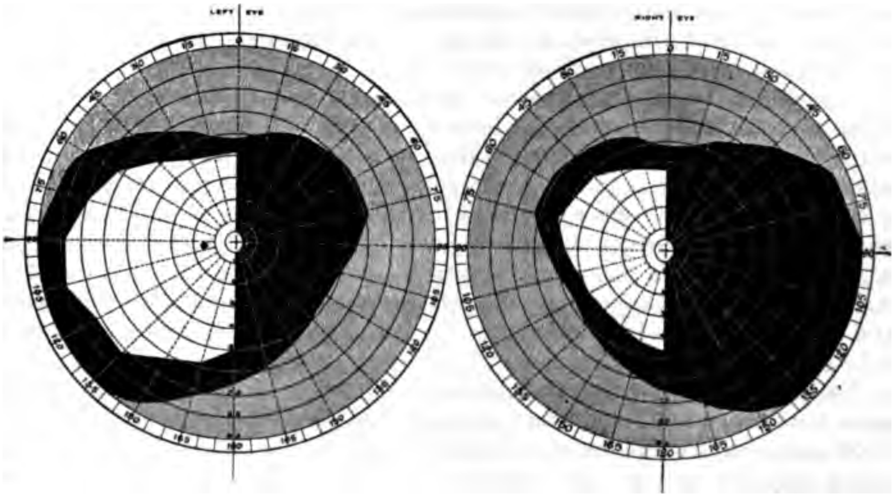


FIG. 152.—Diagram of perimetric charts of right lateral hemianopsia. The dark areas show loss of the nasal half of left and temporal half of right fields, with contraction of the preserved fields. The dividing line passes around fixing point.

BINASAL HEMIANOPSIA, in which both the nasal fields are lost, is rare. If it is true that the crossed and uncrossed fibres of the optic nerve are mingled at the outer half of the chiasm, then a lesion of this structure cannot cause binasal hemianopsia. Shoemaker believes that this defect in the fields is due to an inflammation of the optic nerves.

Both upper or both lower fields may be wanting. In this condition, the lesion is, as a rule, at the chiasm, encroaching on it from above or below.

If the blind halves of the field have lost not only perception of form and light, but also of color, the defect is *absolute;* if only recognition of color is lost, the hemianopsia is *relative.*

HEMIANOPSIA AS A DIAGNOSTIC SYMPTOM.—In lateral hemianopsia the intracranial lesion is on the opposite side from the dark fields. If unassociated with motor or sensory symptoms, the lesion is confined to the cuneus, or the immediately surrounding gray matter; a lesion in one nerve tract, or in the primary optic centres, with symptoms of basal disease, would cause changes in the pupil, and possibly some affection of the nerve head could be recognized. Hemiplegia and hemianæsthesia are often present with lateral hemianopsia, indicating organic disease of the brain, the lesion being situated in the internal capsule. If right hemiplegia and aphasia are associated with lateral hemianopsia, an extensive lesion probably exists of the area supplied by the middle cerebral artery. A lesion of the posterior gray matter of the optic thalamus could produce lateral hemianopsia, with hemianæsthesia and ataxia of one side of body. A cortical lesion is usually associated with concentric contraction of the preserved fields, or is found in cases in which the light sense is preserved, but the color or form sense is abolished.

HEMIANOPIC PUPILLARY-INACTION SIGN.—This is an important localizing sign in hemianopsia, and consists in carefully noting if the pupil reacts to a beam of light thrown upon the non-functionating half of the retina. It is an extremely delicate test to make, owing to the difficulty of restricting the beam of light so that it shall illuminate the non-acting half of the retina without allowing any light to fall upon the seeing half. If the pupil reacts when the light is thrown upon either the blind or the seeing half of the retina, the lesion is back of the primary optic centres; but if there is no reaction when the light falls upon the blind side, but the pupil reacts when the light falls upon the functionating side, the lesion is in front of the primary optic centres, and in that position has affected the motor arc of the pupil. The test should always be made in a well-darkened room, with barely sufficient light to conduct the examination, and should be confirmed by a second observer before basing a diagnosis on its apparent presence. When present it is a valuable sign, but its absence is not decisive, owing to the difficulty of making the test.

Hysterical Amaurosis.—The diagnosis of visual defects due to hysteria is sometimes difficult, although healthy eye-grounds and pupils normally reacting to light would point strongly to hysteria. Cases of hysteric blindness have been reported, however, in which light failed to have any action on the pupil.

If unilateral blindness arises suddenly, following fright, emotional excitement, slight injury, or menstrual pain, hysteria may be suspected. While the defect may be bilateral, it is more often unilateral. It is not uncommon to find, associated with the ocular symptoms, other disturbances of sensation, such as hemianæsthesia of the skin, cornea, or conjunctiva. If the amaurosis is restricted to one eye, under some conditions it may be transferred to the other temporarily; and, again, the unilateral character of the affection may entirely disappear in binocular fixation, as proved by the diplopia if a prism of sufficient strength to prevent normal fusion is placed before one eye.

Not only may the vision be reduced in hysteria, but changes in the peripheral field are common. The contraction in the field is usually equal in the different meridians, and is often of the tubular type, in which the limits of contraction remain the same, no matter what distance the test object is removed from the eye. The field for colors likewise shows concentric contraction, or the limits of one color may overlap that of another, or there may be a complete reversal of the colors.

Optic Neuritis.—Inflammation may affect the optic nerves at their intra-ocular portions (papillitis) or in their course in the orbit (retrobulbar neuritis). Under the term *hyperœmia of the nerve head* is included a type of optic-nerve irritation in which the disks become of dull red color, the surface and margins veiled, and the lymph sheaths of the vessels prominent. It is seen in refractive error, particularly hyperopia and hyperopic astigmatism, after long-continued exposure to intense light or heat, in some types of inflammation of the uveal tract, in orbital and sinus disease, in chronic insanity, and from toxic agents.

Papillitis.—Optic neuritis may be manifest as a true inflammation of the nerve tissue, a swelling of the intra-ocular ending of the optic nerve, or as a descending neuritis. The changes in the optic-nerve head may range from a decided redness, moderate swelling, and blurring of the margins. to an intense rounded protrusion of the disk from inflammatory exudation, reddish gray in color and sloping down into the surrounding retina, the retinal arteries shrunken, and the veins full and tortuous and covered in by infiltration or ending in numerous hemorrhages. Upon subsidence of the inflammation the nerve head becomes grayish white in color, the œdema subsides, and the extent to which the pressure has affected the nerve-fibres is shown by the degree of optic atrophy that follows.

A moderate degree of papillitis, associated with hemorrhages throughout the retina, few changes in the vessels, and spots of fatty degeneration of the retinal elements, is described as *neuroretinitis*, and is the type most frequently found in association with renal disease. The intense swelling of the papilla, with exudation and tortuosity of the veins, is termed *choked disk* or *papillœdema*, and is the usual type found in certain forms of brain tumor.

The neuritis may be due to affections of the orbit, such as fracture, orbital tumors, purulent cellulitis, and sinus disease. Intracranial causes are tumors, meningitis, gumma, abscesses, and aneurisms. The situation of the intracranial portion of the optic nerve tracts at the base of the brain renders them particularly liable to implication in inflammations of the basal portion of the meninges and to the pressure of tumors, abscesses, or aneurisms. In children, tubercular meningitis is usually accompanied by swelling of the optic disk. The absence of affections of the optic nerve does not preclude the presence of a new growth in the brain, although when the base, and particularly the cerebellum, is the seat of a neoplasm, swelling of the optic disk is almost always present. Double optic neuritis of high degree, rapidly progressive, and accompanied by marked exudation in the nerve and surrounding retina, usually indicates a tumor of the cerebellum, while one of slower growth, less intense, and either unilateral or considerably greater on one side than on the other, is seen in neoplasms of the cerebrum. The "stellate figure" in the macula, which is seen in a large proportion of the cases of renal retinitis, is not uncommon in the intense papillitis of brain tumor. Tumors or abscesses of the frontal region rarely cause optic neuritis, although swelling of the optic disk may occur.

Apart from the intracranial causes, papillitis may occur from general infections. These are in the nature of a toxin, occurring in such diseases as influenza, syphilis, malaria, rheumatism, erysipelas, and many of the exanthematous and continued fevers. Lead and alcohol may also cause inflammation of the optic nerve, and the same process is seen in anæmia, loss of blood, sunstroke, and after violent exertion. Syphilis may cause a primary neuritis or act secondarily through gumma of the brain or meninges.

Unilateral optic neuritis may be due to orbital or sinus disease, and in rare instances to cerebral tumor, in which the neuritis occurs on the side of the neoplasm. The inflammation of the retina and optic nerve of nephritis and certain constitutional disorders is often unilateral, but with the progress of the systemic disease the inflammation attacks the other eye.

Perfect central vision is usually unimpaired even in intense papillitis during the acute stage, and if defects in vision occur they partake of the nature of sector-like defects in the visual field.

Retrobulbar neuritis has been considered under Amblyopia.

Optic-nerve Atrophy.—Degeneration and atrophy of the optic nerves may be *primary*, when there has been no previous inflammation or swelling of the papilla, or *secondary*, if preceded by previous optic neuritis. In both forms there are changes in the color of the disk, varying from a gray to grayish white, with the edges usually clear and distinct in the primary forms, but veiled in the secondary.

Primary atrophy is more frequently associated with spinal disease, particularly locomotor ataxia, in which it usually appears before the ataxic symptoms. It is also found in insular sclerosis, paralysis of the insane, and occasionally in lateral sclerosis. It may occur as a result of excessive hemorrhage from the stomach, uterus, or intestines, in the toxæmia of fevers, alcohol or lead poisoning, in chronic malaria, syphilis, and diabetes, in fractures of the base, and in deformities of the skull. Hereditary optic-nerve atrophy is not uncommon, the atrophy appearing in early adult life.

Secondary or Consecutive Atrophy.—The contracted retinal arteries, the dilated and tortuous veins, and the veiling of the surface and edges of the optic nerve point to a previous papillitis. Extensive retinal and choroidal disease also results in atrophy of the nerve, as will pressure upon the nerve-fibres by an aneurism, tumor, or exostosis.

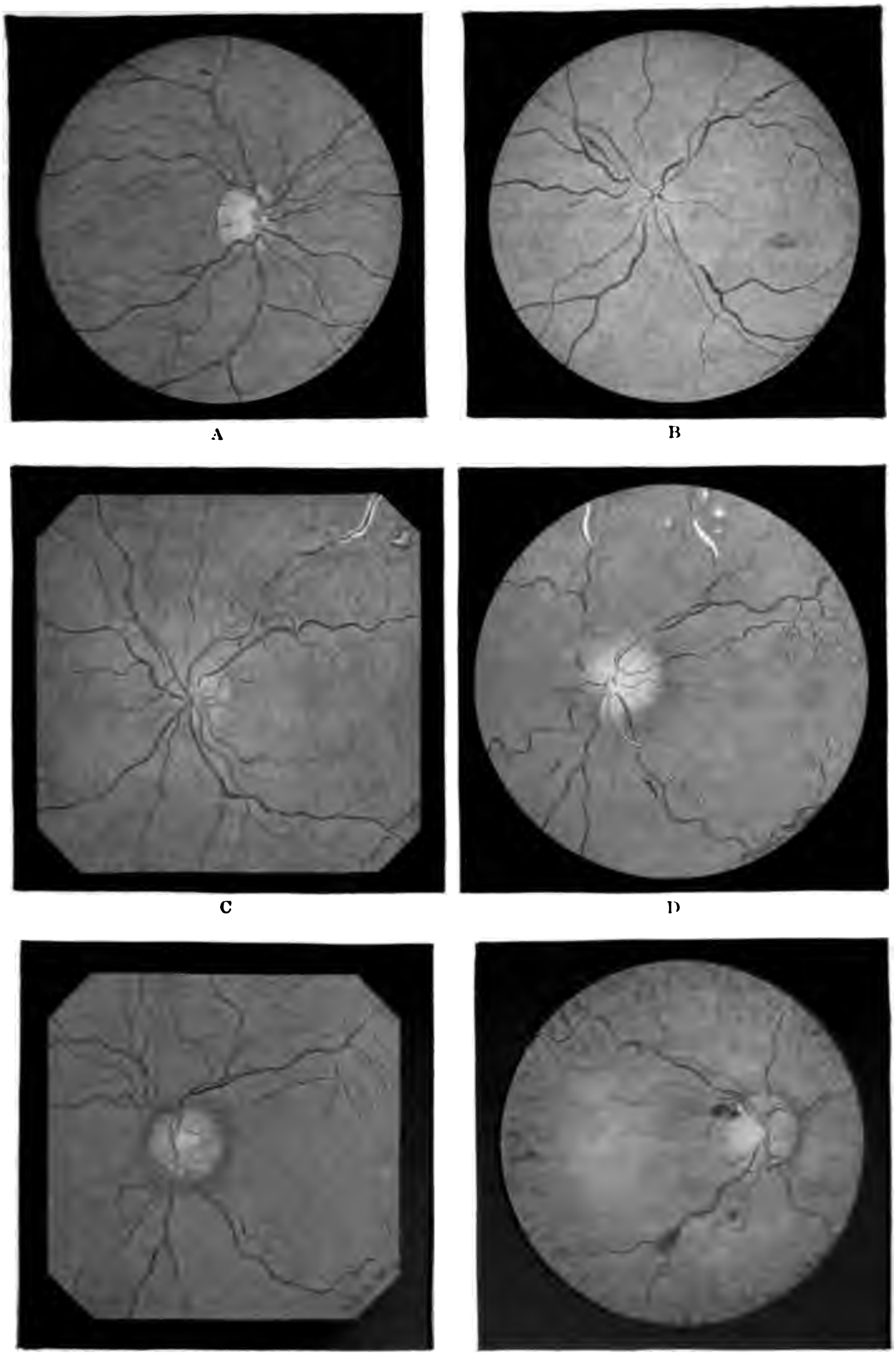
Retinitis.—The retina is implicated in disease affecting the intra-ocular end of the optic nerve, and also from extension of disease from the ciliary body and choroid. The inflammation is associated with œdema and exudation, hemorrhages either in the fibre layer or deeper, small-cell infiltration, and tortuosity of the retinal vessels, with changes in their calibre.

Retinal Hemorrhage.—Extravasation of blood into the retina may occur independently of any inflammation of the retina. It is usually the evidence of extensive vascular disease, organic heart affections, or suppressed menstruation. It may occur in scurvy, purpura, marked anæmia, diabetes, and particularly in the type of neuroretinitis associated with nephritis. Retinitis with hemorrhages resembling those seen in renal disease are often present in simple anæmia and chlorosis. The position and extent of the hemorrhage determines the effect on vision.

Arteriosclerosis.—A study of the changes in the retinal blood-vessels is of extreme importance as bearing on the early diagnosis of various phases of general arteriosclerosis. The early alterations in the retinal circulation which should direct attention to general symptoms indicative of beginning sclerotic changes are tortuosity of one or more of the smaller arteries, the evidence of undue pressure of an artery at its point of crossing of a retinal vein, and an increase of the light reflex of the arteries. At first the vein is simply displaced in the direction of the arterial circulation, and its flow slightly obstructed; later the venous current is markedly impeded, and the vein greatly narrowed where the arterial pressure is exerted, and is distended on the peripheral side. These changes are rarely accompanied by sufficient fibrous thickening to cause white lines of perivascular inflammation along the vessel. As the vessel walls lose their elasticity, the impediment to the flow of blood results in tortuous vessels, the escape of fluid into the surrounding tissues, and retinal œdema. These conditions are not due to old age only, but to actual sclerosis of the vessels from disease.

The importance of early recognition of these ocular changes lies in their association with similar disease of the brain and kidney. There is no difficulty in determining by the ophthalmoscope the evidence in the eye-ground of well-advanced types of arteriosclerosis, but it is important that recognition of these signs should be made before the disease has reached a point where treatment is ineffectual. De Schweinitz called especial attention to the value of early recognition of the signs, even though they be only suggestive, of angiosclerosis of the retinal vessels in persons who have reached the age at which vessel degeneration may begin to appear, and who consult the ophthalmologist for a change of reading glasses. These signs are "a corkscrew appearance of individual vessels, a slight thickening of the perivascular lymph sheaths, a beginning brick-dust appearance of the optic nerve-head, and a flattening of a vein against an artery or a bending in a curve of the vein overlying the artery." With these retinal conditions present the physician should carefully examine the cardiovascular system, and accurately test the arterial tension by approved means, and, should the tests confirm the retinal findings, institute appropriate treatment, which may save not only lesions of the eyes but of other structures, notably the brain, which, if they occur, may prove fatal.

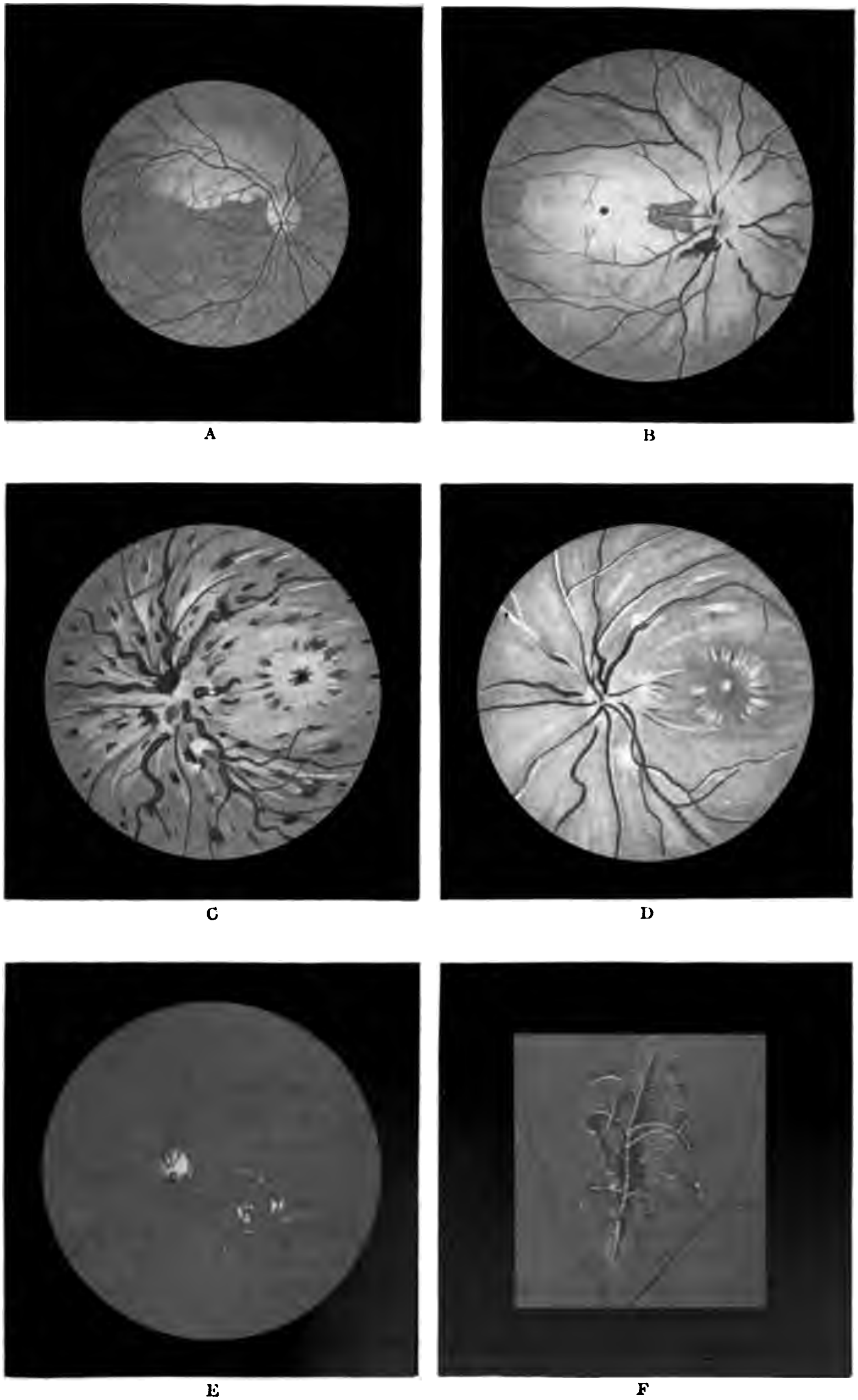
Obstruction of the Retinal Vessels.—An embolism may lodge in the central retinal artery or in one of its branches. Sudden blindness follows complete obstruction of the central vessel, whereas in plugging of one of the smaller vessels the blind area will correspond to the section of the retina supplied by the vessel affected. The fundus picture in embolism of the central artery shows a pallid disk, a grayish white œdema of the retina, and the appearance of a central red spot in the fovea. The affection presents the same general symptoms and changes in the eye-ground as in thrombus. Both occur in endarteritis, heart disease, and changes in the composition of the blood.



[CHANGES IN ARTERIOSCLEROSIS.—After De Schweinitz.

A, Normal fundus. B to F, successive changes occurring in arteriosclerosis, including pallid arteries (B), later assuming a silver-wire appearance (C); indented veins (B, C), afterward showing ampulliform enlargements (D, E); corkscrew capillaries (C, D); corkscrew arteries and veins (D, E); perivasculitis (C, D); sclerosis of vessels (F); œdema of disk (B, C, D, E), hemorrhages (C, F).—D.]

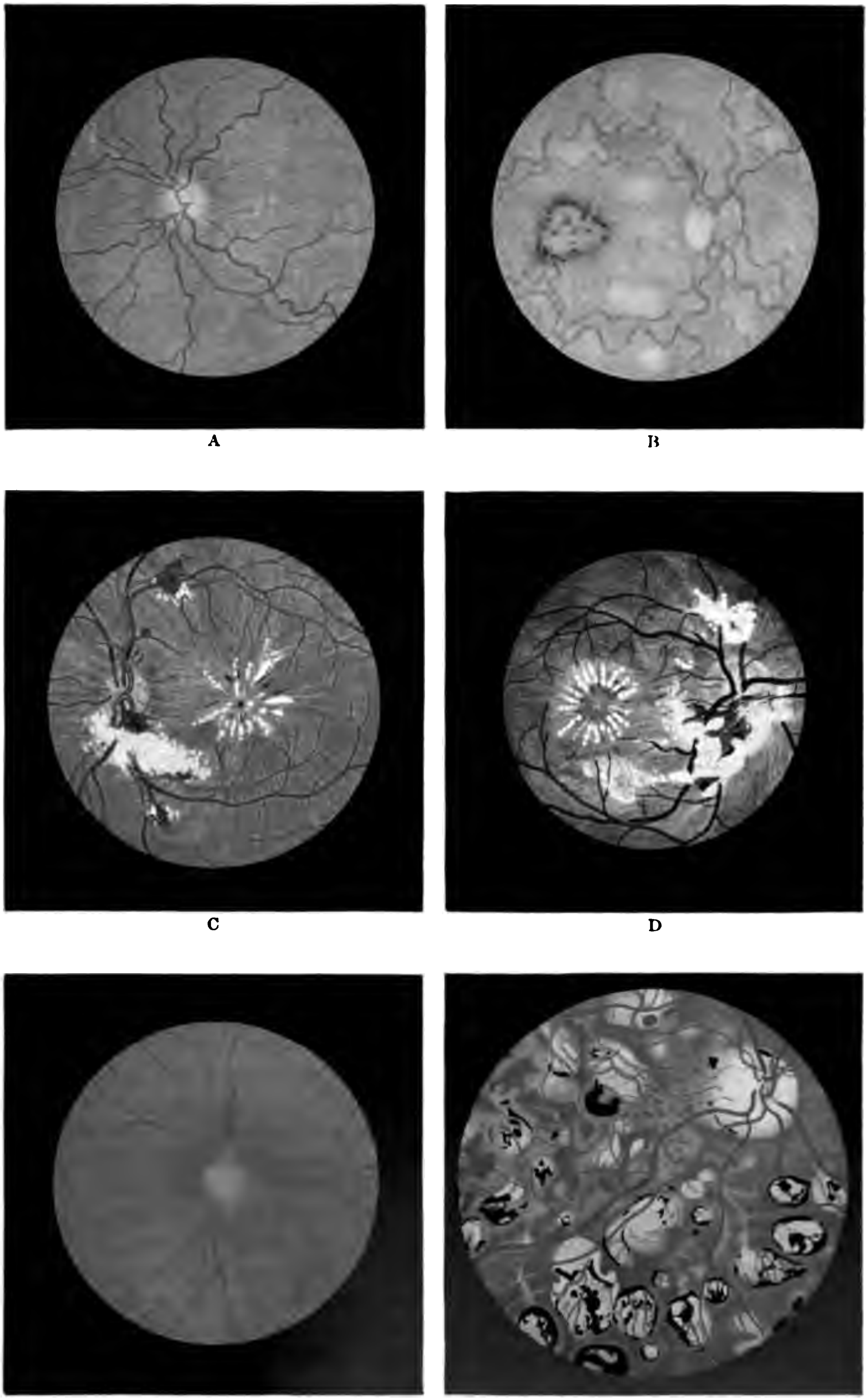
PLATE X.



[Changes in Retinal Vessels.—After Würdemann in Posey and Spiller.

A, Embolism central artery; partial, affecting only inferior branch (Haab). B, Embolism central artery; total within nerve; a cilio-retinal vessel supplies a small area of retina in which function is preserved (Würdemann). C, Thrombosis of central vessels from mumps (Würdemann). D, Same case six months later, showing sclerosis and atrophy (Würdemann). E, Hemorrhages from retinal vessels (Magnus). F, Perivasculitis luetica (Magnus).—D.]

PLATE XI.



A B C D E F

[INFLAMMATIONS OF THE RETINA.—After Würdemann in Posey and Spiller.

A, Œdema in pernicious anæmia (Oliver). B, Leucæmic retinitis (Oliver). C, Albuminuric retinitis and neuritis of pregnancy (Würdemann). D, Albuminuric retinitis in the negro (Würdemann). E, Syphilitic retinitis (Haab). F, Atrophy of retina, chorioid, and nerve following chorio-retinitis luetica (Oeller).—D.]

XI.

EXAMINATION BY MEANS OF RÖNTGEN RAYS.

By PROFESSOR MANGES.

Röntgen made his discovery in 1895. It was a new kind of energy, its true nature unknown, hence he termed it X-ray. Within a month from the time of his discovery he disclosed three facts which have made these rays the most important diagnostic agent in the field of medicine, viz., that they penetrate all objects to a degree somewhat in direct proportion to the atomic density of the object; that they have actinic properties similar to ordinary light in their action on photographic plates; and that they excite fluorescence in certain substances, notably barium platinocyanide and tungstate of calcium. For example, the rays will penetrate all of the structures of the hand, but they will pass through the soft parts more freely than through the bony parts; therefore, if the rays after passing through the hand are permitted to act upon a photographic plate, which is protected from other forms of light, the various portions of the hand will record their respective densities, and after the process of development the plate will become a permanent record of shadows, appreciable to the naked eye. Such a record is termed a skiagram, a radiogram, or preferably a röntgenogram. If a screen covered with an emulsion containing crystals of barium platinocyanide or tungstate of calcium be substituted for the photographic plate, and other forms of light be excluded, the Röntgen rays, in passing through the hand, will be obstructed according to the density of the various parts of the hand and one will see immediately the shadows on the fluorescent screen. Such a screen is known as a fluoroscope, or, better, a röntgenoscope. All Röntgen-ray examinations, then, are made by means of röntgenograms, or by means of the röntgenoscope. The diagnostic value of such examinations depends directly upon the technic and interpretative skill of the röntgenologist. In the hands of the inexperienced and untaught the results are often valueless or misleading; and, because of the modern powerful exciting apparatus, the patient is in danger of being injured.

The röntgenographic method of examination has a far wider field of usefulness than the röntgenoscopic. The röntgenogram is a permanent record, can be studied indefinitely, and shows much finer detail in shadows than can be seen by means of the röntgenoscope. The former is applicable to all parts of the body, whereas the latter has advantages only in the study of movable organs—*e.g.*, the thoracic viscera and the gastro-intestinal tract. Röntgenoscopic examination should be made only with the most protective apparatus and continued for as little time as possible, to insure safety to operator and patient.

Stereoröntgenograms give accurate depth relations and are most useful. To make stereoscopic röntgenograms it is necessary to have a plate holder which will permit of plates being inserted and removed without disturbing the patient or the X-ray tube holder. Two exposures are made, the focus tube being displaced two and one-half inches either laterally or longitudinally. All modern tube holders are supplied with automatic or easily adjusted displacement device. The resultant negatives are then viewed by means of a stereoscope. Reductions may be made from the original negatives and viewed by means of the ordinary hand stereoscope.

Examinations of the head should always be made stereoröntgenographically. In this way the diagnosis of lesions of the accessory sinuses, including the mastoid cells, is made with much accuracy. A sinus filled with pus is more nearly opaque to the rays than one containing air; even granulation tissue or retained mucus casts shadows distinguishable from the normal. The importance of this procedure becomes apparent when one considers the frequency of sinus or mastoid infection as complications or sequelæ to the infectious diseases. Intracranial lesions are shown by the effect they produce on the inner table of the skull or the sutures, or, if they possess a density greater than that of the brain substance, by the presence of foreign shadows. Any localized organic lesion of the cortex of the brain will produce changes in the inner table of the skull either by way of bone atrophy due to increased local pressure or by actual destruction of bone involved in the disease process. When intracranial pressure is increased by dilatation of the ventricles the convolutions of the brain soon produce impressions on the inner table which show clearly on the röntgenogram, and in the cases of children the skull sutures are shown to be more separated than normal. Acute external hydrocephalus causes the sutures to separate, but even the normal convolution impressions are absent. Disorders of the pituitary are manifested by some deviation from the normal in the röntgenographic appearance of the sella turcica. Diseases and abnormalities about the teeth may be shown clearly.

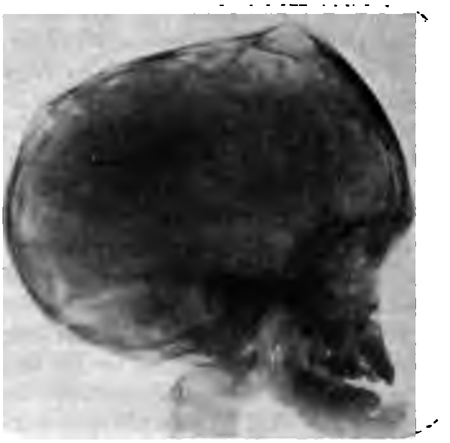


FIG. 153.—Convolution depressions very deep, due to early ossification of the sutures and moderate internal hydrocephalus. Child is a congenital syphilitic.

The thorax, because of the great contrast in density of its contained organs, is most accessible to Röntgen-ray study. One should become thoroughly familiar with röntgenograms of many normal chests before attempting to recognize the abnormal unless it has been carefully indicated by one who is skilled. Only in this way is it possible to appreciate the very early deposits of a tuberculous lesion or other infiltrating process, a thickening of the pleura, inflammatory thickening or dilatation of the bronchial tubes, enlargement of the peribronchial glands, the extent of lung involvement, the presence and extent of pneumothorax or of pleural effusion, enlargement of the thymus gland, existence of substernal goitre, and variations from the normal in the size and contour of the heart and large blood-vessels. Some of these conditions are diagnosed positively without other aid, but as a rule careful physical examination and clinical study are essential to correct differential diagnosis. For instance, the röntgenogram of enlarged peribronchial glands does not indicate whether the enlargement is due to tuberculous or other infection, and a history of measles, influenza, or other infectious disease is sufficient to limit the diagnostic value of the plate. Again, the infiltration of syphilis has much the same röntgenographic appearance as tuberculous infiltration, and so instances might be multiplied. All intrathoracic conditions except

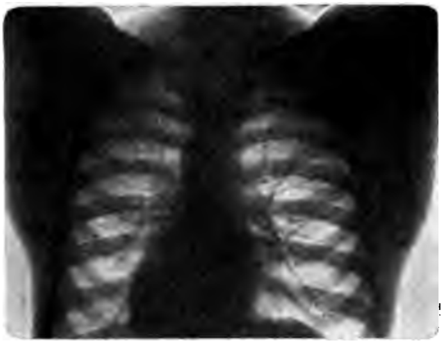


FIG. 154a.—Enlarged peribronchial glands. Lung shadows normal. (Viewed from the back.)

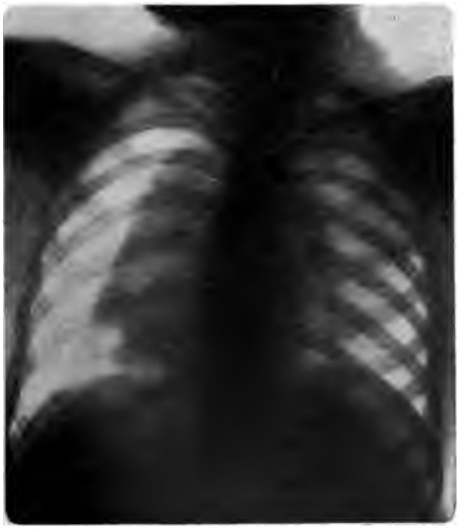


FIG. 154b.—Extensive pneumothorax on left side, slight displacement of heart. (Viewed from back.)

mediastinal tumors, lesions of the circulatory organs, and those involving the diaphragm are best revealed by the röntgenographic method. The presence or absence of pulsation, and the excursus of the diaphragm can only be determined by means of the röntgenoscope. With the modern apparatus one can make a tracing which shows accurately the size of the heart and its relations to other structures, the size and extent of pulsation of an aneurism, the excursus of the diaphragm, etc. The size of the heart in its lateral and vertical dimensions can be shown on a röntgenogram if the exposure is made with the tube at a distance of seven feet or more. Such a picture is known as a teleröntgenogram.

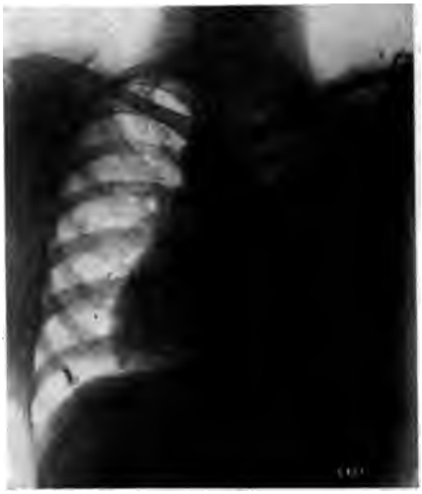


FIG. 155*a*.—Extensive pleural effusion on right side. very little displacement of heart Notice obliquity of upper level of fluid (Viewed from back.)

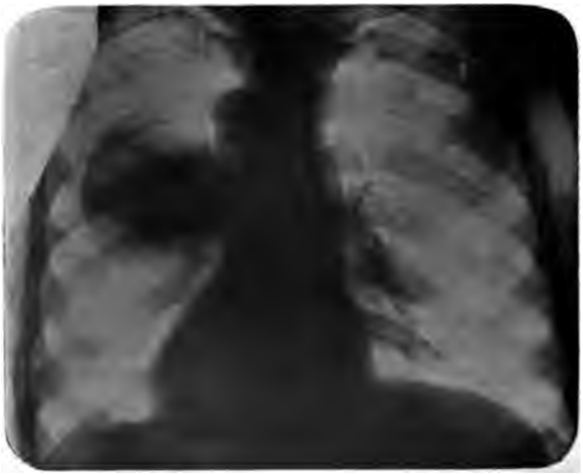


FIG. 155*b*.—Recurrent sarcoma of chest wall. Notice sharp outline of growth. (Viewed from back.)

Again, stereoröntgenograms are of the utmost importance in the study of lung lesions. Only in this way can one determine the depth and true size of isolated foci of disease or cavities. The earliest possible signs of pulmonary tuberculosis are minute areas of infiltration in the parenchyma and a matting or clumping of the very small branches of the bronchial tubes or bronchioles. Even in the normal chest these fine tubes can be traced to the very periphery of the lung if the röntgenograms are of excellent quality. The stereoscope enables one to see whether they proceed to the periphery discretely or whether they are matted together at points. The two plates must be exposed and interchanged in a period of time during which the patient can hold a full inspiration at complete rest. The tube should be displaced parallel to the longitudinal axis of the body. The patient may be in an erect or horizontal position, but exposures should be made from both front and back. The erect posture has the advantage in that a lung cavity partly filled with pus will show an upper level.

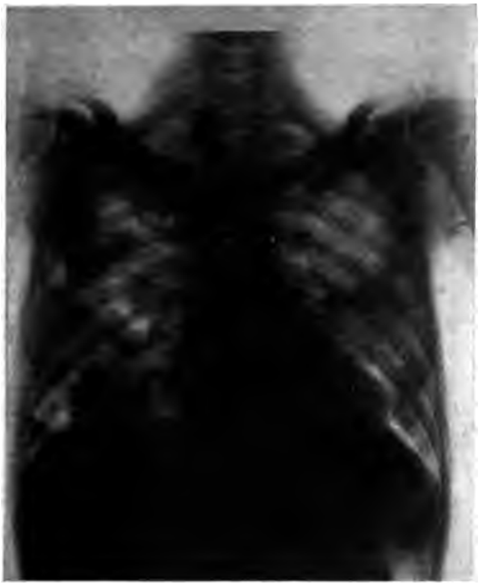


FIG. 156a.—Metastatic sarcomata of both lungs. (Front view.)

Pneumothorax is readily distinguished from every other condition by the complete absence of lung shadow within the area of the plate covered by the air-containing space. Occasionally pneumothorax and pleural effusion coexist. In such cases the examination must be made with the patient sitting or standing, when a sharp transverse upper level of dense fluid shadow will be seen with a less dense pneumothorax area above it. On fluoroscopic examination waves can be set up and seen in the upper level of the fluid by percussing the chest wall. These waves cannot be produced in simple pleural effusion, and the upper level of fluid is obliquely downward and inward. Purulent and non-purulent effusions cannot be differentiated by means of X-ray. Encysted effusions are determined by their more or less rounded form and by the fact that gravity does not determine the inferior boundary. They may be interlobar, or between the parietal and visceral pleura.

Localized or general thickening of the pleura is determined by comparative study of the unaffected portions, the thickened areas producing shadows considerably more dense than normal areas, but not so dense as an area of pleural effusion.

Röntgen-ray examination of the entire alimentary tract is made possible by the ingestion or injection of a substance opaque to the rays, or in certain instances the introduction of air which permits the rays to pass more freely. Bismuth oxychloride, bismuth subcarbonate, and barium sulphate (specially prepared) are the opaque substances in general use. Here the röntgenoscopic method is essential.

Diseases of the œsophagus are distinguished by their effect upon its lumen and course. A diverticulum will retain a portion of the bismuth and cast a shadow of localized, pocket-shaped enlargement. Organic narrowing of the lumen is apt to be irregular, whereas spasmodic stricture is usually abrupt and smooth in outline. The former is constant, the latter intermittent, relieved by antispasmodics, and not accompanied with marked loss in weight. The œsophagus is best seen when the rays pass obliquely through the chest. The calibre of a stricture can be determined by having the opaque mixture of such viscidity that it will pass slowly under swallowing pressure from above through the narrowed portion.

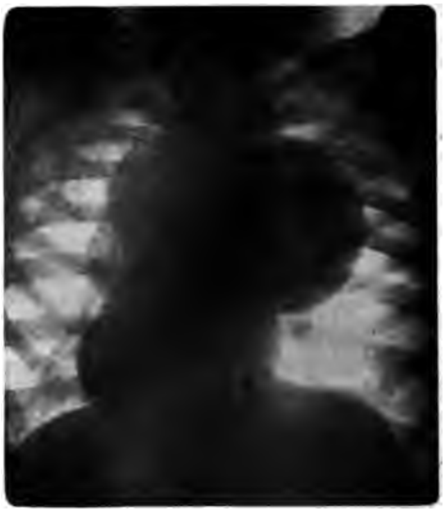


Fig. 156b.—Larger aneurism involving ascending transverse and descending arch. (Viewed from back.)

The wide variations in size, shape, position and relations of the gastro-intestinal tract, within normal limits, make the Röntgen-ray study of these organs a task of large proportions. The variations in diseased conditions are even greater, and then, too, the stomach is reflexly influenced by disease in adjacent organs. Broadly speaking, the normal stomach is one which lies with its upper two-thirds in the upper left quadrant of the abdomen, with the lower third crossing the median line so that the pylorus is from one-half to two inches to the right of the median line, and above the level of the umbilicus. The greater curvature is the lowermost portion, extending from the level of the umbilicus to two inches below this level. It fills gradually from above downward so that the entire lesser and greater curvatures can be seen with a comparatively small quantity of opaque meal of thick fluid consistency, and enlarges throughout when completely filled, the posture being erect. The peristaltic waves begin at the junction of the upper and middle thirds, shallow at first, but become deeper as they approach the pylorus, where they end in the closed pylorus. The waves recur at intervals of about twenty seconds, and are deeper on the greater curvature than on the lesser

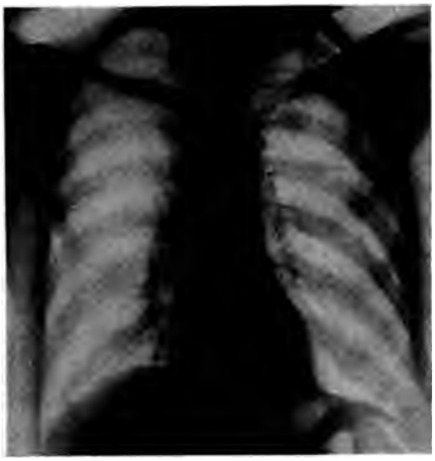


FIG. 157a.—Small particles of bismuth in the lower right bronchial tubes. There is communication with the œsophagus due to carcinoma.

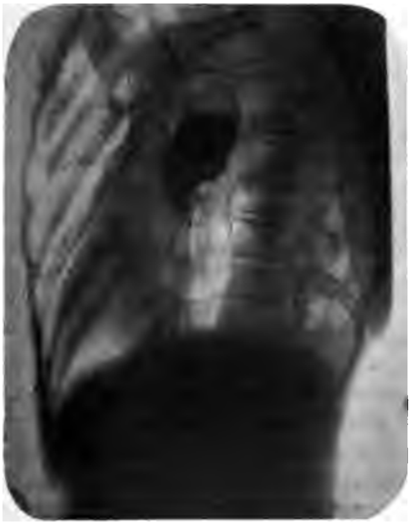


FIG. 157b.—Organic stricture of œsophagus. Note bismuth in dilated portion above the stricture, and irregularity of outline at the point of obstruction. (Oblique view.)

curvature until they approach the pylorus. The entire opaque meal leaves the stomach in from four to six hours.

In the athletic type of person with tense muscular abdominal walls the stomach is usually high, so that even the greater curvature is from one to three inches above the level of the umbilicus, and the long axis becomes more oblique with relation to the long axis of the vertebræ. The peristaltic waves are proportionately more vigorous and the emptying time less than the average normal stomach.

The opposite of this type of normal stomach is that in the individual whose abdominal walls are weak and relaxed. The greater curvature reaches three inches below the level of the umbilicus, while the pylorus is at or a little above the level of the umbilicus and more nearly in the median line. The peristaltic waves are lessened in vigor and the normal emptying time is about six hours.

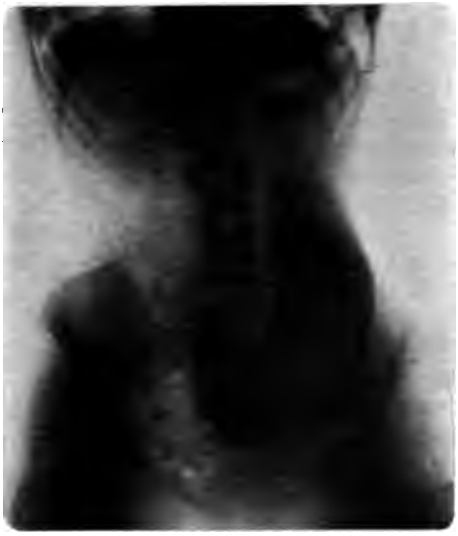


Fig. 158a.—True ptosis of stomach.

When the greater curvature reaches a still lower level it is due either to dilatation or extreme atony, and the pylorus remains at the normal level, or, if it is to be considered true ptosis, the pylorus is at a lower level near the median line, and the greater curvature may extend into the pelvis. The ptotic stomach is usually more or less dilated. The emptying time of the dilated, atonic, or ptotic stomach is from seven hours to indefinite.

Röntgen-ray diagnosis of organic gastro-intestinal lesions should be based very largely on direct evidence; that is, the defect should be demonstrable either on the fluoroscopic screen or on sensitive plates, and often both are essential. Functional disorders and those due to faulty position or musculature require fluoroscopic study combined with palpation. Much of the evidence is direct, but indirect evidence is also of great importance in this class of conditions.

Ulcer is probably the most common of the organic stomach lesions. In the early or acute stages when only the mucosa is involved the X-ray findings are largely inferential. Deep spasmodic contraction is seen on the greater curvature, making a sharp incisure in the shadow of the opaque meal. The ulcer is usually on the lesser curvature at the level of the incisure. Incisure may occur as a reflex phenomenon, but will relax after the administration of antispasmodic drugs. In ulcer cases the incisure may remain for hours or recur intermittently. There is delay in emptying the stomach due to spasmodic condition of the pylorus. There is no point of tenderness, but deep pressure may excite a violent contraction, and the incisure will appear in spite of antispasmodics.

When an ulcer has penetrated the mucous and muscular coats a definite and constant deformity occurs in the shadow of the filled stomach. The deformity has the appearance of being a small diverticulum. Spasmodic contractions may occur opposite the ulcer, but are not constantly associated. If the ulcer has healed in part there will be a more or less complete "hour-glass" contraction. The peritoneal coat may become perforated, and then small particles of the opaque meal can be seen isolated from the stomach shadow, and there is usually almost complete "hour-glass" formation due to cicatricial tissue contraction. There is usually a point of tenderness in deep ulcer. When ulcers occur near the pylorus it requires very careful observation to detect them in the early stages. The peristaltic waves on the lesser curvature are apt not to reach the pylorus, and there is retention. If the ulcer heals partly and contracts so as to produce mechanical obstruction, retention becomes more prominent and dilatation of the stomach follows. Vomiting is a rarely-associated symptom.

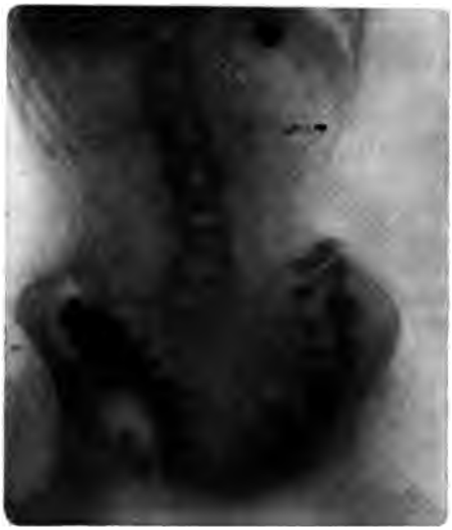


FIG. 158*b*.—Marked ptosis of colon. Same case as Fig. 158*a*

Carcinoma of the stomach can be diagnosed positively only when of sufficient extent to produce a permanent defect in the outline of the stomach shadow. Then the distinguishing feature is the irregular punched-out appearance of the defect. Pyloric spasm is not present and the stomach empties more rapidly than normal if the pylorus is not obstructed. Vomiting and loss of appetite and weight are common symptoms when the lesion is at the pylorus, and hæmatemesis appears when the mucosa is destroyed. It is surprising, however, to what extent the stomach may be involved before symptoms appear when the pylorus is not involved.

In any lesion of the stomach involving the peritoneal layer adhesions form and the stomach cannot be displaced at such points by palpation.

Reversed peristalsis is occasionally seen, and so far as we know is always associated with some organic lesion of the stomach.

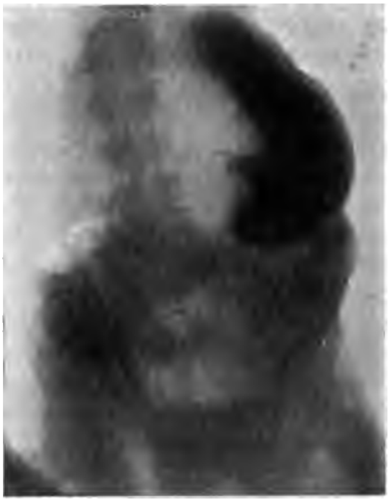


FIG. 159.—Carcinoma of pyloric end of stomach three hours after taking opaque meal. Almost complete obstruction of pylorus very little dilatation. Exposure made obliquely to isolate stomach shadow from spine.

Pyloric spasm and consequent retention occurs reflexly from disease in other organs, such as the gall-bladder, kidneys, or appendix. In such cases further X-ray study is necessary, particularly during deep palpation.

In duodenal ulcer the most accepted signs are: irregularity in outline of first portion (where the vast majority occur); marked gastric hyperperistalsis; rapid emptying of the stomach during the first hour in non-obstructive cases; retention of bismuth in the stomach after six hours in obstructive cases; fixation of duodenum in late cases or in gall-bladder disease with adhesions to the duodenum; and occasionally sensitive pressure point over the duodenum.

In the intestines obstructive lesions are determined by the presence of dilatation of the proximal portion and delay in passage of the bismuth. The colon is to be studied by its action on the ingested meal as well as by injection. All of the usual chemical tests for acidity, blood, etc., should be made prior to the Röntgen-ray examination, as they sometimes influence the interpretation of certain findings. A complete clinical history is also an essential adjunct.

In diseases of the gastro-intestinal tract that are not locally destructive e to new growth the Röntgen-ray examination is of value in confirming acts of relation to other viscera, and not infrequently showing lesions here which produce symptoms referable to the stomach. Kidney stone

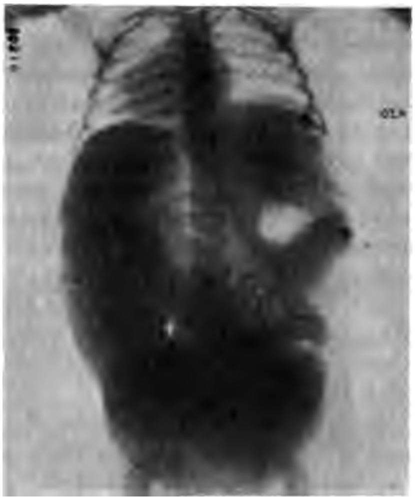


Fig. 160.—Congenital dilatation of the colon. (Viewed from back.)

en responsible for gastric symptoms which mask or exceed the renal toms.

Enlargement of the liver can be shown, gall-stones are demonstrable

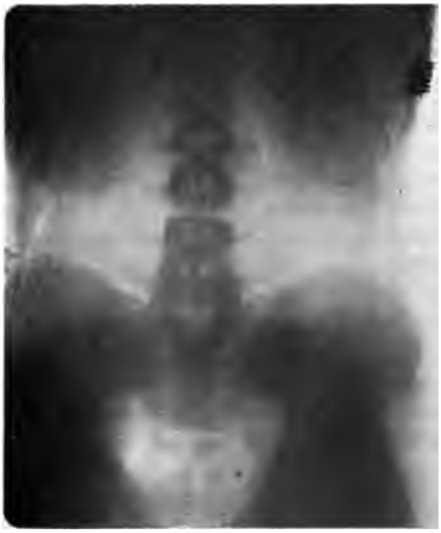


Fig. 161.—Stone in right kidney

out fifty per cent. of the cases, and in slender patients the gall-bladder ded so that it projects below the liver can occasionally be shown. The f the spleen can be determined, especially if the stomach and colon be ded with air or gas.

The urinary organs are studied by means of the röntgenographic method only. The röntgenogram should show enough of the outline of both kidneys to establish approximately the size and position in patients of one hundred and sixty-five pounds or less. This is due to the fact that the cortical portion of the kidney is more dense than surrounding structures, especially in the region of the lower poles. The pelves of the kidneys, the ureters, and the bladder do not cast distinguishable shadows. However, the outline of the entire urinary tract may be shown on the röntgenogram after the injection of a solution of colloidal silver, which is more opaque to the rays than the tissues. By this procedure one can differentiate between normal pelvis, hydronephrosis, tumors of the kidney, and abscess of the kidney if it drains into the pelvis. The shape of the kidney pelvis as shown on the plate is characteristic in each. Dilatations and kinks in the ureter can often be shown in the same manner. Such examination requires the assistance of an expert in ureteral catheterization. Renal, ureteral, and vesical calculi are readily demonstrable except those composed purely of uric acid. This variety is occasionally found in the bladder.

Diseases of the bones and joints which cause absorption, destruction, or proliferation of lime salts or cartilage lend themselves most aptly to röntgenographic study. In children the development of the skeletal structures is readily portrayed. The extent of the disease as well as its nature is usually shown, hence a prognosis as well as diagnosis may be more accurately made.

The value of the Röntgen-ray method of examination in excluding suspected conditions is scarcely less than in determining existing conditions.

The knowledge of the expert is as necessary to the explanation of skiagrams as to the making of them and it is always to be borne in mind that X-ray pictures are to be interpreted as shadows and not as lesions.

PART III.

OF SYMPTOMS AND SIGNS.

I.

GENERAL CONSIDERATIONS.

Symptoms and Signs.—The clinical phenomena of disease are divided into two general groups: (a) subjective, those obtained by inquiry, and (b) objective, those learned by observation. The former are known as SYMPTOMS, the latter as SIGNS. These may be general, as fever, debility, or emaciation, or local, as pain, dyspnœa, or dulness upon percussion. The dividing line between symptoms and signs is not well defined. Pain and nausea are symptoms of which nothing can be learned by observation alone; an endocardial murmur or pleural friction rub, signs concerning which the patient can give no personal account; while retching, cough, and palpitation partake at once of the nature of both signs and symptoms and may be referred to the one or other group of clinical phenomena according to the point of view from which they are regarded. SYMPTOMATOLOGY is that department of medical science which has for its object the consideration of the symptoms of disease; SEMEIOLOGY (σημεῖον, a sign), that which has for its object the consideration of the signs of disease. Just as symptoms and signs are not always to be closely distinguished, so the scope and subject matter of these sub-sciences of medicine largely overlap, and symptomatology and semeiology are frequently used interchangeably. Pure symptoms are limited in number as compared with signs, and, since they are wholly subjective and our knowledge of them is based upon the statements of the patient, who may, according to his temperament or for purposes of his own, either unintentionally or wilfully misrepresent them, they are of far less value in diagnosis than signs. Symptoms, which have their origin in the deranged sensations of the patient, stand in contrast to signs, which are dependent upon changes in organs or tissues. For this reason qualifying adjectives are sometimes employed, and we speak of rational symptoms and physical signs. It is customary, however, to apply the word symptom to many of the objective phenomena of disease.

SYNDROME is a term used to designate a set of concomitant symptoms, especially the concurrence of a group of symptoms not indicating a disease with well-determined anatomical lesions, as for example fever, seasickness, and astasia-abasia; a SYMPTOM-COMPLEX; a SYMPTOM-GROUP.

Diseases upon one basis of classification are divided into constitutional or general, namely, those in which the organism as a whole reacts to the pathogenic influence; and local or organic, in which the lesions involve primarily or chiefly a viscus or an anatomical tract. General symptoms are often the expression of a local disease and local symptoms the expression of a general disease; thus emaciation, pallor, and a profound cachexia attend the progress of carcinoma of the stomach, while tenderness in the right iliac fossa, diarrhœa, and tympany are symptoms of enteric fever.

II.

APPEARANCE; TEMPERAMENT AND DIATHESIS; FACIES; FORM AND NUTRITION.

APPEARANCE.

The general appearance of the patient when first seen forms the groundwork for the study of his present condition—the *status præsens.* Whatever knowledge may be subsequently obtained of the facts in the case, the general appearance constitutes the point of departure for the direct diagnosis. In the successful clinician the habit of observing and noting its various details with great rapidity is cultivated in a high degree. The facial expression, state of nutrition, movements and attitude, mode of speech, and mental condition are at once observed. An opinion is formed as to what manner of man the patient is. Information as to his social position, occupation, and habits may be learned from his dress: Is he neat or slovenly? Are his clothes buttoned awry? Is his collar loose to make room for swollen glands or a goitre? Do his trousers show the white stains of diabetic urine? Has he the tabetic or steppage gait or the festination of paralysis agitans? Has he the flushed face with dilated venules and the trembling tongue of the alcoholic, or the enlarged girth and the waistband lengthened with a loop of string, of hepatic cirrhosis with ascites? The hands tell a story of their own. We note at a glance that they are white and soft and the finger-nails are clean, as occurs mostly, but not always, with men of leisure and professional men; that they are large and callous, as in those who follow the sea; coarse, sunburnt, and freckled, as in the farmer; or that they bear the oil and grime of the mechanic who has hurried from his work. The trembling hand of alcoholism, the pill-rolling movements of paralysis agitans, the nodules and tophi of gout, the deformities and relaxed ligaments of arthritis deformans, and the spade-like hands of myxœdema tell their own tale.

The diagnosis may sometimes be made at a glance. The flushed face, hurried breathing, unilateral movement of the chest, and rusty sputum of pneumonia scarcely demand the additional data of chill, crepitant râle, and percussion dulness; nor the intense headache, opisthotonos, vomiting, herpes, and petechiæ the history of sudden onset or the epidemic prevalence of cerebrospinal fever; while the paroxysm of whooping-cough is in itself diagnostic. The diagnosis thus made cannot, however, be called intuitive. In truth there is no such thing as an intuitive diagnosis. Before a conclusion is reached, however brief the time, the clinician, usually without being aware of his mental processes, has been weighing and sifting the symptoms and assigning to each its proper value and importance. Such a diagnosis must in all cases be personally regarded as provisional and not announced, however tempting the circumstances, until sufficient facts for its full support have been obtained. There are pitfalls in the way of him who makes what in the language of the ward classes is called a "snap diagnosis." It is never complete; the pneumonia may be complicated with pericarditis. Such a diagnosis is sometimes altogether false; there are cases of enteric fever in neurotic individuals in early life that closely simulate the symptom-complex of meningitis—the so-called cerebrospinal form of enteric fever—and a child long convalescent from whooping-cough may under emotional excitement or when suffering from an attack of subacute laryngitis have paroxysms of cough with whooping.

TEMPERAMENT AND DIATHESIS.

Under the teaching of the French school great importance was at one time attached to temperament or diathesis as a condition of the hereditary constitution manifested in the general appearance of the patient. Less attention is paid to this subject now than formerly, but no very close observation is required to establish the fact that many individuals have in common peculiarities of physical and mental organization very different from those of others and that corresponding differences in general health and tendency to disease occur. Disregarding misleading refinements and combinations the following principal forms may be described:

The sanguine, sometimes called the Arthritic or Gouty Temperament.—The characteristics of this condition are well-developed bones and muscles, a fine skin, good hair, fair complexion, good nutrition, a general appearance of health and energy. The physiological processes are active, the digestion excellent, the bowels regular, the respiratory excursus large, the action of the heart regular, the pulse full and strong. In disposition persons of this temperament are cheerful and hopeful, hence the adjective "sanguine" often applied to them. Mentally they are active but of deliberate judgment and they do a large part of the world's work. They are especially liable to bronchitis and other catarrhal affections and to arteriosclerosis, and in advancing life prone to sclerotic changes in the valves ol the heart, aneurism, angina pectoris, or apoplexy.

The nervous diathesis shows itself commonly in slender or undersized individuals who are often poor and irregular eaters, not well nourished. Such persons are alert and active but often incapable of sustained effort. The physiological processes are not always well performed. They often possess high intellectual ability and are subject to intense emotions. They are predisposed to derangements of the digestive apparatus and to headache from slight causes. They take things to heart, as the phrase goes, and are liable to break down under stress of work and worry. Neurasthenia, hysteria, migraine, and other functional diseases of the nervous system and insanity are common.

The Bilious Diathesis.—The complexion is dark, the hair dark brown or black, often coarse and oily, the skin shows a tendency to local pigmentation which varies in intensity, especially about the eyes and brow.

The appetite is irregular, often poor, fats and sugars are digested with difficulty, and the bowels are sluggish. The nutrition is not good, though women of this temperament often grow stout in middle life. They are subject to attacks of that form of gastrohepatic catarrh popularly described as biliousness and are often at such times faintly jaundiced. Nausea and headache are common. Such persons often lack energy.

The Lymphatic Diathesis.—The muscles are soft and flabby, there is very often a slight excess of subcutaneous fat. The skin is pallid, the physiological processes are sluggishly performed. Mentally such persons are dull and unresponsive. They are subject to enlargements of the subcutaneous lymph-nodes and are liable to chlorosis and other forms of anæmia.

The Strumous Diathesis.—The word struma, meaning scrofula or glandular tuberculosis, though still applied in another sense to goitrous swellings, has lost its significance in medicine and has almost disappeared from the modern literature. The term strumous diathesis has, however, a very definite significance and is applied to a bodily constitution unfortunately too common. The bony framework departs widely from the normal type. The chest is small and flat, the shafts of the long bones slender, their epiphyses enlarged and thickened. The musculature is undeveloped and soft. The appearance is characteristic, the head is large, the cranial bosses prominent, the forehead broad and protuberant, the lips full, the nose short and broad, its alæ thickened, the lower jaw small, the teeth carious, the complexion commonly fair, the hair fine and the eyelashes long. The nutrition is poor and the general appearance of such individuals delicate and frail. The cervical lymphatics are often enlarged. Such persons are subject to tuberculosis of the glands, bones, and lungs and miliary tuberculosis, and usually die at an early age. They are frequently the offspring of tuberculous parents. Whether the constitutional peculiarities which go to make up the so-called strumous diathesis are to be ascribed to profound derangements of nutrition, transmitted from tuberculous parents, or to a latent tuberculosis acquired in the earliest period of life cannot always be determined.

Cachexia is a term used to describe the ravages of certain chronic wasting diseases, especially untreated malaria, the graver forms of syphilis, and carcinoma, particularly when it involves the digestive organs. The cachexiæ present a combination of profound anæmia, extreme emaciation and debility, and a diffuse, faint, muddy pigmentation of the skin.

Dyscrasia is a depraved state of the system, especially of the blood, due to constitutional disease. In the words of Bristowe it is a general deterioration of health and functional disturbance caused by the deflection of nutrition. We speak of a tuberculous, malarial, syphilitic, or cancerous dyscrasia.

FACIES.

The appearance of the face is often of diagnostic importance. It frequently indicates the subjective sensations and not rarely the psychical condition. To say that a patient's expression is that of suffering, acute pain, anxiety, overwhelming illness, or that it is excited, dull or, stupid, is intelligible without further comment. The face is an index of the physiological age of the patient. The gray hair, wrinkled brow, arcus senilis, and hanging folds of skin about the neck are very suggestive. They enable the clinician to compare the apparent age of the patient with his actual age as measured by years. The facies of fever patients is often characteristic. In the stage of excitement there is an intensification, in that of depression a blurring of expression, accompanied by a peculiar moist appearance of the eye, a feverish flush and often a very slight turgescence of the skin of which I shall speak more fully in a later paragraph. Equally characteristic is the facies of dyspnœa. Here also puffing or turgescence is present, sometimes marked, and there is cyanosis, and with these symptoms dilated nostrils, an open mouth, and hurried breathing. The flushed face and bright eyes that follow too much wine, and the bloated countenance with its blurred lines, dilated venules, thickened nose, acne, and trembling tongue of some forms of chronic alcoholism are sadly familiar. The characteristic change of the countenance seen in those about to die, especially in patients suffering from ileus, peritonitis, cholera, and similar diseases, is described as the FACIES HIPPOCRATICA. The changes are largely due to a diminution in the contents of the blood- and lymph-vessels and muscular relaxation. The skin falls back upon the bones, the lines of expression are more sharply defined than normally, the nose is sharp and pinched, the eyes sunken, the temples hollow. With the pallor there is some degree of cyanosis which gives the skin a leaden or faintly livid hue. The surface is cool and bathed with sweat.

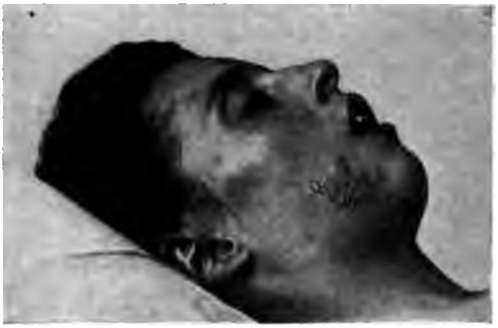


FIG. 162.—Cerebrospinal fever, fourth day of attack. Fever facies; patch of herpes; retraction of head.—Municipal Hospital, Royer.

The appearance of the face in the following conditions is suggestive if not always characteristic:

Enteric Fever.—In well-developed cases the face is dull, expressionless, pallid, with a faint, dusky flush over the cheek bones, often slightly drawn. The eyelids are half closed, the lips pallid and separated, in neglected cases sordes may be seen upon the teeth. Such also is the facies of patients profoundly septic. It occurs in the so-called "typhoid state" and is seen in puerperal septicæmia, malignant endocarditis, infection after surgical operations, and the like.

Acute Peritonitis.—The expression is one of intense suffering, the face is pale and drawn, sometimes the upper lip is contracted so as to show the teeth.

Pneumonia.—A circumscribed flush of one or both cheeks may be seen; it may be bright or dusky. When one cheek only is flushed it is usually that upon the side of the pulmonary lesion. With this appearance in grave cases are associated the facial changes peculiar to dyspnœa. A similar appearance accompanies the symptomatic fever of phthisis—hectic fever. The flushing is, however, brighter and not so distinctly circumscribed.

It is in strong contrast with the pallor of the brow and neck. The striking appearance of such patients is intensified by expanding nostrils, hurried breathing, bright moist eyes, and an intense, often eager, expression.

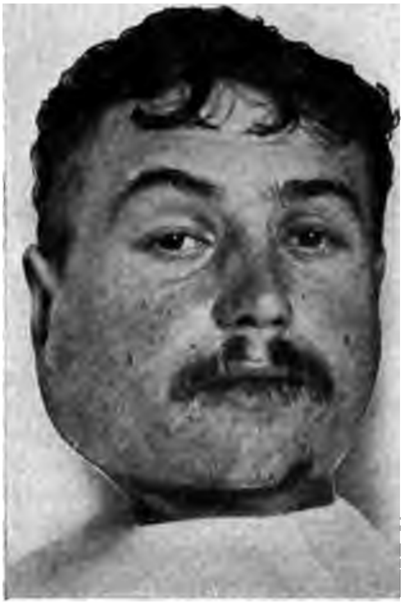


Fig. 163.—Parotid bubo complicating enteric fever. —Jefferson Hospital.

Tetanus.—The facial peculiarity is startling. Under the action of the toxin of the disease all the muscles of expression are thrown into tonic spasm more or less intense at the same time, whereas under ordinary circumstances the varying moods are expressed by the contraction now of one set of muscles, now of another. The lips are parted and the corners of the mouth drawn up as in laughter or grinning, while other parts of the face and especially the brow are contracted and thrown into folds as though in grief or anger — RISUS SARDONICUS of the older writers.

The Exanthemata.—The appearance of the face in the eruptive infectious diseases is usually diagnostic. The diffuse uniform rash, often in strong contrast with the white border around the mouth, and the slightly tumid skin of scarlet fever, the turgid skin, coarse measly rash with its crescentiform arrangement and coryza in measles, the pock in different stages of development with its umbilicated vesicles or hideous pustules and crusts and swollen and disfigured features in the variolous diseases, and the pellucid hemispherical vesicles or crusts without areolæ, scattered singly or in groups about the brow or mouth in varicella, are characteristic.

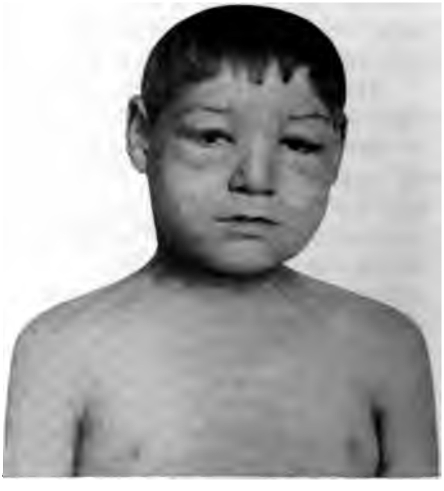


Fig. 164.—Œdema in acute nephritis.—Jefferson Hospital.

Mumps.—The deformity, alike when it involves one or both sides, is characteristic. The swelling is at first limited to the region of the parotid gland, behind the jaw and below the ear, but the surrounding œdema sometimes assumes remarkable proportions. The lobule of the ear stands out at right angles to the side of the head. When the swelling advances upon the cheeks, the corners of the mouth are slightly drawn up. The parotid bubo which occurs in some cases of enteric fever, pneumonia, septicæmia, and other grave infections superadds to the facies of those conditions a deformity somewhat like that of mumps. The overlying skin is usually of a dusky red color.

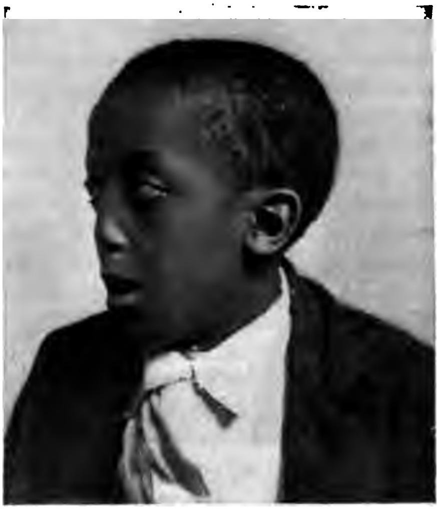


FIG. 165.—Facies in a case of adenoid vegetations of the nasopharynx.—Merrick.

Renal Disease.—The striking appearance of the patient in some forms of acute nephritis and very commonly in chronic parenchymatous nephritis is of diagnostic importance. It is characterized by intense waxy pallor, marked œdema of the eyelids, and general puffiness of the face by which the lines of expression are to some extent impaired.

Hepatic Disease.—In chronic diseases of the liver and especially in cirrhosis and gall-stone disease the facies is peculiar. The features are as a rule sharp, the face thin, conjunctivæ muddy, venules dilated, lips red, and skin slightly jaundiced or subicteroid—FACIES HEPATICA of the older writers.

Malaria.—The pallor, sallowness, and muddy conjunctivæ which are so commonly seen in intensely malarious districts are not without considerable value in the diagnosis of the cachexia of this disease.

Syphilis.—The face may show characteristic eruptions at different stages of the disease. The coppery flat papule or papulosquamous syphilide upon the temples and forehead—CORONA VENERIS—is common. The face of the patient under treatment may show not the eruption of the disease but the drug exanthem produced by the iodides. Babies suffering from inherited syphilis are usually pale, weazened, and wrinkled. They look curiously like miniature old men. They have snuffles and superficial excoriations about the angles of the nose and corners of the mouth.

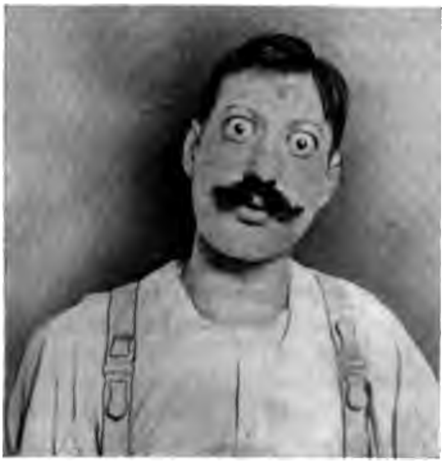


FIG. 166.—Exophthalmic goitre.—Jefferson Hospital.

Rickets.—The frontal and parietal eminences are exaggerated and the top of the skull flattened, so that the head assumes an appearance of squareness and is sometimes spoken of as box-shaped.

Hydrocephalus.—The head is characterized by its spherical shape, great size, and protruding eyeballs, the result of depression of the orbital plate of the frontal bone. The exophthalmus is sometimes so marked that the eyelids cannot be closed. The size of the head is often enormous, its diameter may reach 20 to 25 cm. in a child a few years old. The face on the contrary appears very small, its expression vacant and fatuous. The cranial bones are separated and exceedingly thin. The hair is scanty and the veins may be seen beneath the skin.

Hypertrophy of the Tonsils and of the Adenoid Tissue of the Pharnyx.—As a result of habitual mouth-breathing the expression of the countenance gradually undergoes characteristic changes, the face becomes apathetic and vacant, the nostrils are narrow, the lips thick, and there is projection of the upper jaw and lip.

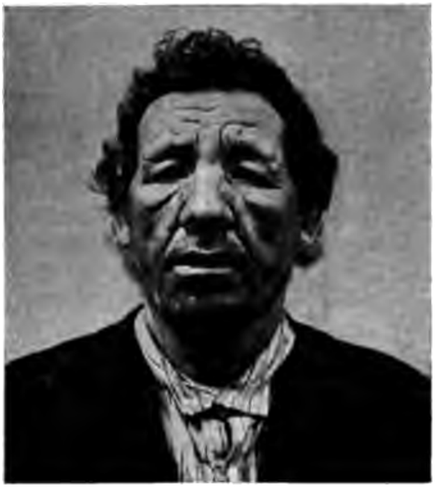


FIG. 167.—Leprosy.

Myxœdema.—The face is "moon-shaped," swollen and flattened, the nose broad, the mouth coarse and large, the lines of expression obliterated. The skin is yellow, waxy, dry, and scaly, the hair thin and scanty, the cheeks and nose flushed.

Cretinism.—The face is large, the lips thick, the tongue large and protruded, the mouth open and drooling, the nose flattened, the skin pallid and waxy, the expression idiotic.

Acromegaly.—The bony hypertrophy is especially manifested in the supra-orbital arches, the malar bones, and in the projecting lower jaw. The forehead is receding, the nose is increased in size, its alæ distended, the eyelids enlarged and thickened. The cartilages of the ear are also enlarged so that very often the ears stand out conspicuously from the head.

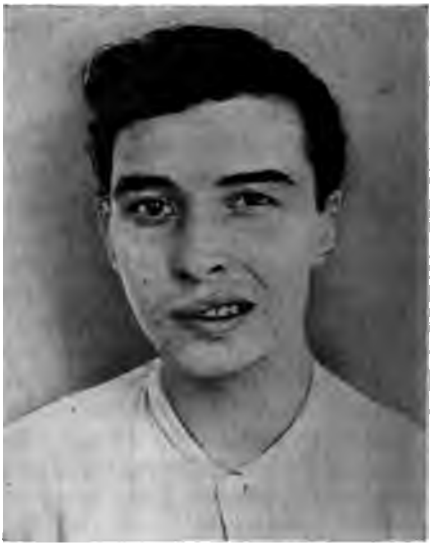


FIG. 168.—Facial paralysis following cerebrospinal fever.—Jefferson Hospital.

Exophthalmic Goitre.—The protrusion of the eyeballs, sometimes so marked that the patient can no longer close his eyes, produces a remarkable change in the expression. In its lighter grades the air is that of surprise, but when the exophthalmus is marked the patient has a frightened or astonished look which is intensified by the characteristic tremor. The enlargement of the thyroid body as well as the visible pulsation and venous distention add to the peculiar expression of the countenance in this disease.

Leprosy.—The development of leprous nodules upon the face and the thickening of the skin give rise to remarkable deformities. The chin, lips, nose, eyelids, and ears undergo peculiar and characteristic changes, in consequence of which the face assumes the appearance of a hideous expressionless mask. Baldness, loss of the eyebrows, lashes, and beard, and ulceration also occur. The disfigurement suggests the conventional face of the satyr or the lion and is often spoken of as leonine—FACIES LEONINA.

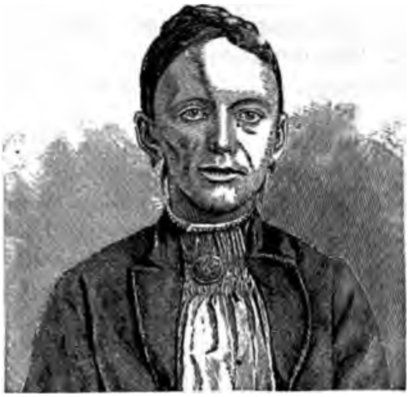


FIG. 169.—Hemiatrophia facialis.—After Hirt.

Nervous and Mental Disease.—In functional nervous diseases there are frequently changes in the countenance more easily recognized than described. The pallid, slightly drawn face of the neurasthenic with its habitual air of depression is familiar to the practitioner. These traits, somewhat intensified, are common in women broken down by frequent child-bearing and in those who suffer from disease of the pelvic organs—FACIES UTERINA. In hysteria the face usually remains free from the motor disturbances so common elsewhere, particularly in the lower extremities. There is neither spasm, paralysis, nor other constant characteristic save that it reflects, often intensely, the varying uncontrolled emotions of the patient. Equally without cause laughter succeeds tears or vivacity is followed by an air of sullen and dogged indifference. Central or peripheral disease of the nervous system may manifest itself in spasmodic twitching of the facial muscles. Mimetic spasm or convulsive tic consists of clonic contractions of the muscles supplied by the facial nerve. They are usually limited to the region about the eye or above the corner of the mouth. Sometimes they involve the greater part of one or both sides of the face. Similar contractions of the muscles of expression occur in children and are known as habit spasm. In peripheral facial palsy the affected side is smooth and motionless, the wrinkles of the forehead and the labionasal fold disappear, the corner of the mouth is lowered and frequently drools, and the mouth itself is slightly drawn toward the sound side. The eyelids

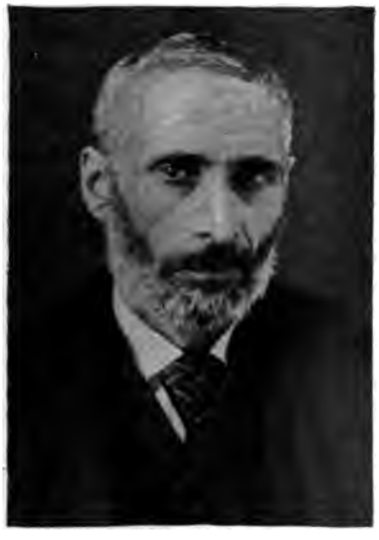


FIG. 170.—Paranoia, homicidal type.—Chase.

are motionless and can only be partly closed. The tears fall over the cheeks. The contrast with the opposite side is intensified upon efforts to smile or close the eye. When the paralysis is due to central causes the lower segment of the face is chiefly involved. In old cases, after contracture has taken place the mouth is drawn toward the affected side.

In organic disease tremor and paralysis are significant. Tremor of the lips and tongue occurs in chronic alcoholism. Fibrillary tremor is frequently associated with progressive palsy. In bulbar paralysis the lips are thin, compressed, and tremulous, the tongue is wasted and protruded with difficulty, and there is dribbling of saliva. In paralysis agitans the appearance of the countenance is very strikingly changed. The face has a curious stiff, expressionless immobility which has given rise to the name Parkinson's mask. There is often druling from the partially closed mouth and the lips and tongue frequently share in the general tremor. In general paresis local twitchings of the face, irregularity of the pupils, and slight tremulousness of the lips are suggestive. The rare disease facial hemiatrophy is a trophic neurosis affecting one side of the face, commonly the left. The soft tissues and bones are alike involved in the atrophic process, which is sharply limited at the mesial line. The eye is sunken and the corresponding half of the tongue and soft palate may be implicated.

The facies in disease of the mind is often characteristic. The depression of melancholia, the agitation and eagerness of acute mania, the alert slyness of chronic mania, the irregular contractions of the facial muscles in paresis, the fixed expression in paranoia with homicidal tendencies, the fatuous face of the imbecile, are well known to every student of psychiatry.

FORM AND NUTRITION OF THE BODY.

These are important factors in the problem of diagnosis. The normal of different individuals varies within wide limits. It is scarcely necessary to say that persons may be tall or short, stout or slight, fat or lean, without manifesting, even in wide ranges of difference in these respects, either the predisposition to or the symptoms of disease. Health consists in the ability of the individual organism to maintain its normal activities in the environment in which it happens to be placed. The somewhat pallid, hollow-chested and slender book-keeper often has good health and length of days while the clear-eyed, bright-skinned, deep-chested, well-developed athlete not infrequently breaks down in early middle life. Variations in these respects give rise to predisposition or constitute the indications of disease when they become excessive. We say that a man has a splendid or powerful physical development when the measurements of his body transcend the average, but the health of another who does not reach the average may be equally good. Not only anatomical structure but also physiological function are to be considered. Subtle hereditary tendencies, the value of which we cannot always estimate, and the shifting balance between the powers of the organism and the work which it is called upon to do engage our attention. To say that the physical organization is feeble, delicate, slender, robust, or muscular needs no further explanation. The condition of nutrition shows itself furthermore in the development and tonicity of the muscles and especially in their relation to the amount of subcutaneous fat—panniculus adiposus. On the one hand a moderate amount of subcutaneous fat is not incompatible with excellent health, an excess is alike inconvenient and dangerous, and obesity constitutes a positive disease. On the other hand a spare man may be equally healthy, while rapid loss of fat is a suspicious symptom and emaciation an alarming sign of disease. In estimating the value of these conditions the hereditary tendencies, the occupation, the age, and the sex of the patient must receive due consideration. In young infants the panniculus is well developed. In infancy it often dwindles, only to increase again as puberty approaches. At this period it not infrequently again becomes excessive. In advanced life the fat commonly diminishes and the aged as a rule are spare. The panniculus is usually greater in women than in men and very often increases after the menopause. In most chronic diseases the nutrition is impaired because either sufficient nourishment is not taken on account of loss of appetite, or that which is taken is not assimilated. A high degree of emaciation attends diseases of the digestive organs and chronic febrile diseases, for example, carcinoma, especially carcinoma of the œsophagus or pylorus, enterocolitis with excessive diarrhœa, some forms of diabetes mellitus, phthisis, and enteric fever with repeated relapses. Wasting of fat is accompanied by wasting of muscle.

Weight.—The weight of the body and the amount of subcutaneous fat may be approximately estimated by inspection, but this method is uncertain and practically valueless in determining the progress of gain or loss. Accurate data can only be obtained by the use of scales at regular intervals and the preservation of the records for comparison. Small platform scales provided with a device for ascertaining the height of the patient are indispensable in the consulting room of the medical clinician engaged in the treatment of chronic cases. The automatic weighing machines found in public places in the cities are unreliable. Allowance must be made for the clothing and its variations in the different seasons, and when practicable the weight should be obtained shortly after the voidance of urine and an action of the bowels, and before a meal. Errors of two or three pounds may thus be eliminated, though for practical purposes in the long run slight fluctuations in the weight may be disregarded in the course of chronic disease. Many healthy individuals show an annual oscillation of several pounds in net weight, allowance being made for clothing, the minimum being reached in the spring or early summer, the maximum in the autumn or beginning of the winter. The body weight should be taken according to the requirements of individual cases at regular intervals of a week or longer. Daily observations are unnecessary.

The relation of the average body weight to the age and height of healthy individuals is set forth in the following tables:

Average Weight of Healthy Adult Males.—Hutchinson.

4 ft. 6 in. to 5 ft. 0 in.	92.26 lbs.
5 ft. 0 in. to 5 ft. 1 in.	115.52 lbs.
5 ft. 2 in. to 5 ft. 3 in.	127.86 lbs.
5 ft. 4 in. to 5 ft. 5 in.	139.17 lbs.
5 ft. 6 in. to 5 ft. 7 in.	144.29 lbs.
5 ft. 8 in. to 5 ft. 9 in.	157.76 lbs.
5 ft. 10 in. to 5 ft. 11 in.	170.86 lbs.
5 ft. 11 in. to 6 ft. 0 in.	177.25 lbs.

Infants, whether nursed or artificially fed, should be weighed at regular intervals of some days or a week. Important information is thus obtained not only as to the appropriateness of the food in kind and quantity but also as to the presence of assimilative disorders. The average normal weight of the newborn is, according to Uffelmann, in girls 3000 grammes, in boys 3500. During the first three or four days of life there is a decrease of from 220 to 300 grammes. After this there is in healthy children a progressive increase.

AVERAGE DAILY INCREASE IN WEIGHT DURING THE FIRST YEAR OF LIFE.—**GERHARDT.**

1st month	**25 grammes.**
2nd month	**23 grammes.**
3rd month	**22 grammes.**
4th month	**20 grammes.**
5th month	**18 grammes.**
6th month	**17 grammes.**
7th month	**15 grammes.**
8th month	**13 grammes.**
9th month	**12 grammes.**
10th month	**10 grammes.**
11th month	**8 grammes.**
12th month	**6 grammes.**

The weight index is the ratio of the weight of a given infant to the weight of the average normal infant of the same age.

Useful figures to remember are that the initial weight is doubled at 5 months and trebled at 15 months; also that the weight at one year is doubled at 7 years and that this weight is again doubled at 14 years (Rotch).

In infants and young children misleading inferences may be drawn from a consideration of the weight alone. There are some who are fat and flabby and not healthy. Such children are pallid, they lose and gain weight rapidly and have but little resisting power to disease. Then there are many who are bright and rosy, whose flesh is firm and solid, whose nutrition is good, who gain in weight normally and are not liable to the wasting diseases.

In the following table the comparative average weight of the sexes is shown. It will be observed that from birth until the sixth year the average weight in the two sexes is nearly the same. From this period for some years the weight of the female is considerably less than that of the male. About the age of puberty the difference becomes less marked, though the weight of the female is decidedly below that of the male.

AVERAGE NORMAL WEIGHT IN THE TWO SEXES AT DIFFERENT PERIODS OF LIFE—**QUETELET.**

	Males.		**Females.**	
New-born	3.1 kgs.	6.82 lbs.	**3.0 kgs.**	**6.60 lbs.**
1st year	9.6 kgs.	19.80 lbs.	**8.6 kgs.**	**18.92 lbs.**
2nd year	11.0 kgs.	24.20 lbs.	**11.0 kgs.**	**24.20 lbs.**
3rd year	12.5 kgs.	27.50 lbs.	**12.4 kgs.**	**27.28 lbs.**
4th year	14.0 kgs.	30.80 lbs.	**13.9 kgs.**	**30.58 lbs.**
5th year	15.4 kgs.	33.88 lbs.	**15.3 kgs.**	**33.66 lbs.**
6th year	17.8 kgs.	39.16 lbs.	**16.7 kgs.**	**36.74 lbs.**
7th year	19.7 kgs.	43.34 lbs.	**17.8 kgs.**	**39.16 lbs.**
8th year	21.6 kgs.	47.52 lbs.	**19.0 kgs.**	**41.80 lbs.**
9th year	23.5 kgs.	51.70 lbs.	**21.0 kgs.**	**46.20 lbs.**
10th year	25.2 kgs.	55.44 lbs.	**23.1 kgs.**	**50.82 lbs.**
11th year	27.0 kgs.	59.40 lbs.	**25.5 kgs.**	**56.10 lbs.**
13th year	33.1 kgs.	72.82 lbs.	**32.5 kgs.**	**71.50 lbs.**

	Males.		Females.	
15th year	41.2 kgs.	90.64 lbs.	40.0 kgs.	88.00 lbs.
17th year	49.7 kgs.	109.34 lbs.	46.8 kgs.	102.96 lbs.
19th year	57.6 kgs.	126.72 lbs.	52.1 kgs.	114.62 lbs.
20th year	59.5 kgs.	130.90 lbs.	53.2 kgs.	117.04 lbs.
25th year	66.2 kgs.	145.64 lbs.	54.8 kgs.	120.56 lbs.
30th year	66.1 kgs.	145.42 lbs.	55.3 kgs.	121.66 lbs.
60th year	61.9 kgs.	136.18 lbs.	54.3 kgs.	119.46 lbs.
70th year	59.5 kgs.	130.90 lbs.	51.5 kgs.	113.30 lbs.

In cases of tardy or interrupted convalescence from an acute disease systematic observations of the weight of the patient at intervals of a week are of great use. A sudden arrest or decrease in weight may mark the development of a tuberculous process. Loss of weight is of great importance in the diagnosis of early phthisis. An arrest of the loss, still more a gain in body weight, must in most cases of this disease be regarded as favorable. There are, however, exceptional cases in which after a considerable steady gain in weight the tuberculous process suddenly makes grave or even fatal progress.

The weight is not in all cases merely an indication of the general nutrition and amount of fat. It is sometimes made up in considerable part of dropsical effusions, as in advanced disease of the mitral valve with rupture of compensation, of accumulations in the serous sacs, as in massive serofibrinous pleurisy or the ascites of cirrhosis of the liver, of the contents of cysts, as in enormous monolocular disease of the ovary, or of new growths of large size, as in the splenic tumor in leukæmia or sarcoma of the kidney in young children. In a dropsical patient the successful use of salines or diuretics may be followed coincidently with the subsidence of the anasarca by a reduction in weight amounting to many pounds in a few days. The tendency to accumulate excessive fat at middle life, especially in women after the menopause, must be regarded as pathological, and obesity, as has been said, constitutes a positive disease. The gain in weight occurs at the time of beginning decrease of muscular power, at the period of physiological involution. The individual must carry about a growing mass of inert fat with lessening ability on the part of the skeletal muscles to bear it and of the heart to carry on the circulation, and the disproportion between the burden and the ability to bear it increases with advancing years. Visceral fat accumulations occur, also fatty changes in the myocardium and vessels. Obese persons in early middle life bear the acute infections and especially enteric fever badly. Very often the fat accumulations are largely local, as in the mammæ, abdomen, and hips in women, or in the abdomen, abdominal walls, and omentum in men of sedentary life and given to the pleasures of the table.

III.

BONES; JOINTS; MUSCULATURE; POSTURE, ATTITUDE, AND GAIT; POSTURE AND MOVEMENTS OF INFANTS.

BONES.

The skeleton determines the stature and frame of the individual. As has been already pointed out the normal limits of variation in the measurements of the bony framework are very wide. Excess in either direction, as in gigantism or dwarfism, is pathological and has been ascribed to derangements of the functions of the pituitary body.

Average Height in Males and Females at Different Periods of Life.—Quetelet.

	Males.		Females.	
New-born	50.0 cm.	20.00 in.	49.4 cm.	19.76 in.
1st year	69.8 cm.	27.92 in.	69.0 cm.	27.60 in.
2nd year	79.1 cm.	31.64 in.	78.1 cm.	31.24 in.
3rd year	86.4 cm.	34.56 in.	85.4 cm.	34.16 in.
4th year	92.7 cm.	37.08 in.	91.5 cm.	36.60 in.
5th year	98.7 cm.	39.48 in.	97.4 cm.	38.96 in.
6th year	104.6 cm.	41.84 in.	103.1 cm.	41.24 in.
7th year	110.4 cm.	44.16 in.	108.7 cm.	43.48 in.
8th year	116.2 cm.	46.48 in.	114.2 cm.	45.68 in.
9th year	121.8 cm.	48.72 in.	119.6 cm.	47.84 in.
10th year	127.3 cm.	50.92 in.	124.9 cm.	49.96 in.
15th year	151.3 cm.	60.52 in.	148.8 cm.	59.52 in.
20th year	167.0 cm.	66.80 in.	157.8 cm.	63.12 in.
25th year	168.2 cm.	67.28 in.	157.4 cm.	62.96 in.
30th year	168.6 cm.	67.44 in.	158.0 cm.	63.20 in.
40th year	168.6 cm.	67.44 in.	158.0 cm.	63.20 in.
60th year	167.6 cm.	67.04 in.	157.1 cm.	62.84 in.
70th year	166.0 cm.	66.40 in.	155.6 cm.	62.24 in.

There is a constant relationship in healthy persons between the muscular development and the size and strength of the bones. In puny individuals with small and flabby muscles the skeleton is usually more or less under-developed. In this nutritional relationship between the muscles and the bones the muscles constitute the controlling factor. In a similar manner the bony walls of the cranium and thorax undergo changes corresponding to changes in the viscera which they contain.

Thorax.—In bed-ridden individuals and those suffering from wasting diseases the involution of the lungs from diminished functional activity or their diminution in size from pathological changes causes alterations in the shape and contour of the thorax, which tends to assume permanently the EXPIRATORY FORM; while an active life in the open air by increasing the volume of the lungs modifies the chest, which under these circumstances tends to assume permanently the INSPIRATORY FORM. Analogous changes in the chest result from lesions which increase the volume of the thoracic viscera, as in pulmonary emphysema and great cardiac enlargement. The point for the student to bear in mind is that many general and local changes in the form of the chest are primarily due to visceral disease and not to disease of the bones. Some of these are more marked when the visceral disease takes place early in life, as in precordial prominence and the development of Harrison's furrows, others late in life, as in fibroid phthisis and emphysema. There are, however, exceptions to this general statement, an example of which is to be found in the changes of the shape of the chest which result from disease of the spine, as kyphosis.

Cranium.—The skull may be abnormal in size and shape either as the result of arrest of development of the brain with or without malformation, or as the result of pathological increase in the size of the brain. The short diameters and peculiar shape of the head of the microcephalic idiot and the globe-like cranium of chronic hydrocephalus developing at birth or in early infancy with its wide sutures, open fontanelles, and card-like thinness of the bones, are examples of the influence exerted by changes in the soft parts upon the bony walls containing them.

Skeletal Changes.—The bones themselves undergo pathological changes. These changes may be general or local. In ACROMEGALY there is hypertrophy of the bones of the hands, feet, and face, especially the inferior maxilla. The clavicles, sternum, and in some instances the long bones of the extremities also participate in the over-growth. In OSTEITIS DEFORMANS or PAGET'S DISEASE there is thickening of the bones of the skull and changes in those of the face, the outline of which becomes triangular with the apex at the chin; the long bones are involved and become deformed. In RICKETS, a disease of childhood, the head is large and square, the forehead prominent, the anterior fontanelle open, the epiphyses of the long bones are enlarged, nodules develop at the junction of the ribs with their cartilages. Changes in the shape of the chest and protrusion of the sternum cause the deformity known as CHICKEN OR PIGEON BREAST. The spine is curved, the clavicle bent, the pelvis deformed, and the long bones of the lower extremities show deformity. Rachitic children are often bow-legged; those who reach maturity are under-sized. OSTEOMALACIA is characterized by resorption of the lime salts. The bending of the softened bones under the action of gravity and muscular tension gives rise to extraordinary deformities. These affect the spine, thorax, pelvis, and long bones. In some instances the superficial bones crepitate upon pressure and can be indented by the finger. They are readily fractured and this accident may follow a trifling fall or blow or, in the case of the femur or humerus, result from the muscular force exerted in turning in bed. PULMONARY OSTEO-ARTHROPATHY—OSTÉO-ARTHROPATHIE HYPERTROPHIANTE PNEUMONIQUE OF MARIE—a condition encountered in certain chronic diseases of the lungs and pleura, is characterized by bulbous enlargement of the terminal phalanges of the fingers and toes and of the distal epiphyses of the bones of the upper and lower extremities. The finger-nails are hypertrophied and strongly incurved. The bones of the head and face are not affected.

JOINTS.

There are affections of the joints which lie on the border line between surgery and medicine. To the former belong traumatic and operative conditions; to the latter lesions arising in consequence of various constitutional affections. Commonly the question of diagnosis first rests with the medical clinician. Those joint affections which properly come within the scope of internal medicine may be comprehensively described as the MEDICAL ARTHROPATHIES. The large and small joints may be affected. The chief symptoms are pain, especially upon movement, impairment of function, and the signs of inflammation or disorganization, namely, changes in color, size, and shape. The requisites to the proper examination of a diseased joint are a knowledge of the local anatomy and pathology and of the constitutional diseases in which joint affections occur.

Pain. - Pain is an important symptom. It may be spontaneous. More commonly it is caused by movement. Pain upon pressure occurs in acute forms of arthritis and is often intense. Pain is commonly referred to the affected joint, sometimes to a distant part, as the pain in the knee in hip-joint disease. In consequence of the freer movement permitted by muscular relaxation during sleep the pain is worse at night. There may be insomnia, or sleep may from time to time be broken by sudden agonizing pain. This is especially the case in tuberculous joint-disease. The patient very often awakes with a sharp cry of pain. The pain in myalgia and various forms of neuritis is frequently attributed to diseases of the joint; upon movement the pain is found not to involve the joint, but other structures, and the joint is neither tender nor swollen. In chronic joint affections movement is sometimes attended by a sensation of grating or crepitus, or there may be a catching sensation attended with crackling sometimes audible at a distance.

Color. The color of the joint in acute inflammation is pinkish or red; when intense it is cyanotic or dusky. When there is marked periarticular œdema the overlying skin is pale.

Changes in Size.—In acute inflammation the joints are usually enlarged. This enlargement is attended with alteration in the contour. These changes are due to effusion, which may be articular or periarticular. The former may be serous, purulent, or hemorrhagic. The latter may be œdematous or exudative. These conditions are often combined. In chronic arthritis there is infiltration of the tissues entering into the formation of the joint. Enlargement due to effusion within the joint may be recognized by palpation, especially in large joints. In the knee the patella floats. Rounded local swellings fluctuating upon palpation may indicate the distention of the synovial sac. Enlargement may be due to changes in the ends of the bones.

Irregular diminution in the size may occur in chronic disease of the joints, as rheumatoid arthritis or other diseases characterized by resorption or retrogressive processes. Not only the tissues of the joint but the periarticular structures undergo atrophy and subluxations occur, or there may be diminution in the soft parts with thickening of the bones. All these processes are associated with changes in contour.

The posture is of importance. In forms of acute arthritis, flexion or semi-flexion and immobility are present - the attitude of least tension and therefore of least pain. The mobility of the joint is determined by passive movement. Fixation may be voluntary because it relieves pain. It may result from muscular spasm or large effusion. Sudden locking of a joint, especially the knee, may be due to floating cartilages or "joint mice" becoming arrested between the anterior surface of the bones and the capsular ligament. In late cases the immobility is due to ankylosis, which may be adhesive, fibrous, or bony. Movement may be limited or prevented by the development of osteophytes in the region of the joints. Crepitus may be detected upon palpation.

Any of the joints may be involved in general diseases. The knee, hip, and shoulder are especially important, because of the frequency with which they are implicated, the disabling results, and the tendency to disorganization and ankylosis.

The medical arthropathies are inflammatory or infective, degenerative, and neuropathic.

Primary Arthritis.—Simple acute synovitis with effusion is very common especially in adolescents and young adults. It most frequently involves the knee-joint. Traumatism and sudden chilling are causes. Some of the cases appear to be monarticular rheumatism with trifling fever. There is marked tendency to recurrence and chronicity.

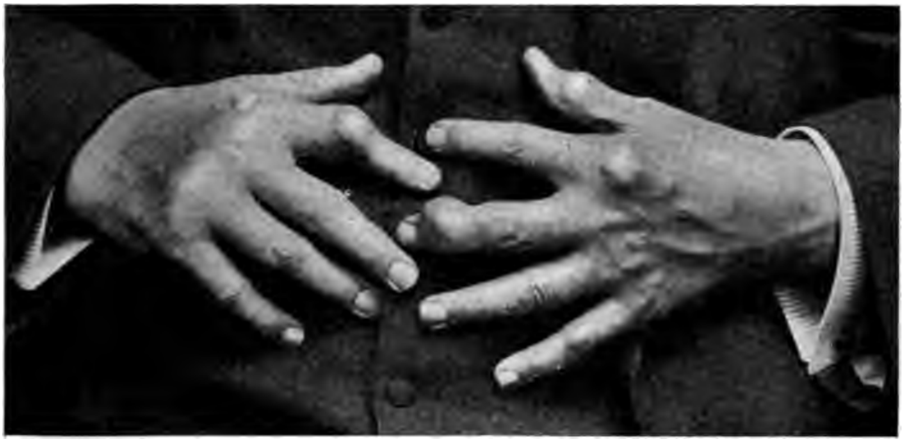


Fig. 171.—Tophaceous deposits in gout.

Rheumatic Fever.—The affected joints are swollen, hot, usually slightly reddened, and painful upon motion. The amount of swelling is variable. The intra-articular effusion is usually slight or moderate, the periarticular œdema being commonly marked. When the wrists and ankles are implicated there is marked swelling of the hands and feet. The joint effusion of rheumatic fever is fugacious. The tendency to rapidly subside in one joint and develop in others is characteristic. The process is rarely limited to a single joint. Any joints may be affected, but the knees, ankles, and wrists are especially liable to the rheumatic inflammation.

Chronic Rheumatism.—This term is applied to a chronic condition in which the joints are painful, stiff, moderately swollen, and but slightly deformed. It is common in individuals who have been much exposed to the vicissitudes of the weather or have lived in damp places. Its etiological affinity to rheumatic fever may well be questioned. Some of the cases described under this term are undoubtedly subacute forms of rheumatoid

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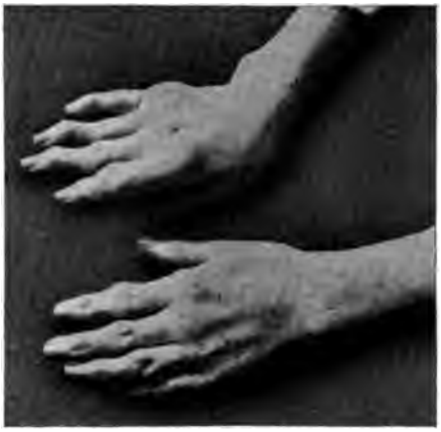
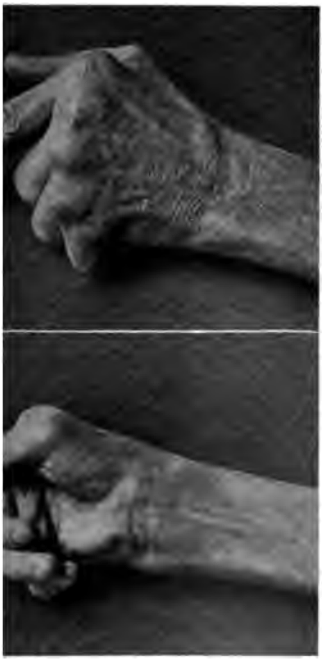


Fig. 173.—Arthritis deformans.—Jefferson Hospital.



thritis deformans with extreme ulnar
rmity.—Jefferson Hospital.

Tuberculous Arthritis.—Tuberculous joint disease is common. It is often secondary to tuberculosis of the bones. It was formerly known as white swelling—*tumor albus*. The process is comparatively subacute but tends to permanent disorganization. Tuberculous joints are usually swollen. In the course of the disease chronic inflammatory infiltration takes place into the capsule, ligaments, and periarticular connective tissue. Caseation and softening result in abscess formation and burrowing along the lines of least resistance. Tortuous fistulous passages occur. The hip, elbow, knee, and wrist are frequently affected. There may be evidences of tuberculosis in the lungs or elsewhere. More commonly the process is limited to the affected joint and adjacent structures.

Syphilis.—The acute joint affection of new-born infants sometimes
ıs rheumatic is mostly syphilitic. It is a form of primary exuda-
itis with fibrous thickening of the capsule. Gummatous inflam-
the neighboring tissues may involve a joint by extension. In

acquired syphilis subacute synovitis occasionally occurs during the period of eruption. The sternoclavicular joint shows a peculiar liability. In late syphilis, forms of chronic arthritis, the result of gummatous infiltration of the tissues forming the joint, occur.

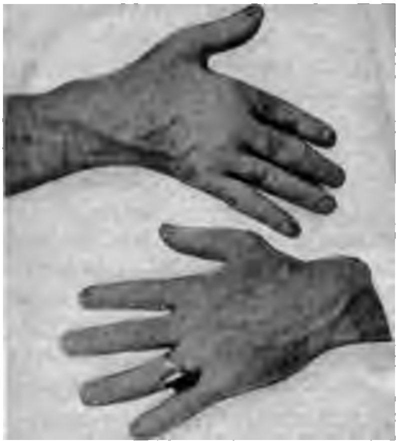


FIG. 175. Gonorrhœal arthritis.—Pennsylvania Hospital.

Actinomycosis.—The joints are sometimes involved by metastasis. In other cases they are invaded by extension, as when the disease reaches the articulations of the cervical vertebræ or when prevertebral actinomycosis attacks the spine or the disease extends from the thorax to the sternoclavicular joints or from the abdomen to the hip-joints.

Neuropathic Joint Affections.—Hysteria especially may simulate disease of the joints. The impairment of function is caused by contracture of muscles. Pain is more diffuse and spontaneous than in actual arthritis. The patient avoids movement and does not coöperate in the examination. The signs of effusion, inflammation, and erosion are lacking. These are the cases in which spontaneous cures sometimes occur under profound mental suggestion. In some instances, from prolonged disuse, infiltration, and thickening of the periarticular tissues, false ankylosis and atrophy of the associated muscles occur. It is important to bear in mind that hysterical symptoms may be superadded to those of actual joint disease. The differential diagnosis between traumatic joint disease and a hysterical joint in traumatic hysteria is occasionally attended with difficulty. Vasomotor changes with swelling, tension, and redness sometimes occur and the surface temperature may be two or three degrees higher than that in the axilla. These symptoms are not associated with fever or the evidences of constitutional disturbance and are commonly transitory and recurrent.

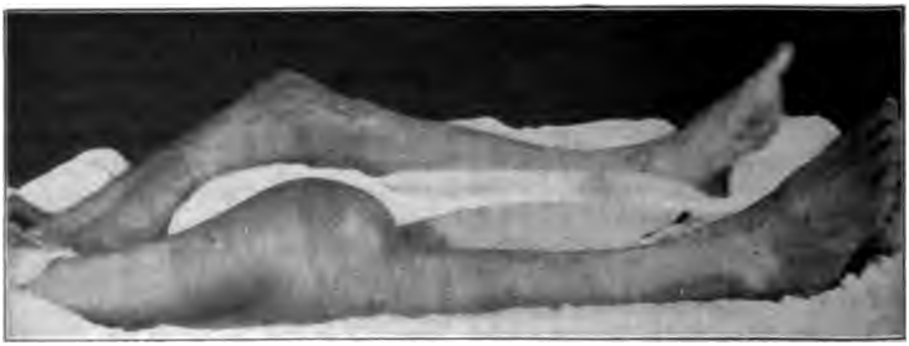


FIG. 176.—Ataxic knee-joint.—Young.

More important are the changes that take place in connection with certain diseases of the nervous system—CHARCOT'S JOINTS, TABETIC JOINTS—particularly locomotor ataxia, syringomyelia, less frequently in anterior poliomyelitis and other diseases of the spinal cord. The joint affection in tabes is much more common in the joints of the lower extremities, especially the knee, less frequent in the hip and ankle; that of syringomyelia is by far more common in the upper extremities. The derangements are primarily trophoneurotic. The process is frequently monarticular. The pathological and clinical changes correspond to those of the milder and graver forms of rheumatoid arthritis. In the more severe forms they differ in suddenness of onset, intra-articular effusion, and a rapid, disintegrating course without pain. Subluxations and luxations take place. When the tarsal articulations are implicated flat-foot occurs with characteristic deformities—the tabetic foot.

MUSCULATURE.

Diagnostic criteria of importance are obtained by an examination of the condition of the muscles. Wide variations in the bulk and tonicity of the general musculature is encountered within the limits of health. These variations depend largely upon the hereditary constitution, occupation, and bodily activities of the individual and are not of diagnostic significance. Trophic derangements result in hypertrophy and atrophy.

Hypertrophy.—True hypertrophy, that is to say, increased volume with increase of power, is exceedingly rare. It occurs in Thomsen's disease. CONGENITAL HYPERTROPHIA MUSCULORUM VERA has been described. Pathological increase in the muscles is almost always a pseudohypertrophy. The abnormal volume is not due to an increase in the contractile tissue but to a proliferation of the connective tissue and fat. This muscular dystrophy occurs in its most pronounced form in the so-called pseudohypertrophic muscular paralysis of childhood, and very rarely in some of the affected muscles in certain cases of chronic progressive muscular atrophy.

Atrophy.—Atrophy of the muscles may be simple or inactivity atrophy—the atrophy of disuse. The affected muscles are diminished in size, soft, and flaccid; there is loss of the contractile substance; the interstitial connective tissue is not increased. This form of atrophy occurs in certain forms of paralysis, and supervenes upon mechanical fixation of a limb or the prolonged immobility resulting from joint pain or ankylosis. Complete loss of movement usually gives rise to a high grade of simple atrophy. Atrophy from disuse rarely attains the degree often seen in the degenerative atrophies. In simple atrophy there is general diminution in the volume of the affected limb, while in the degenerative atrophies single muscles or groups of muscles are exclusively or chiefly involved. The electrical reactions in simple atrophy are quantitatively and not qualitatively changed. The nutritional muscular atrophy which occurs in starvation, in the course of acute infections, and in the chronic wasting diseases must be regarded as a diffuse form of simple atrophy.

Myoidema.—This phenomenon consists in a sudden contraction of muscular fibres when smartly tapped with the finger or hammer, with transitory humping at the point of impact. It is manifested in muscles that are undergoing rapid wasting, especially in phthisis, and is as a rule best developed in the muscles of the chest.

Degenerative Atrophy.—The degenerative muscular atrophies, which are characterized not only by loss of contractile substance but also by an overgrowth of the interstitial connective tissue, may be referred to two groups: (a) the progressive muscular atrophies, and (b) the atrophic paralyses.

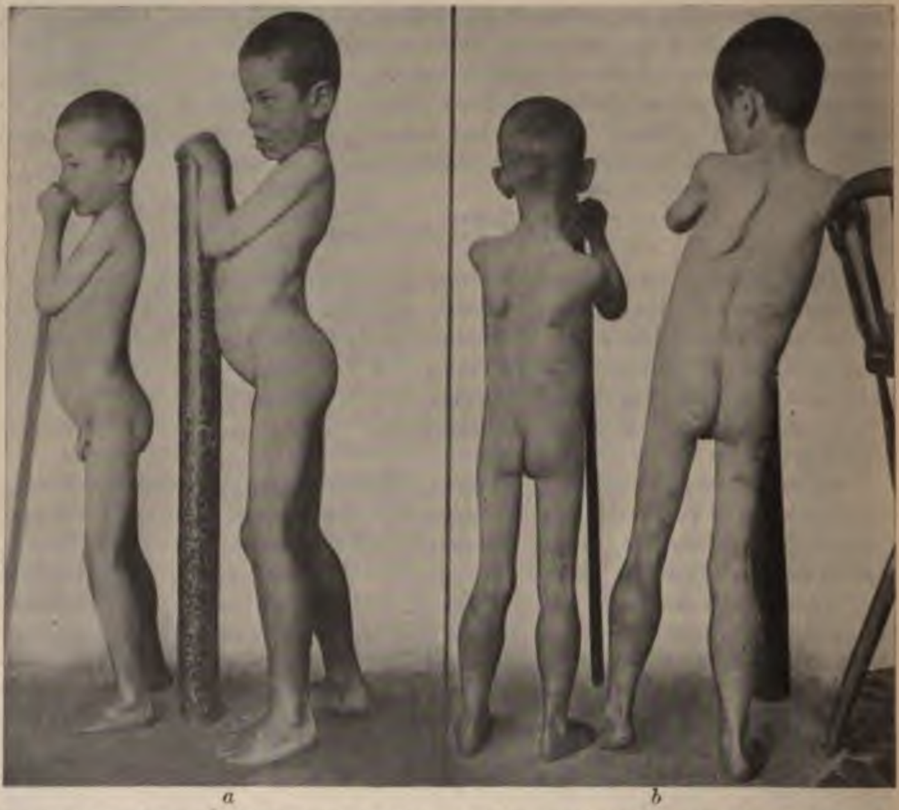


FIG. 177.—Pseudohypertrophic muscular paralysis. Brothers, eight and ten years old. *a*, showing the lordosis; *b*, showing atrophy of back and enlarged calves.—Rotch.

The progressive muscular atrophies may be divided into myopathic, peripheral, and central or nuclear according to the seat of the essential pathological process, which may primarily involve the muscles, or result from an acute or chronic peripheral neuritis, or from degenerative changes in the ganglion cells of the anterior horns of the cord, or the motor nuclei of the brain. There is progressive atrophy of individual muscles and muscle groups; diffuse atrophy of an entire limb occurs only in advanced stages; the strength of the muscles is diminished in proportion to the diminution of their volume. In this respect the progressive muscular atrophies are in contrast with the secondary degenerative atrophies which follow the atrophic paralyses. In the latter the paralysis comes first, the atrophy afterwards. The discrimination between myopathic, neural, and nuclear muscular atrophies rests upon the fact that in the different forms particular groups of muscles are affected. In the myopathic forms of degenerative atrophy—the muscular dystrophies—the following principal types occur: 1. Pseudohypertrophic muscular atrophy of childhood—the so-called pseudohypertrophic muscular paralysis. 2. The juvenile type of Erb—dystrophia musculorum progressiva; the atrophy begins in the shoulder girdle and is not preceded by pseudohypertrophy. 3. The juvenile type of Leyden-Möbius; the atrophy begins in the lower extremities. This form is closely allied to the progressive pseudohypertrophy of childhood. 4. The infantile type of Duchenne—the facio-scapulo-humeral type of Landouzy-Dejerine. This form begins in the face. The loss of power in the muscles of expression gives rise to the characteristic FACIES MYOPATHICA. The eyes can no longer be completely closed, the cheeks are sunken, the lips thickened and everted, speech is impaired, and the ordinary changes in the countenance in laughter and crying are not seen. The myopathic atrophies are commonly hereditary and almost always show themselves in early life. Neural atrophy begins commonly in the under extremities in the distribution of the peroneal nerve—the peroneal type of Charcot and Marie—and may lead to the development of club-foot, usually pes equinus or pes equinovarus. It differs from other forms of

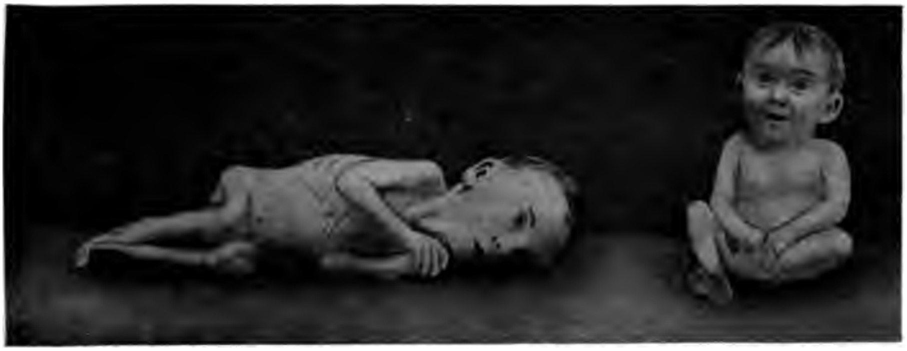


FIG. 178.—*a*, infantile atrophy from improper feeding (female ten months old); *b*, recovery after three months.—Rotch.

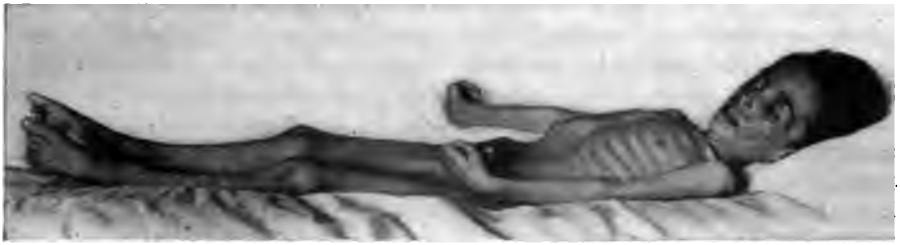


FIG. 179.—General atrophy of the muscles in a case of cerebrospinal fever; fifty-fifth day of illness.—Royer.

myopathic atrophy in the frequent occurrence of derangements of sensation, pain, and fibrillary contractions and in the occasional presence of the reaction of degeneration. In many cases of peripheral neuritis the affected muscles undergo degenerative atrophy. Spinal or nuclear atrophy usually first shows itself in the intrinsic muscles of the hand and by extension early involves the tongue, lips, palate, pharynx, and larynx, giving rise to the picture of bulbar paralysis. Fibrillary contractions of the muscles are common and reactions of degeneration occur. The disease develops almost exclusively in adult life and is not hereditary.

The Atrophic Paralyses.—The muscles undergo secondary degenerative atrophy. The lesion which interferes with the transmission of motor impulses at the same time interrupts trophic influences to the muscle. The paralysis shows itself first and is followed by atrophy, which in the course of some weeks becomes marked and often reaches a very high grade. The reactions are those of degeneration. In this form of degenerative atrophy fibrillary contractions are frequently present.

THE POSTURE, ATTITUDE, AND GAIT.

Posture.

Patients who are very ill of an acute disease or in the advanced stages of chronic disease are usually seen in bed; those suffering from trifling affections or in whom the symptoms of grave disease are not yet urgent or disabling continue to be about, but this is not always the case. Whether, on the one hand, a patient remains up and about, endeavoring to attend to his ordinary duties while suffering from serious symptoms or, on the other hand, betakes himself to bed upon the occurrence of trifling symptoms is often a matter of temperament. It is not uncommon for a patient suffering from enteric fever to come to the consultation room or dispensary in the second week of the attack with a temperature of 104° F. (40° C.) and a well-developed rose rash—WALKING TYPHOID. Patients who realize their condition very often feel compelled by circumstances to continue the discharge of a daily duty or are buoyed up by the hope of speedy improvement, and again there are acute diseases which run a favorable course which begin with urgent and distressing symptoms. The physician usually finds those patients in bed who have high fever, prostration, or a general sense of serious illness, and those who suffer from dyspnœa, pain, vertigo, and other symptoms intensified by movement or exertion. In meningitis, peritonitis, rheumatic fever, pericarditis, typical croupous pneumonia, and in well-developed cases of the acute exanthemata it is impossible for the patient to be out of bed. It is to be noted, however, that upon the appearance of the eruption in the variolous diseases the symptoms of onset often undergo such an amelioration that the patient regards himself as convalescent and insists upon getting out of bed.

Decubitus is the posture of the patient in bed. It is of diagnostic importance. It is in moderate illness, as in health, easy and unconstrained. The patient arranges the bed-clothes, changes his position when it has become uncomfortable, lies naturally upon his back—ACTIVE DORSAL

DECUBITUS—or turns upon the side—ACTIVE LATERAL DECUBITUS. The posture of weak, helpless, or unconscious individuals in bed is wholly different. The muscles play little part in maintaining the position. The relaxed body yields to the law of gravity and sinks toward the foot of the bed, where it remains. The patient, even when his breathing is hindered and his position is uncomfortable, is unable to change it. The attendants must again and again lift him upon the pillows. The condition is wholly passive—PASSIVE DORSAL DECUBITUS. In rare instances the patient in this state lies upon the side—PASSIVE LATERAL DECUBITUS.

Forced or imperative attitudes are very characteristic of certain diseases. The following are the most important:

The Dorsal Posture.—In acute peritonitis, whether general or local, the patient lies upon the back with the thighs flexed upon the abdomen and the legs upon the thighs. Movement is avoided and the patient shrinks from pressure upon the abdomen.

The Reclining Dorsal or the Sitting Posture.—In diseases attended with difficult respiration, especially certain diseases of the respiratory and circulatory organs and the kidneys, the patients are forced to assume a semi-upright posture on the bed-rest or propped up with pillows, or to sit upright. Attempts to lie flat in bed increase the difficulty of respiration. The sitting position relieves it by favoring the action of the accessory respiratory muscles, especially when the arms are used to elevate and fix the shoulders. In the case of peritoneal effusions the respiratory movement of the diaphragm is less interfered with in the sitting posture unless the effusion be very large, in which case the abdomen is somewhat compressed by the thighs. This attitude, furthermore, favors the return of the venous blood from the brain. For this reason high grades of dyspnœa are described under the term ORTHOPNŒA. When the difficulty of respiration is extreme the patients can no longer remain in bed but are obliged to sit upright, fixing the shoulders by placing the hands upon the side of the chair or its arms in order to facilitate the use of the accessory muscles and to relieve the abdomen from the pressure of the thighs. The distress is also to some extent relieved by the gravitation of venous blood and the fluid of general dropsical effusions to the lower extremities. Orthopnœa is present during the paroxysms of asthma, in extreme cases of valvular disease of the heart with ruptured compensation, in large pleural and pericardial effusions, in massive peritoneal effusions, and in general anasarca, which may be cardiac or renal but is very often cardiorenal. It occurs also in advanced pulmonary emphysema and in obstructive diseases of the larynx, as croup and diphtheria. Except in extreme cases it is usually paroxysmal, the attack being brought on by movement, coughing, conversation, or other exertion.

Lateral Postures.—Patients suffering with unilateral disease of the thoracic organs very often lie upon the affected side. This is especially the case in large pneumonic exudates, pleural and pericardial effusions, and other conditions which greatly diminish the respiratory surface of the affected lung. In this posture the respiratory excursus of the sound side is not hampered by the weight of the diseased organs. In painful conditions, however, the patients sometimes lie upon the sound side. In acute fibrinous pleurisy the pain of which is greatly intensified by breathing, the lateral decubitus upon the affected side is assumed by preference because the weight of the body somewhat diminishes the respiratory excursus of that side of the chest. Patients suffering from heart disease and many individuals in good health lie more comfortably upon one side than upon the other; sometimes the right side is preferred, sometimes the left. In cardiac hypertrophy the patients usually lie more comfortably upon the left side, and in large aneurisms of the aorta, upon the affected side. Patients suffering from harassing cough in the dorsal position are sometimes relieved by turning upon one side. This happens in certain cases of unilateral pulmonary cavity and the relief is obtained by turning upon the affected side. The explanation of this phenomenon is purely physical; while the patient lies upon his back or upon the sound side the secretion formed in the cavity escapes into the bronchus little by little, causing irritation which manifests itself by cough, while, on the other hand, if he continues to lie upon the affected side it collects without producing reflex cough until the cavity overflows. The lateral decubitus with the thighs and legs flexed upon the abdomen and the spine and neck strongly arched forward is usually assumed during the pains of parturition and is common in hepatic and intestinal colic. In acute cerebrospinal meningitis the patient frequently lies upon the side with the thighs and legs strongly flexed and the spine extended in the position of OPISTHOTONOS. In some cases the lower extremities are extended—COMPLETE OPISTHOTONOS.

The ventral posture is sometimes assumed in cases of abdominal pain, as colic, gastralgia, or enteralgia. The patient lies prone upon the bed with his face buried in the pillow. Tenderness upon abdominal pressure, as in peritonitis, renders this attitude impossible. It sometimes affords relief to the pain of abdominal aneurism and in certain cases of caries of the spine. In most cases of gastric ulcer this posture is avoided on account of the epigastric tenderness upon pressure. In some cases of this disease, however, the pain is relieved by the ventral decubitus, probably because the ulcer is so situated as to escape in this position the pressure of the contents of the stomach. Patients suffering from headache very often assume this posture.

Restlessness in bed is a very common symptom. The patient is unable to maintain the same position for any length of time; he tosses about, turns from side to side, fusses at the bed-clothes, and his hands and feet are in constant motion. Restlessness may be the manifestation of nervous irritability or of pain. It is common in affections attended with burning and itching of the skin, as scarlet fever and urticaria. It occurs also in some cases of shock and accompanies profuse hemorrhage, in which case it is attended with pallor, urgent thirst, and rapid, small pulse. In truth the association of restlessness with these symptoms, occurring suddenly without visible bleeding, warrants a provisional diagnosis of internal hemorrhage. The term JACTITATION is used to designate a high degree of restlessness. The patient tosses about violently; the constant efforts of the attendants are necessary to keep him in bed. Jactitation occurs in maniacal delirium, in cases of violent chorea, in which it is accompanied by constant twitching of the muscles, as a temporary manifestation in me forms of hysteria, and in a high degree during the stage of clonic nvulsions in epilepsy.

OPISTHOTONOS, predominating tonic contraction of the spinal muscles, that the body rests upon the head and heels; EMPROSTHOTONOS, or nding forward of the trunk; PLEUROTHOTONOS, arched lateral posture;

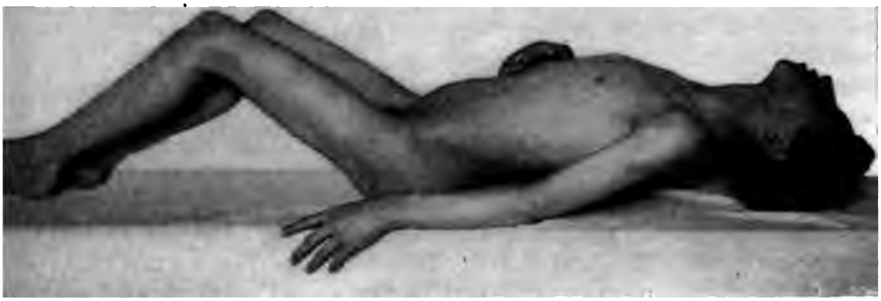


FIG. 180.—Opisthotonos in a case of epidemic cerebrospinal meningitis.—Royer.

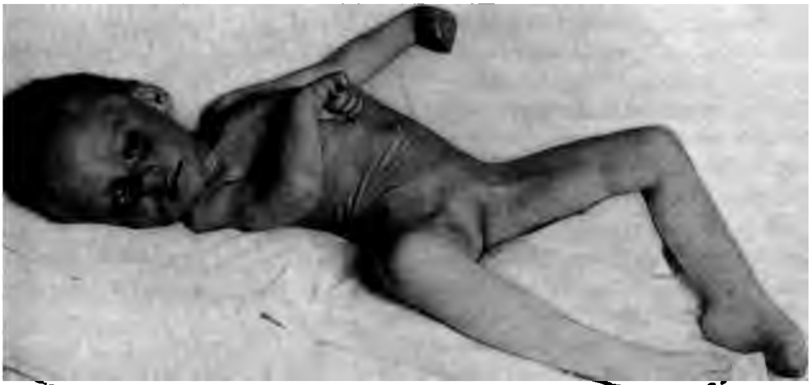


FIG. 181.—Pleurothotonos in a case of epidemic cerebrospinal meningitis.—Royer.

d ORTHOTONOS, in which the trunk and neck are rigidly extended in a raight line, are all symptoms that occur in tetanus and in some cases of eningitis and strychnine poisoning.

Attitude.

The attitude and movements of patients who are able to be about equently convey important information in regard to their condition. e young and the strong carry themselves erect and walk briskly and mly; the aged and feeble and those mentally depressed are bowed and ove slowly and with effort. The convalescent from a prostrating disease at first weak and shaky; he can scarcely stand; an hour in the arm-chair tigues him. In a little time he makes the journey around his room with slow and uncertain gait, and is soon obliged to rest. With returning strength comes the erect carriage and firmer step. Modifications of attitude and gait constitute characteristic symptoms in many diseases. In general they are due to skeletal defects, as in caries of the spine, hip-joint disease, or ankylosis of the knee; derangements of the muscular power or function, as in pseudohypertrophic muscular paralysis, chorea, and the shaking palsies; derangements of the balance between antagonistic muscle groups, as in forms of spinal curvature and club-foot; derangements of coördination, as in cerebellar disease and tabes; forms of paralysis, as in hemiplegia, anterior poliomyelitis; and contractures, as in the cross-legged progression of children suffering from spastic paraplegia.

Station is technically the ability to maintain the erect position while standing. It depends largely upon muscular and visual coördination. Within limits it is better the wider the base of support, hence the test should be made with the feet parallel and the heels and toes touching, first with the eyes open, later with them closed. Hinsdale found in normal individuals of both sexes the average oscillation in the above position, as determined by an instrument devised for the purpose, to be about an inch in a forward and backward line and three-quarters of an inch laterally. The oscillation in children is greater than that in adults. Upon closing the eyes it is increased about 50 per cent. In diseases characterized by impairment of the power of coördination, as tabes and lesions of the cerebellum, station is greatly impaired and the patient may be wholly unable, under the conditions of the test and with closed eyes, to keep his balance—ROMBERG'S SYMPTOM. During paroxysms of Ménière's disease—aural vertigo—the power of standing in the erect posture is wholly lost. *Astasia* is a term employed to designate inability to stand, *abasia* the inability to walk, in the absence of paralysis. ASTASIA-ABASIA is a syndrome of hysteria in which the patient is unable to stand or walk but can usually creep about like a child, upon the hands and knees.

The following peculiarities of attitude are to be noted:

In HEMIPLEGIA and paralysis of one leg the patient supports himself almost entirely upon the sound leg. In CHRONIC SCIATICA the patient spares the affected limb both in walking and standing by fixation of the hip-joint, and in doing so develops a scoliosis, the spinal column showing a double curvature, the lower convex, the upper, which is compensatory, concave toward the affected side, the general inclination of the body being toward the sound side. In PARALYSIS AGITANS the attitude is characteristic. The head and upper part of the body are inclined forward, the elbows and knees being slightly flexed. The striking appearance of the patient is heightened by the expressionless countenance, the tremor, and the movements of the fingers and hands. In PSEUDOHYPERTROPHIC PARALYSIS the patient stands with his feet separated, the belly protruding, and the shoulders thrown back as the result of marked lordosis. In the sitting posture the curvature of the spine is corrected.

Gait.

In a number of diseases, especially those affecting the nervous system, the gait is much modified and its peculiarities often justify conclusions regarding both functional derangements and anatomical lesions. The following symptomatic gaits are frequently observed:

The Paraplegic Gait.—In paresis of the lower extremities the gait is feeble and uncertain. Both feet are slowly advanced and dragged upon the floor. The patient stumbles over trifling inequalities and elevations of the surface. The loss of power is frequently more marked on one side than on the other. Crutches become necessary and at length the loss of power is complete. This gait is seen in chronic myelitis.

The Hemiplegic Gait.—When the hemiplegic has sufficiently recovered to walk, the gait is characteristic. The sound limb is advanced, the paralyzed limb dragged after it. In other cases the step of the paralyzed limb is accomplished by lifting the pelvis and a movement of circumduction. When contractures have taken place the affected arm is rigid, strongly flexed at the elbow and wrist and carried across the body, and the fingers and thumb are flexed upon the palm.

The Spastic Gait. — In spastic paresis of the lower extremities such as occurs in forms of spinal paralysis there is peculiar stiffness of the legs, which are scarcely bent at the hip- and knee-joints, while the thighs interfere with each other by reason of the contraction of the adductors. The contraction of the gastrocnemii produces pes equinus. The patient walks with two canes and in stepping leans upon one, lifting the pelvis of the opposite side as he steps, and dragging the foot in circumduction. In some cases the contact of the foot with the floor produces ankle clonus which adds to the peculiarity of the gait. A modification of the spastic gait, sometimes seen in children, is known as cross-legged progression. In consequence of the contraction of the adductors and calf muscles there is close circumduction of the knees, and in stepping the legs are crossed and the advancing foot brought down not only in front of but to the outside of its fellow.

The Steppage Gait.—In some cases of peripheral neuritis the paralysis of the extensors of the feet causes a peculiar modification in progression. In stepping forward the knee is strongly flexed and the foot sharply advanced in order that the dragging toes may be lifted from the ground; the heel is brought down first and the appearance is that of a person stepping over obstructions.

The waddling gait occurs in pseudohypertrophic muscular paralysis and is not less characteristic than the attitude in this disease. In consequence of the lordosis the shoulders are thrust back and the belly forward, the legs are separated, the feet raised slowly with the toes dropping, the centre of gravity being alternately shifted over the foot upon which the patient throws his weight. The manner in which the child, after lying down upon the floor, gets up is especially characteristic. He rolls over upon the abdomen, gets upon all fours, and first extends the arms, then the legs. The hands are next drawn toward the legs until he can grasp one knee with the corresponding hand. He pushes himself up until the other knee can be grasped and assumes the erect posture by gradually raising the point of support of the hand upon the thigh. Late in the disease, when the atrophy involves the muscles of the upper extremities, it becomes impossible to rise.

The ataxic gait is that of incoördination of the lower extremities. It is observed in its most typical form in tabes dorsalis. In stepping the foot is raised higher than usual with a jerk and rapidly advanced with an awkward and irregular movement, the toes slightly drooping. It is then brought down with an abrupt stamp upon the heel or the entire sole. Progression is irregular and it is impossible for the patient to walk with one foot before the other, as in following a crack upon the floor or a chalked line. He walks with a swaying motion. The legs are separated in order to increase the base of support, which is further enlarged as the disease makes progress by the use first of one cane, later of two. In advanced cases walking becomes impossible without the aid of one or even two attendants. Finally, the power of locomotion is entirely lost. These symptoms of impaired coördination are greatly increased upon closing the eyes. Patients who can go about fairly well in daylight cannot walk at all in the dark.

The gait of sciatica derives its characteristics from muscular fixation of the hip-joint voluntarily brought about to diminish pain.

The Gait in Chorea.—In severe chorea the irregular muscular contractions interfere greatly with ordinary movements. The gait of the patient is often hopping or sliding, sometimes it resembles the movements of skating. In the worst cases walking becomes impossible.

The reeling or staggering gait is a form of the ataxic gait. It occurs in conditions attended with marked disturbance of coördination, such as drunkenness, cerebellar disease, lesions of the labyrinth, and some forms of paralysis of the muscles of the eye.

The Festinating Gait.—This modification of walking occurs in paralysis agitans and is not less characteristic than the attitude in that disease. The patient bends forward, the elbows are slightly abducted and flexed, the knees are also flexed, and the patient walks with the appearance of haste, as though to overtake his advancing centre of gravity. He cannot halt at once. The peculiarity of the gait is largely due to stiffness and weakness of the muscles. The gait is sometimes described as propulsive. A similar gait and inability to stop immediately sometimes shows itself in exhausted pedestrians. Retropulsion may occur.

Posture and Movements of Infants.

The position and movements of infants are of diagnostic importance. The healthy baby uses its muscles and joints. Its postures are active, its movements constant, and a source of evident pleasure. It loves to be fondled and played with. How different the baby who is really ill! Its postures are passive. Its head drops and rolls from side to side with the motion of the pillow upon which it rests. Its limbs dangle helplessly, and voluntary movements are slight and infrequent. In many febrile diseases there is cerebral irritation, shown by the drawn face and head pressure deep into the pillow. In severe rickets there is tenderness of the muscles and bones, motion is painful and therefore avoided; in infantile scurvy a similar condition exists, and in well-developed cases the attitude is almost diagnostic, the child lying upon its back with the thighs and legs strongly flexed, shunning all movements and screaming with fear if it is approached. In cerebrospinal fever and other forms of meningitis there is painful retraction of the muscles of the back of the neck—opisthotonos.

IV.

TEMPERATURE; FEVER; HYPOTHERMIA; SIGNIFICANCE OF ABNORMAL TEMPERATURES.

TEMPERATURE.

Variations in the temperature of the body constitute symptoms of great importance both in acute and in chronic disease. From the earliest times practitioners estimated the heat of the body by the hand and thus sought to determine the presence or absence of fever. The introduction of the clinical thermometer into medical practice marked an important advance in modern medicine. (See Part II, Clinical Thermometry.)

Heat Mechanism.—The temperature of homothermous or warm-blooded animals is constant within narrow limits and is not materially influenced by changes in the temperature of the medium in which the organism lives. In the human being the amount of heat produced and dissipated at different parts of the body varies. The equilibrium of temperature is maintained in part by direct conduction but chiefly by the circulating blood and lymph. The internal parts of the body have nevertheless a higher temperature than the external and some internal organs are warmer than others. The heat production is greater in organs when they are active than when they are at rest, and the temperature varies in different regions of the surface of the body. The heat mechanism is made up of two factors: (a) heat production or *thermogenesis*, and (b) heat dissipation or *thermolysis*. Under normal conditions these two functions so nearly balance that the mean bodily temperature is maintained within very narrow limits. The regulating mechanism is expressed by the term *thermotaxis*. It is obvious that thermotaxis may be deranged by alterations in either thermogenesis or thermolysis.

THERMOGENESIS accompanies oxidation. Hence almost every structure of the body may be regarded as the source of heat. In this respect the skeletal muscles and the glands play the chief part. The general thermogenic centres have been shown to be in the spinal cord. Thermogenic centres probably exist in the caudate nuclei, pons, and medulla oblongata; excitation of these regions is followed by a rise in heat production—puncture pyrexia. They are therefore known as thermo-accelerator centres. Irritation of the region of the sulcus cruciatus and at the junction of the supra-Sylvian and post-Sylvian fissures in the dog is followed by a decrease in heat production. These centres are therefore known as thermo-inhibitory.

Thermolysis or heat dissipation is the result of radiation and conduction from the surface, of the evaporation of water from the lungs and skin, and of the warming of the food, drink, and inspired air.

Thermotaxis or heat regulation is brought about by reciprocal changes in heat production and heat dissipation through the action of cutaneous impulses and of variations in the temperature of the blood upon the thermogenic and thermolytic centres. Thus in an animal exposed to moderate cold, heat dissipation is increased, but cutaneous impulses are generated which excite the thermogenic centres and heat production also is increased, whereas an increase of the temperature of the blood increases the activity of the thermolytic process. In either case the temperature of the body is maintained. Under abnormal conditions this reciprocal influence is deranged.

Abnormal thermotaxis is a term used to designate the regulation of the heat mechanism under pathological conditions in which the body temperature is maintained at a range higher or lower than that of health.

Under ordinary circumstances the presence or absence of hyperthermia may be determined by the hand, but this mode of observation yields no accurate data either for comparison or record. An impression as to the surface temperature is thus obtained but this does not always correspond with the internal temperature of the body. During a chill the temperature of the skin, in consequence of the contraction of the arterioles, is in most instances greatly reduced, while the internal temperature, as determined by the thermometer, is high. On the other hand, when the skin is active and perspiring and evaporation is prevented by the bed-clothing, the surface may feel hot to the hand while the internal temperature remains normal. The normal axillary temperature ranges about 98.6° F.—37° C. It undergoes diurnal oscillations of a degree to a degree and a half, falling to 97.5°-98° F. in the early hours of the morning and rising to 99°-99.3° F. toward evening. It is very probable that this physiological oscillation is dependent upon the alternations of sleep and waking. Observations upon men who have habitually slept during the day and watched during the night have shown an inversion of the curve. A slight physiological rise takes place during gastric digestion. Violent physical exercise is frequently followed by a temporary rise of two or three degrees; this fact may, in part at least, explain the elevation of temperature sometimes observed after a violent general convulsion and which is very common in the *status epilepticus*. In children and adolescents the range is somewhat higher than in adults and also less stable, that is to say, the diurnal physiological oscillations are slightly greater and the sensitiveness of the temperature to pathogenic influences more marked.

Kieffer states that careful observation has shown that permanent increase of external heat in the tropics is followed by a rise of bodily temperature of .05° F. for every degree of external heat above the mean annual norm and that as a direct consequence the respiratory function is diminished, the pulse-frequency slightly decreased, the digestion, appetite, and assimilation unfavorably affected, the functional activity of the skin greatly increased, and the nervous system distinctly depressed.

In aged persons the diurnal temperature range in health is slightly lower and may fall to 97° F. (36.1° C.). In very aged persons, on the other hand, the range may be as high as in children.

The action of prolonged or intense heat and cold upon the temperature must be regarded as pathological.

Abnormal Temperature.—Variations in the body temperature may be plus to progressively higher ranges, designated subfebrile and febrile, the latter comprising (a) slight fever, (b) moderate fever, (c) high fever, (d) hyperpyrexia; or minus—subnormal temperature and the temperature of collapse.

The term pyrexia is used to designate conditions characterized by elevation of temperature; hyperpyrexia, those marked by excessively high temperature; and apyrexia, the absence of fever. Hypothermia is the term applied to conditions in which the temperature is subnormal.

FEVER.

Elevation of temperature alone does not constitute fever. Extreme transient rises of 104° F. (40° C.) have been observed after violent, prolonged gymnastic exercises, and much higher temperatures in hysteria, in neither case associated with the other symptoms which enter into the modern conception of fever. These symptoms are, in addition to elevation of temperature, subjective sensations of illness, cerebral phenomena, weakness, loss of appetite, thirst, increased frequency of pulse and respiration, altered urine, and derangements in nutrition which cause wasting of the body. It is furthermore essential to our conception of fever and necessary to the complete manifestation of the symptom-complex that the process should occupy a certain time. There are, however, febrile periods of minimal duration, as for example in the course of the ague paroxysm, in which most of the objective symptoms occur or in which, if the paroxysm is repeated for some time at quotidian or tertian intervals, all of them, including wasting of the body, are manifest. On the other hand, the acute febrile infectious diseases usually run a self-limited course, measured by days or weeks; again, in certain of the chronic infections, as forms of tuberculosis, there may be fever every day for months. Nor are these symptoms altogether dependent upon or caused by the elevation of temperature, as is shown by the fact that artificial over-heating of the body produces certain of them but not all, that in different diseases their intensity by no means corresponds to the degree of the temperature, and that marked falls of temperature can occur either spontaneously or as the result of antipyretic treatment without a corresponding amelioration in other respects. Elevation of temperature is nevertheless a constant and essential element in the condition known as fever and in certain cases dominates the clinical picture. In a majority of instances, however, the associated conditions constitute a much more important measure of the gravity of the case than the range of temperature.

Causes of Fever.—It is evident that the causes of fever act through the nervous system and thus produce derangements of the heat-regulating function. At the same time they also produce derangements of the normal tissue changes with increased oxidation and heat production. They consist of soluble toxic substances circulating in the blood and are, (a) the result of infection by micro-organisms, which may be general or local, or (b) the result of intoxication, which may arise within the body itself from faulty metabolism or be introduced from without, as in food poisoning. In cases of infection with profound nutritive disturbances toxins derived from both these sources are present. In either event, whether the fever-producing agent be a toxin produced by the growth and development of micro-organisms or an albumose, ferment, or ptomaine produced by faulty cell metamorphosis within the organs or tissues themselves, the condition constitutes a toxæmia.

Sapræmia is an infection of the blood by putrefactive products.

It is probable that in the rare cases of fever attributed to intense emotion, as fright, or to violent pain or peripheral irritation, the rise of temperature is caused by the sudden derangement of physiological processes, with the production of toxins, rather than by direct action upon the heat-regulating processes, and that in many, though not all, of the cases of cerebral disease accompanied by fever, as thrombosis, hemorrhage, and tumor, the elevation of temperature is due to local infection rather than to implication of the heat centres, while the symptom-complex and the condition of the blood in sunstroke render it highly probable that the elevation of temperature is due not so much to the direct effect of heat upon the nervous system as to toxic substances generated by the action of heat upon the tissues of the body and especially upon the muscles. It is thus seen that many different pathogenic principles developed within the body or introduced from without are directly or indirectly capable of producing the reaction which we designate by the term fever.

Symptoms of Fever.—These substances not only cause elevation of temperature and more or less marked disturbances of nutrition but they also produce subjective sensations of illness and cerebral symptoms, such as headache, somnolence, stupor, and, in grave cases, coma and delirium, which may be mild and wandering or active and maniacal. Among the effects produced upon the nervous system must be included the profound sensation of weakness often present in the early stages of febrile diseases and which bears no direct relation to the inability to take food or to the wasting of the tissues of the body which occurs later. They produce derangements of the normal secretions, which are manifested on the part of the skin by dryness and heat or, in some cases, and especially at the time of defervescence, by profuse, even colliquative sweating, on the part of the gastro-intestinal tract by thirst, loss of appetite, dry, furred tongue, impaired digestion, and constipation, and on the part of the urinary apparatus by scanty, high-colored urine of increased specific gravity.

Pulse in Fever.—Derangement of the pulse-frequency is a constant phenomenon of fever. To what extent it is due to elevation of the temperature and to what extent to the action of fever-producing toxins upon the nervous system cannot be determined. In almost all cases of fever there is an acceleration of the pulse-rate, the frequency of which usually corresponds to the intensity of the fever. Liebermeister found that for every degree centigrade (1.8° F.) of elevation of temperature above the normal there is an increase of eight beats of the pulse. This parallelism between the temperature and pulse may be regarded as relatively favorable, whereas a greatly increased pulse-frequency indicates serious cardiac or vasomotor disturbance and is of unfavorable prognostic significance. A pulse-rate of 140–160 in the adult while resting quietly in bed is in itself a very serious symptom. The pulse-frequency in children suffering from febrile diseases is relatively high. In phthisis with moderate fever or even in the absence of fever there is commonly a quickened pulse. There are cases in which, notwithstanding marked elevation of temperature, the pulse-rate remains low. This departure from the ordinary parallelism is of diagnostic importance. High temperatures with slow pulse are observed in cases of cerebral disease in which there is pressure at the base, as tuberculous meningitis, in yellow fever, and in febrile diseases in individuals suffering from cardiac lesions attended by diminished pulse-frequency, as sclerosis of the coronary arteries and myocarditis. It is to some extent characteristic of enteric fever that the pulse-frequency is moderate as compared with the elevation of temperature, and this want of correspondence is of importance in the differential diagnosis between enteric fever and acute miliary tuberculosis or septicæmia, in both of which the pulse-rate is high.

Respiration in Fever.—Increased frequency of respiration occurs in almost all cases of fever. That this phenomenon is in part due to the stimulating effect of the heated blood upon the respiratory centre has been shown experimentally; exposure to artificial heat increases the frequency of breathing. That it is also in part due to the direct action of the fever-producing toxins upon the respiratory centre is rendered probable by the fact that the acceleration of breathing bears no direct ratio to the elevation of temperature but varies greatly at the same temperature in different diseases. It is a matter of experience that cases of febrile disease in which, in the absence of complications on the part of the respiratory organs, the respiration frequency is greatly increased are almost always of grave import.

Emaciation.—Wasting accompanies fever. Even in febrile attacks of moderate duration the loss of flesh may be marked; in prolonged fevers emaciation may be extreme. The blood undergoes analogous changes, the patient becomes anæmic, and the loss of flesh at the close of a prolonged fever is not more striking than the pallor. A decrease in the number of the erythrocytes accompanies all cases of pyrexia, but requires some time to become manifest. There is progressive loss of the albumins of the plasma.

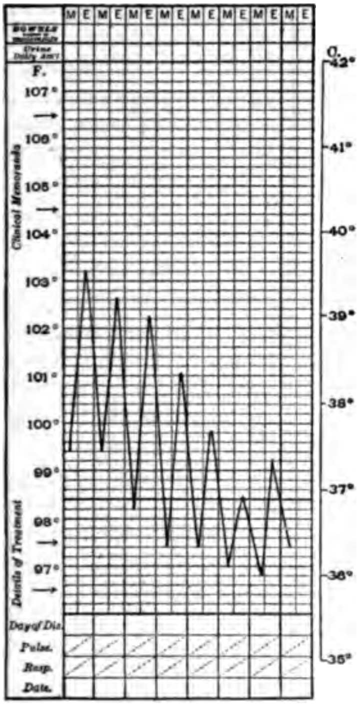
Pyrexia a Symptom.—The clinical significance of fever would be much less important were it not for the fact that the febrile movement, in its mode of onset, intensity, course, and decline, bears a relation to the particular morbid condition in which it occurs, frequently definite and always suggestive.

Until recently much stress was laid upon the distinction between symptomatic fever and essential or idiopathic fever. The former was regarded as a manifestation of some local malady, the latter as constituting the actual disease. The stimulus given to the study of causes by the science of bacteriology has shown that this distinction is more apparent than real and that in the light of modern pathology pyrexia is always a symptom

IDIOPATHIC FEVER.—Nevertheless there is a group of acute infectious diseases in which fever is not only constantly present but also the most conspicuous symptom, and in which the morbid process is literally coextensive with the febrile movement, which is self-limited, the illness beginning with the rise of temperature and the convalescence setting in with defervescence. This group constitutes the idiopathic fevers or, more simply, the fevers.

Varieties.—Subdivisions, arranged according to the course of the febrile movement, are (a) the continued fevers, as influenza and enteric fever, and (b) the periodical (malarial) fevers, as intermittent, remittent, and pernicious fever. In some of the continued fevers other symptoms, as eruptions, are no less constant or characteristic than the course of the fever,—a fact which led to the establishment of a further subdivision upon an entirely different basis of classification, which comprises the EXANTHEMATA OR THE ERUPTIVE FEVERS. Furthermore, in certain of the diseases which are regarded as continued fevers, a characteristic periodicity occurs, or the course of the disease is interrupted by periods of apyrexia of considerable duration, an example of which is relapsing fever, whereas in the periodical fevers, strictly so-called, namely, the malarial infections, there are certain cases in which the febrile movement lacks distinct periodicity—continued malarial fever—or is absent altogether—malarial infection without fever. On the other hand there is a large group of diseases that has nothing to do with malaria in which the occurrence of febrile paroxysms, separated by very definite periods of apyrexia, in other words, distinct periodicity, is characteristic—for example, the hectic fever of pulmonary tuberculosis, hepatic fever, urethral fever, and the fever in some cases of malignant endocarditis. Finally, there are local and general infections in which the symptom fever is inconstant and irregular. For these and other reasons, the principal of which is that fever is always symptomatic and never of itself an actual disease, the distinction between symptomatic fever and essential or idiopathic fever has been abandoned—a long step in the direction of a scientific or etiological basis for the classification of diseases. Terms and phrases that have long lost their original significance remain to encumber the literature and embarrass the study of medicine and the period is remote when we shall cease to speak of scarlet fever or yellow fever.

Type in Fever.—Type is a term loosely used to indicate the intensity of fever. Thus we speak of fever of mild type or fever of grave type. It is applied more accurately to the course or range of the temperature as depicted upon clinical charts. There are three principal types of fever: (a) the CONTINUED, in which the limits of the diurnal range do not usually exceed 1.8° F. (1° C.), the fall occurring in the morning, the rise in the evening. This is about the measure of the diurnal oscillation in health. There is, therefore, a parallelism between the temperature of health and fever of the continued type, the latter being elevated two or more degrees above the former and fluctuating in harmony with it. Since the temperature range upon the chart is represented not by a straight but by a curved line showing the diurnal oscillations it is better to describe this as the SUBCONTINUOUS type. Fever of this type is characteristic of the fastigium uncomplicated enteric fever. (b) The REMITTENT type, characterized by lls of several degrees in the temperature, which does not, however, reach e normal. The remissions may take place at any hour of the day and are ten accompanied by free sweating. They are followed in the course of a w hours by exacerbations of greater or less extent. There is no parallelism etween fever of this type and the normal temperature range. This is the pe seen in some forms of estivo-autumnal malaria and in septic condions. (c) The INTERMITTENT type, characterized by a fall of temperature om febrile ranges to the normal or below it, a period of apyrexia of varile duration, and the recurrence of fever. The febrile paroxysms are of ort duration as compared with the intermission and commonly begin th a chill and terminate in profuse sweating. During the intermission e patient usually feels fairly comfortable or quite well. Fever of this pe occurs in malaria. The repetition of the paroxysms may extend er a considerable time. Intermittent fever in which the paroxysm recurs ily is known as *quotidian;* when the paroxysm recurs upon the third y, including the day of onset, it is *tertian;* when it recurs upon the fourth y, *quartan.* The paroxysms may occur at any period of the day and ually at the same hour. In malaria they ordinarily recur in the foreon, in hectic fever in the afternoon. (d) THE INVERSE TYPE. The tem-



ɔ. 182.—Fever of remittent type: lysis in enteric fever.

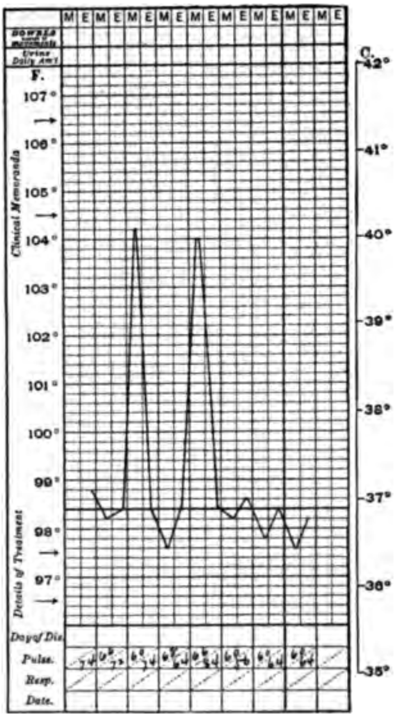


FIG. 183.—Intermittent malarial fever, tertian type; single tertian infection. Man, aged thirty-three.

perature in fever of the continued type and in many cases of the remittent type undergoes diurnal oscillations of wider excursus than those of health but corresponding to them in time. That is, the remission occurs in the early morning hours, the exacerbation toward evening. In exceptional cases the remission takes place in the evening and the exacerbation in the morning—*inverse type.* Fever of this type occasionally occurs in tuberculosis and in rare instances in enteric fever.

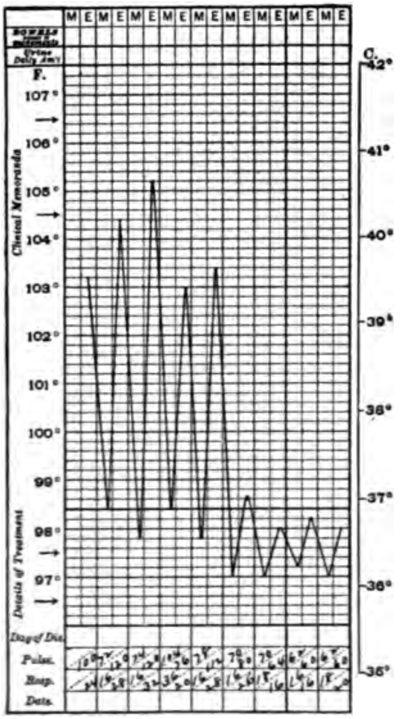


Fig. 184.—Intermittent malarial fever, quotidian type; double tertian infection. Man, aged twenty-nine.

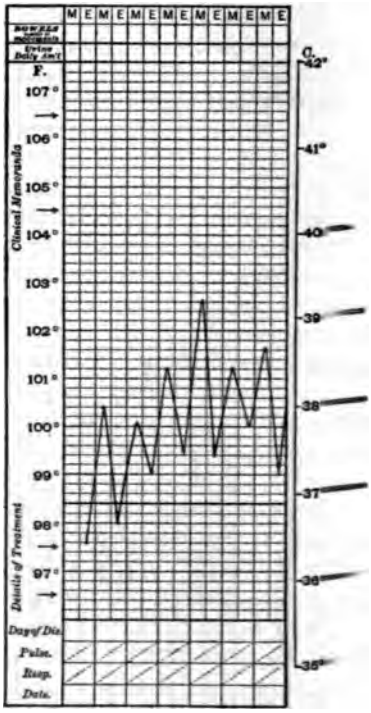


Fig. 185.—Temperature of inverse type.

Atypical Fever.—In many febrile diseases the temperature range is altogether irregular. This is especially the case in diseases in which the symptoms in general are irregular or atypical, as diphtheria and the various septic infections.

The Type of Fever in Particular Diseases.—Many of the febrile infections have a characteristic temperature range. The febrile movement in uncomplicated cases on the one hand is self-limited and on the other undergoes definite modifications at different stages in the course of the affection and upon the occurrence of special manifestations, as the appearance of an eruption. In a more narrow sense the temperature range in such diseases is said to be typical or to conform to type. It is to be borne in mind, however, that marked departures from type may occur in consequence of variations in the intensity of the infection, peculiarities on the part of the individual, the occurrence of complications, and from the action of drugs. The type of fever, both as regards the daily range and the temperature curve throughout the course of the attack, constitutes a valuable aid to diagnosis, and is always to be taken into consideration. It is rarely possible, however, to make a diagnosis from the temperature alone, nor is it desirable. In connection with the temperature we must consider the other symptoms and signs, the surrounding circumstances, and the previous treatment.

In well-developed cases, unmodified by complication or treatment, the temperature curve may be said to be characteristic in the following diseases: tertian and quartan malaria, enteric fever, typhus fever, relapsing fever, and croupous pneumonia. It conforms in a general way to type, but less closely, in scarlatina, measles, erysipelas, and the variolous diseases. It is variable and atypical in cerebrospinal fever, rheumatic fever, endocarditis, and the septic infections.

Stages.—The course of the attack may be divided into (a) the stage of prodromes, (b) the onset or stage of invasion, (c) the fastigium, and (d) the defervescence or stage of decline. In typical cases of the different febrile diseases each of these periods has a definite duration and a characteristic curve upon the temperature chart.

(a) **The Stage of Prodromes.** — This period is usually marked by vague feelings of discomfort, lassitude, pain in the back, unsound sleep, and feverishness, the temperature reaching subfebrile or even mild febrile elevations in the later part of the day. These symptoms are often absent. Prodromes usually occur in diseases of gradual development. They are common in enteric fever.

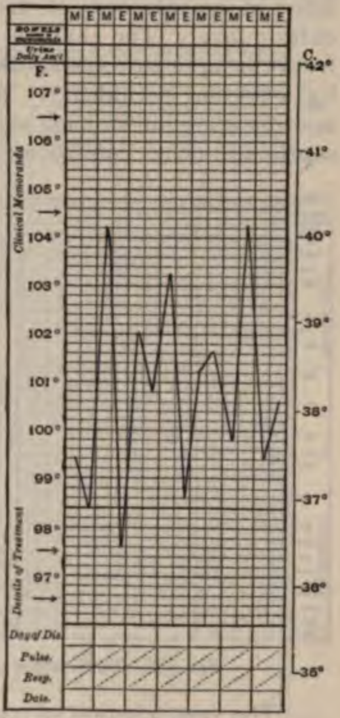


FIG. 186.—Fever of irregular periodicity.

(b) **The Onset or Stage of Invasion.**—The rise of temperature may be gradual or abrupt. When gradual the evening exacerbations exceed the morning remissions in such a way that the temperature rises progressively to the fastigium or acme. Under these circumstances the stage of invasion may occupy a period of several days, as in enteric fever. When abrupt the acme is reached at once or in the course of a few hours, as in scarlet fever, influenza, or croupous pneumonia. The onset is very often attended by chilliness or a chill. This symptom may vary in intensity from transient sensations of cold, with shivering, pallor, and slight cyanosis of the lips and finger-tips, to a severe and prolonged chill or rigor, with violent shaking or tremor of the whole body, chattering teeth, cold extremities, and marked cyanosis. The temperature of the surface of the body is much reduced and the patient experiences a sensation of extreme cold, whereas the internal temperature, taken in the rectum, is high, 104°–107° F. (40°–42° C.). The violence of the chill commonly corresponds with the abruptness of the onset. The insidious and gradual invasion of enteric fever is not often attended by chills. The abrupt onset of croupous pneumonia very frequently manifests itself by a prolonged chill of great severity, occurring without warning in a condition of apparent health. The chill which ushers in the febrile paroxysm or ague fit of malaria is intense and prolonged, and the congestive chill of the algid variety of pernicious estivo-autumnal malaria may terminate in death. Chills occurring later in the attack may mark the development of an intercurrent disease, as croupous pneumonia in the course of enteric fever. The chills of malignant endocarditis cannot be distinguished from the ague paroxysm, the resemblance to which is frequently heightened by a regular periodicity. Ague-like chills occur in some cases of phthisis and are common in local suppurations with pent-up pus, cholelithiasis, and septic and other conditions attended by fever of intermittent type.

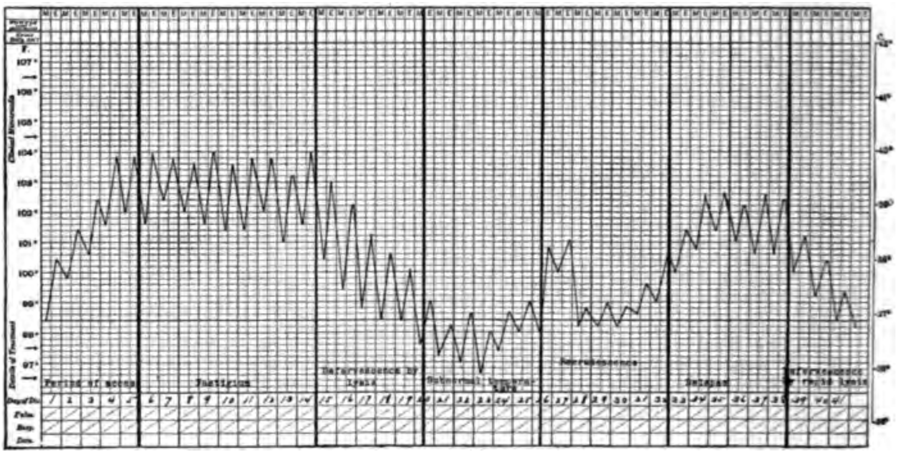


Fig. 187.—Temperature curve in enteric fever, with recrudescence and relapse.

The perturbation of the nervous system, which is manifested in the adult as a chill, may show itself in the child as a convulsion, sudden stupor, or very rarely as an outbreak of delirium. In the adult the onset of an acute febrile infection may be marked by sudden maniacal delirium and patients developing pneumonia or enteric fever have in some instances been regarded as insane and committed to an asylum.

The chill which attends the general or local infections and which is of varying intensity must be distinguished from the so-called nervous chill which sometimes occurs in persons of neurotic constitution under conditions of excitement, intense pain, moderate shock, or great fatigue. Under such circumstances there may be trembling and agitation, but the pulse remains good, the normal color is preserved, and the thermometer does not show a rise in temperature.

(c) **The Fastigium or Acme.**—Fastigium is literally the summit or ridge of a building. The temperature range in the continued fevers shows diurnal remissions and exacerbations corresponding to those of health, but somewhat greater. The elevation above the normal differs in different iseases and in different cases of the same disease. In croupous pneumonia nd in typhus and relapsing fevers the elevation is high. In many cases of nteric fever it is moderate. A parallelism with the temperature of health s to some extent maintained in the continued fevers. This parallelism nay, however, be interrupted by accidents, as hemorrhage or perforation n enteric fever, complications, as empyema in pneumonia, the occurrence f pseudocrises, the action of antipyretic drugs, or the external applicaion of cold by means of baths or otherwise. In the periodical fevers the

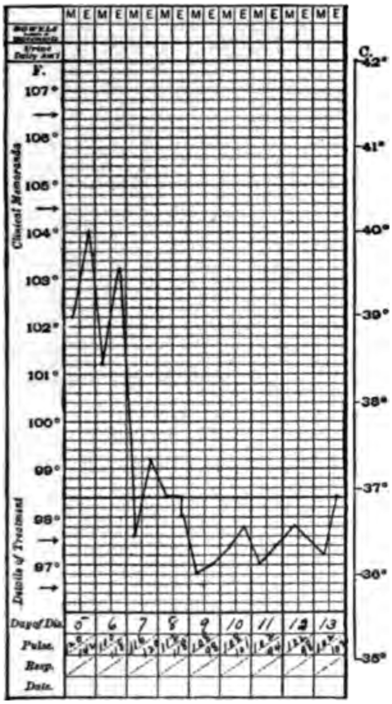


FIG. 188.—Croupous pneumonia in a child three ears old. Defervescence by crisis on the evening f the sixth day.

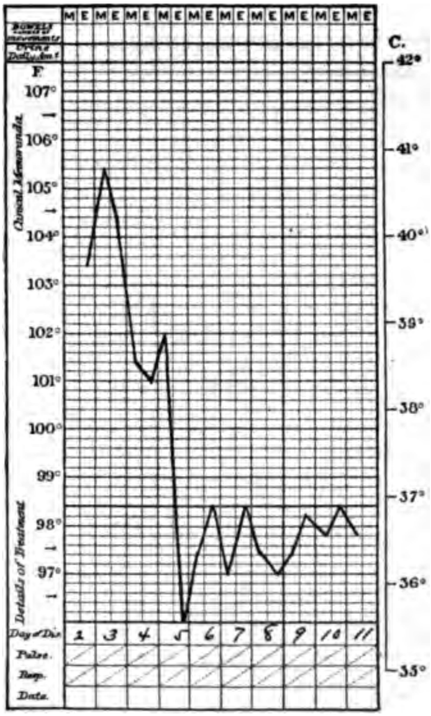


FIG. 189.—Pneumonia. Early defervescence; interrupted crisis.

iurnal range is much greater than in health. The term acme is used to ndicate the summit of the range when the febrile movement is transient, s in influenza or the ague paroxysm.

An abundant hemorrhage from any surface, especially intestinal hemrrhage in enteric fever, causes an abrupt fall of the temperature to normal r below it. The shock of perforation is likewise accompanied by a fall of everal degrees.

A normal temperature in the course of fever may thus assume the gnificance of an abnormal temperature.

In abortive cases of enteric fever, especially in children, the deferescence is often critical. Intercurrent diseases and complications may ause a rise above the range of the fastigium.

(d) **The Defervescence or Stage of Decline.**—The fall of temperature may be abrupt, or gradual. The former is known as *crisis* or *critical defervescence*, the latter as *lysis*. The abrupt fall in crisis amounts to several degrees in the course of a few hours. The temperature usually reaches subnormal ranges from which it reacts gradually. The fall may be broken by a slight rise—*interrupted crisis.* It is often attended by critical discharges, such as copious perspiration, passage of a large quantity of urine, or large liquid stools. Not infrequently it occurs during, or is followed by, a deep and prolonged sleep from which the patient awakes refreshed but weak and exhausted. There is a corresponding fall in the pulse and respiration frequency. The gradual fall in lysis takes place by progressive increase in the morning remissions and decrease in the evening exacerbations until normal or subnormal ranges are attained. This process frequently extends over several days, as in enteric fever. The term *rapid lysis* is applied to a gradual defervescence of shorter duration. Febrile diseases of sudden onset, such as croupous pneumonia, for instance, not infrequently terminate by crisis, while those of gradual invasion commonly terminate in lysis.

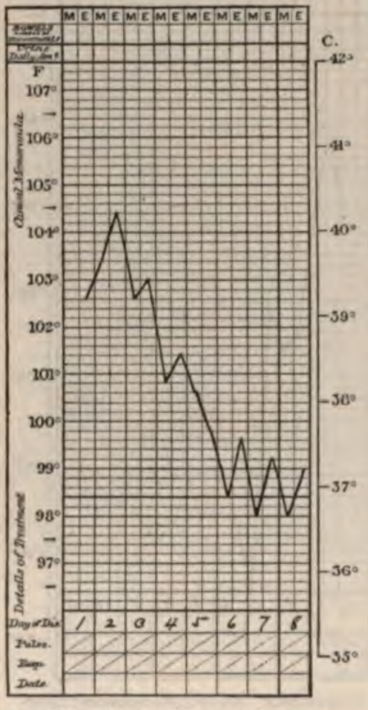


FIG. 190.—Scarlet fever: defervescence by lysis. Boy, aged twelve.

Persistence of fever beyond the normal period in a self-limited disease is due usually to a complication; sometimes to relapse. The febrile course of measles is frequently prolonged by bronchopneumonia; of scarlet fever by middle-ear disease, endo- or pericarditis, pleurisy, or nephritis; of enteric fever by phlebitis, abscess formation, cholecystitis, necrosis of cartilage or bone, some form of secondary infection, or by relapse. Cases of enteric fever extending to the fifth week or longer, in which no complication can be discovered, are mostly instances of intercurrent relapse.

The Temperature during Convalescence.—In the early days of convalescence from acute febrile disease the temperature range is frequently subnormal. It is also *labile,* that is to say, very readily disturbed by trifling influences, such as constipation, the return to solid food, mental excitement, or over-exertion. A transient rise of temperature produced by any of these causes is known as a *recrudescence.*

Relapse.—A recurrence of fever, together with the characteristic symptoms of the primary attack, due to reinfection. Instances of two or more relapses—*multiple relapse*—are of occasional occurrence. That form of relapse which begins before the defervescence from the primary attack is completed is known as *intercurrent relapse.*

Bed Fever.—Patients who have passed through febrile diseases sometimes develop during convalescence a moderate febrile movement, the evening exacerbations ranging as high as 100° or 101° F. This fever tends to run on indefinitely but may quickly disappear if the patient is allowed to sit up. A diagnosis of bed fever should never be made until other fever-producing conditions are excluded.

Paroxysmal Fever.—The fever recurs at intervals. The temperature is high and the accompanying symptoms usually severe. The febrile movement is of short duration and commonly preceded by a chill and followed by profuse sweating.

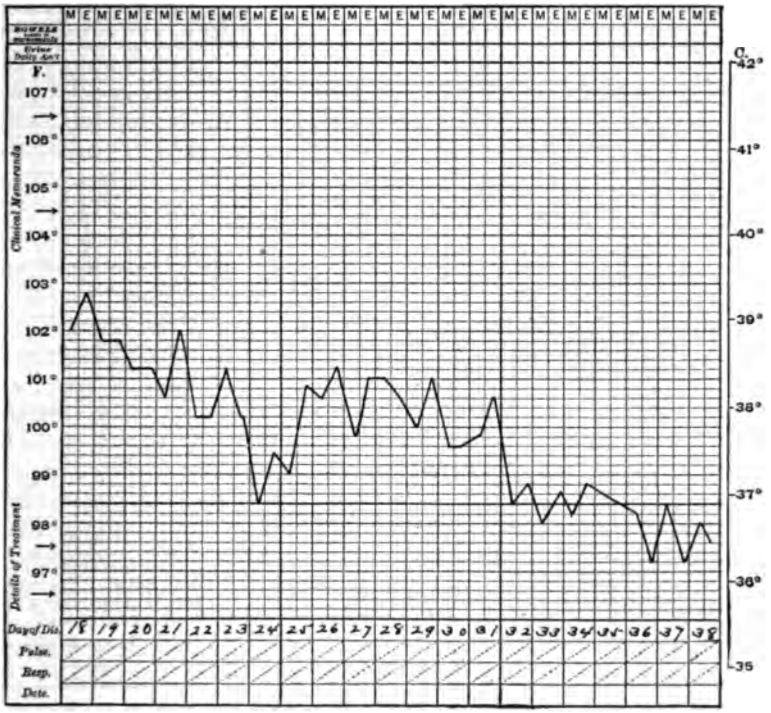


Fig. 191.—Interrupted lysis.

Periodical Fever.—The periodicity may be regular or irregular. Intermittent and remittent fevers are periodical. Tertian and quartan malaria constitute the very type of the regularly recurring periodical fevers. The periodicity of the estivo-autumnal fevers is not so well defined; the type is blurred and in some cases wholly obliterated. We observe forms of continued malarial fever due to estivo-autumnal infection.

Other febrile diseases are characterized by periodicity—a matter of great practical importance in diagnosis.

Paroxysmal fever, often of regular periodicity, may occur in the following conditions: (a) Abscess formation and other suppurative processes, as empyema. In cerebral abscess the temperature may be continuously normal or subnormal. Evacuation of pus and free drainage is followed by disappearance of fever. (b) Pyæmia and septicæmia. (c) Malignant endocarditis. (d) Suppurative and infectious processes in the liver and bile-passages—*hepatic fever.* Under this heading are abscess of the liver, diffuse cholangitis, cholecystitis, inflammation of the hepatic, cystic, and common ducts, gall-stone disease, especially impacted gall-stones, and hypertrophic cirrhosis. (e) Infections of the genito-urinary tract, as cystitis and pyelitis, prostatic abscess, and after the passage of the catheter or sound—*catheter fever, urinary fever.* (f) Tuberculosis. Paroxysmal fever is present in the acute miliary form, the early stages of many cases and the later stages—after cavity formation—of almost all cases of pulmonary phthisis, and very often in acute tuberculous processes involving the bones, joints, and glands. Sometimes in the last few days of consumption the fever wholly ceases. (g) Hodgkin's disease and leukæmia. Periods of fever may be separated by prolonged periods of apyrexia. (h) Syphilis. The initial fever may come on within four or six weeks after infection and persist for several weeks. Paroxysmal fever in some instances accompanies the development of gummata and other lesions of the tertiary period. (i) Rapidly growing malignant neoplasms. (j) Very rarely in morphinism, the febrile paroxysm being preceded by a chill and followed by copious sweating. If the minimal temperatures fall to or below the normal the fever is intermittent in type; if they fail to reach the normal

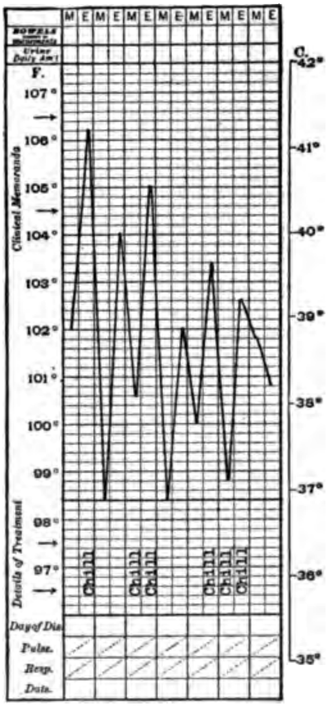


FIG. 192.—Urethral fever. Man, aged sixty-four.

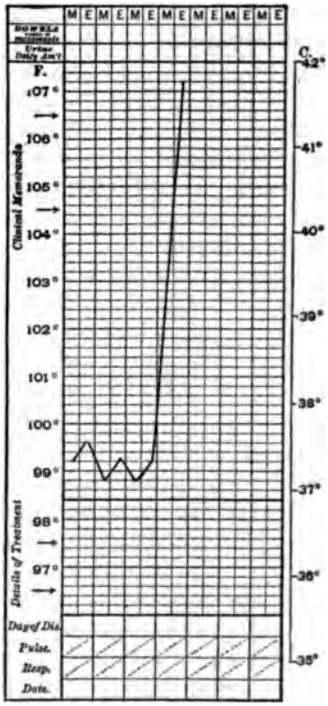


FIG. 193.—Cerebral hemorrhage. Hyperpyrexia. Pre-agonistic rise of temperature.

it is of remittent type. In the course of any of the foregoing diseases the fever may change from one to the other of these types and frequently it becomes irregular and wholly atypical. In the course of defervescence by lysis as the fever gradually falls to normal it passes from the subcontinuous type of the fastigium first to the remittent type, then to intermittent.

The febrile paroxysms, in some cases of pyæmia, malignant endocarditis, and tuberculosis, and of disease of the liver and bile-passages, are ushered in by rigor and terminate by sweating, and recur with such regularity that they closely resemble the ague paroxysms of malaria. Errors of diagnosis are common, but readily avoided by close observation, examination of the blood, and the therapeutic test of quinine.

Hyperpyrexia.—Excessively high temperatures are occasionally observed. The thermometer may register 105.8° F. (41° C.) and higher in injuries involving the cervical portion of the spinal cord, and in tetanus, rheumatic fever, scarlet fever, enteric fever, yellow fever, and sunstroke. Very high temperatures occur in croupous pneumonia, the paroxysms of malarial fever, relapsing fever, and erysipelas. A marked rise may occur in the acute infections just before death—*pre-agonistic rise*. Excessive temperature when transient is not necessarily of grave prognostic import; if continued for some hours it is apt to be followed by death. Da Costa has recorded a temperature of 110° F. (43.3° C.) in a case of cerebral rheumatism, Jacobi has seen in scarlet fever 107.6° F. (42° C.), Sahli 113° F. (45° C.) in enteric fever, Richet 107° F. (41.7° C.) in sunstroke, with recovery. The literature contains many instances of recovery after such temperatures. There are well authenticated cases of even higher temperatures with recovery. The most remarkable is that of Teale, reported to the London Clinical Society in 1875. A lady fell from her horse and sustained serious spinal injuries. For sixty days she had frequent rises of temperature to 111.2° F. (44° C.) and higher but eventually recovered. Bryant, Guy's Hospital Reports, 1894, has recorded the facts of one hundred cases of hyperpyrexia, several of which, however, are not above suspicion. Many of the cases of excessively high temperature have occurred in hysterical persons and several of the most remarkable instances on record are obviously the result of deception.

HYPOTHERMIA.

Subnormal Temperature.—Hypothermia may be present under the following conditions:

(a) The intense action of external cold. A transient body temperature of 86° F. (30° C.) may occur, yet recovery take place.

(b) After a pronounced crisis at the close of an acute infectious disease, as pneumonia. Postcritical falls to 95° F. (35° C.) or even to 93.2° F. (34° C.) have been observed.

(c) In shock and collapse. The fall of temperature is associated with signs of failure of the circulation, frequent, small, or imperceptible pulse, colliquative sweating, great relaxation, and extreme pallor. The mind, except in the presence of cerebral lesions, usually remains clear. The condition may be transient or it may be the immediate forerunner of death.

Subnormal temperature may be the result of internal or external hemorrhage, traumatism, surgical operation, prolonged anæsthesia, the apoplectic insult in cerebral hemorrhage, embolism or thrombosis, the sudden rupture of a hollow viscus with the discharge of its contents into the peritoneum, or finally the action of intense pain or a sudden, overwhelming, depressing emotion in a neurotic individual. When reaction takes place the temperature rises very often to febrile ranges, either as the result of infection or, in the case of cerebral or spinal lesions, from irritation of the tissues which constitute the heat centres. In lesions of the cerebrospinal axis the reactive fever is frequently due to inflammatory reaction in the neighborhood of the lesion.

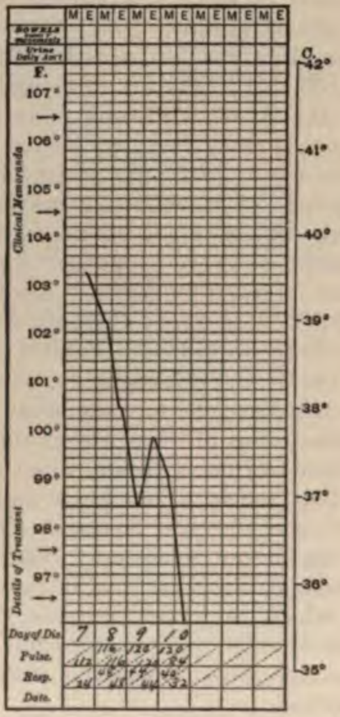


FIG. 194.—Pneumonia. Death in collapse after crisis.

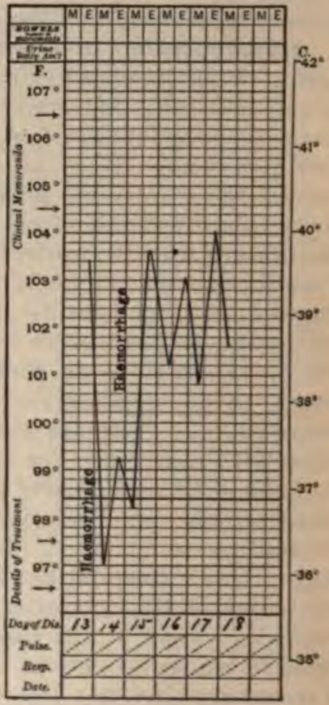


FIG. 195.—Enteric fever; subnormal temperature due to repeated hemorrhage.

(d) In various conditions attended by greatly diminished tissue change or profound disturbance of the heat mechanism, as in the coma attending acute poisoning from alcohol, illuminating gas, carbolic acid, and other toxic agents, starvation, carcinoma of the œsophagus, other internal cancers, abscess of the brain, myxœdema, sclerema neonatorum, and in some forms of mental disease, as melancholia. Subnormal temperature ranges are also occasionally observed in profound anæmia, the terminal stages of tuberculous processes, especially tuberculous peritonitis, and in diabetes.

The Action of Drugs upon the Temperature.—Antipyretic drugs, most of which are synthetic products of coal-tar, while capable of producing marked effects upon febrile temperatures, have little influence upon the body temperature in health. Their free use in fever is followed by a tendency to collapse, and the resulting fall of temperature is of short duration.

External Antipyretics.—Cold baths or gradually cooled baths, sponging, packs, ice-bags, circulating coils for the application of iced water, and cold enemata reduce the febrile temperature not only without the perturbating effects of drugs but, if rightly employed, with a favorable influence upon the general condition of the patient.

Trifling rises of temperature follow the administration of full doses of atropine, cocaine, strychnine, caffeine, and certain other drugs, while correspondingly slight falls occur after morphine, quinine, alcohol, and the general anæsthetics.

THE SIGNIFICANCE OF ABNORMAL TEMPERATURES.

To recapitulate: A rise of temperature, if moderate, may be physiological—digestion, violent muscular effort. Such rises are commonly transient. If the rise be accompanied by other symptoms of fever it may indicate (a) an infection, either general or local; (b) an intoxication, which may arise within the body from faulty metabolism or be introduced from outside the body, as in the case of food or drink; (c) a lesion involving the heat-regulating mechanism of the nervous system.

As a rule there are associated symptoms which render practicable the differential diagnosis of these conditions.

A fall of temperature may indicate blood loss, which may be internal and concealed, as in a small rupture of the wall of the heart not presently fatal; a similar leakage from an aneurism; collapse, as in apoplexy; excessive radiation, as in exposure; diminished metabolism, as in convalescence, starvation, forms of poisoning, and certain nutritional and nervous diseases. The fall may be transient or sustained.

Whether the temperature be higher or lower than the normal it serves to exclude malingering and, as a rule, hysteria. It is important to bear in mind that remarkable departures from the normal temperature are observed in some cases of hysteria, and that the clever malingerer often plays tricks with the thermometer that are as difficult of detection as they are puzzling.

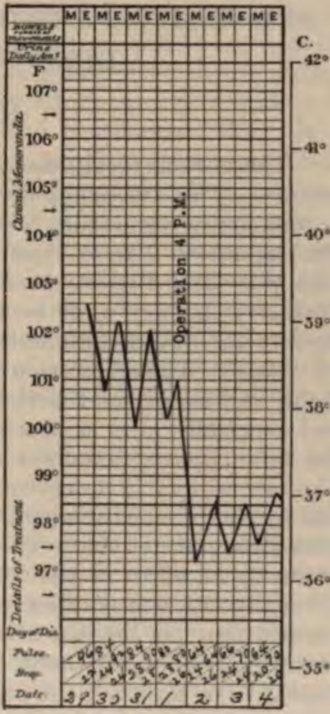


FIG. 196.—Ischiorectal abscess. Fever immediately relieved by incision and drainage.

The Prognostic Significance of Abnormal Temperature.—The height of the temperature is important, since the danger increases with the intensity of the fever. A rise of 7.2° F. (4° C.) or 9° F. (5° C.) is of itself ominous. If sustained for some hours death almost always follows, though remarkable exceptions to this rule have been observed. Abrupt rises from the range of health, such as are seen in malaria or relapsing fever, are less dangerous than sudden rises above the fastigium in the course of enteric or other continued fever.

Variations from type in the temperature curve modify the prognosis unfavorably. The irregular pneumonias of moderate temperature, 102° F. (38.8° C.) are attended by greater danger to life than the typical forms with intense fever, 104° F. (40° C.) or higher.

The temperature in childhood is less stable than in adult life. It is elevated by slight causes, and reacts more readily to antipyretic treatment. Forms of ephemeral fever are more common than in later life. High temperatures are less dangerous.

V.

RESPIRATION; MODIFIED RESPIRATORY MOVEMENTS; COUGH AND ALLIED PHENOMENA; SIGNIFICANCE OF COUGH IN DIAGNOSIS; EXPECTORATION.

RESPIRATION.

The normal respiration-frequency in the adult is from 16 to 24 in the minute. The average pulse-frequency varies between 64 and 96. The normal pulse-respiration frequency is therefore 1 to 4–4.5. In early life the respiration is more rapid, the average being in the new-born 44; in the fifth year 26. Posture exerts a marked influence. In normal adults the average frequency while recumbent is 14, while sitting 20, and in the erect posture 22. These differences are exaggerated in those who are enfeebled by disease. The respiration is slightly less frequent in the morning than at night, and about one-fourth less during sleep. It is more rapid after eating and especially after a hearty meal, a fact which finds its explanation in the more limited excursus of the diaphragm when the stomach is full. The rate is very little influenced by the external temperature. It is modified by the internal temperature and much increased in fever. It increases with muscular activity. The respiration frequency may be modified within limits by an effort of the will and is profoundly affected by the emotions. Clinically the rate is often quickened by the knowledge on the part of the patient that his breaths are being counted. It is therefore important, if possible, to count without his being aware of it, for instance, when you appear to be counting the pulse. Failures of observation may be controlled by counting for one or more entire minutes. The rate and depth of the respirations bear an inverse relation to each other: the greater the frequency the less the depth, or the slower they are, the deeper. General abnormal conditions and local diseases, especially those which involve the organs of respiration, modify both the frequency and extent of the respiratory movements.

Tidal Air.—This term is used to designate the inflow and outflow of air during quiet respiration. It amounts to about 500 cubic centimetres—30 cubic inches. COMPLEMENTAL AIR is the volume that can be inspired after the completion of an ordinary inspiration; RESERVE OR SUPPLEMENTAL AIR, the volume that can be expelled after an ordinary expiration; RESIDUAL AIR, the volume remaining in the lungs after the most forcible expiration; STATIONARY AIR, the volume remaining in the lungs after an ordinary expiration and equal to the reserve air plus the residual air. VITAL CAPACITY is the volume of air that can be expired after the fullest inspiration. The average is about 3400 cubic centimetres for men and 2500 cubic centimetres for women. LUNG CAPACITY is the total quantity of air in the lungs after full inspiration, and is equal to the vital capacity plus the residual air.

Vital Capacity.—The measurement of the vital capacity is determined by various modifications of the spirometer devised by Hutchinson. It is affected by age, sex, stature, posture, occupation, and disease. It increases with age, the maximum being attained at about the thirty-fifth year. It is greater in men than in women of the same height in the ratio of 10 to 7.5. It increases in proportion to the stature up to the twenty-fifth year, and Arnold found that, in the adult, for each centimetre of increase or decrease of height beyond a mean standard there is a corresponding rise or fall in the vital capacity of 60 cubic centimetres for men and 40 for women. The ratio as modified by posture is 0.96 in the prone, 1.11 in the sitting or erect, and 1.13 in the standing position. The vital capacity is greater in those leading an active than in those who lead a sedentary life. It is obvious that improper clothing and tight lacing and all pathological conditions which interfere with the full and free expansion of the chest, whether general, as in wasting diseases of every kind, or local, as in thoracic or abdominal diseases, must diminish the vital capacity; nor is it to be overlooked that pregnancy or a sedentary life not in itself incompatible with excellent health may exert a similar influence. The spirometer, partly for these reasons, partly by reason of its inconvenience of application and uncertainty as an instrument of precision, and, finally, because there are other methods far more available and accurate, has fallen wholly into disuse in ordinary clinical work.

Peculiarities in the respiratory phenomena are expressed by the following terms:

EUPNŒA is a condition of normal respiration observed during bodily and mental quiet. APNŒA is a temporary suspension of the respiratory movements. HYPERPNŒA is a term used to designate increased respiratory activity. HEAT-DYSPNŒA and POLYPNŒA are forms of hyperpnœa due to direct excitation of the respiratory centres, as the result of an increase in the temperature of the blood or of reflex excitation of the cutaneous nerves by external heat. DYSPNŒA is difficult or labored breathing; the respiratory frequency is often less than normal but may be increased. ASPHYXIA OR SUFFOCATION is the term used to express the condition caused by deprivation of air. The respirations are at first increased in frequency and depth, then a period of increasing dyspnœa follows, with violent spasmodic expirations and convulsions. The final condition is that of collapse, which is ushered in by progressive slowness and shallowness of the respiistration of this drug. The prognosis of the underlying condition is always grave, and this form of respiration is seen in profound illness from which, exceptionally, the patient rallies in a short time or, and this is the general rule, presently dies. In some cases of cardiac or renal disease, however, Cheyne-Stokes respiration recurs from time to time for months.

3. JERKING RESPIRATION.—The act may be spasmodic. Usually it is the inspiration that is jerking, less commonly the expiration, rarely both. Jerking inspiration is seen in sobbing, hysteria, hydrophobia, sometimes in asthma; jerking expiration, in acutely painful respiration, such as occurs in pleurisy, especially diaphragmatic, in pleurodynia, or in the case of a broken rib. Jerking respiration is more apt to occur when the breathing is of the costal type than when it is abdominal.

Dyspnœa.—This term includes a variety of respiratory derangements which, much as they may differ among themselves in detail, have one thing in common, namely, difficulty in breathing. The clinical conception, whatever the cause or whatever the derangement, rests upon inadequate oxygenation of the blood. Dyspnœa may arise from a deficiency of oxygen or from an excess of carbon dioxide in the blood. Cardiac and hemorrhagic dyspnœas are due chiefly to a deficient supply of oxygen. In cardiac dyspnœa the poor supply of blood to the tissues results from the enfeebled action of the heart. In hemorrhagic dyspnœa there is enfeebled action of the heart on the one hand and a diminished quantity of blood on the other. All conditions which lessen the force of the circulation or the quantity of hæmoglobin tend to cause dyspnœa; hence individuals who suffer from cardiac lesions or forms of anæmia, or who are enfeebled by disease, experience difficulty in respiration after slight exertion. Conditions which interfere with the interchange of oxygen and carbon dioxide in the lungs, such as bronchitis of the smaller tubes, forms of pneumonia, emphysema, extensive pulmonary tuberculosis, laryngeal obstruction, abdominal tumors, and large ascites, tend to the production of dyspnœa, especially upon exertion.

The respiration may be more or less frequent than normal. Two principal forms may be distinguished, namely, that in which the breathing is rapid and shallow and that in which it is slow and deep. In the former the ratio between inspiration and expiration is not usually much deranged; difficulty in breathing attends both acts and the condition is spoken of as *mixed dyspnœa.* In the latter the ratio is disordered, sometimes the inspiration, sometimes the expiration being more prolonged. To the one is applied the term *inspiratory dyspnœa;* to the other *expiratory dyspnœa.* The first is characterized by inspiratory, the second by expiratory stridor. In all forms of dyspnœa certain muscles which ordinarily are little or not at all used in respiration and have other functions are brought into play. They are the auxiliary muscles of respiration. Among them are the scaleni, trapezii, levatores scapulæ, the sternocleidomastoid, sterno- and thyrohyoid muscles, and the pectorales. The action of these muscles is more powerfully exerted in the erect or sitting posture—*orthopnœa*—the position usually assumed in inspiratory dyspnœa. In expiratory dyspnœa the abdominal muscles are used as auxiliary muscles.

In dyspnœa of high grade the muscles of facial expression are also brought into play, with the effect of dilating the nostrils and separating the lips and jaws. The action of these muscles gives to the facies a very characteristic and distressed appearance. Very striking is the play of the nostrils in young children suffering with pneumonia. The entrance of air is to some extent favored by the action of these muscles, but the explanation of their participation in the dyspnœal movements is to be sought in the implication of associated muscle groups in the intense innervation supplied to the essential muscles of respiration.

The term dyspnœa is sometimes used to designate the sensation of breathlessness which attends difficult breathing. Hence *subjective* and *objective dyspnœa.* These two forms are usually associated, but to this statement there are exceptions. Cases occur in which, with persistent obstruction of the respiration and well-marked objective dyspnœa, there is no sense of breathlessness or oppression. Cyanosis may even be present without subjective dyspnœa. As death approaches and objective dyspnœa becomes urgent carbon-dioxide narcosis develops and subjective dyspnœa disappears. There are cases, however, in which objective dyspnœa is slight or absent altogether, yet the patient complains of distressing subjective dyspnœa. To this category must be referred the precordial distress of melancholia and the frequent desire of nervous patients to take a series of deep inspirations. Pure subjective dyspnœa is rare.

Cyanosis attends every obstruction to respiration of high grade, whatever the cause. The blood becomes progressively richer in carbon dioxide and poorer in oxygen. In chronic conditions attended with dyspnœa the organism may gradually become adjusted to subnormal oxygenation of the blood, so that the other functions are fairly well performed and the patient, although manifestly dyspnœic, especially upon exertion, and constantly cyanosed, has little subjective dyspnœa. On the other hand a similar degree of obstruction to respiration, if rapidly established, is attended with the most urgent and distressing dyspnœa.

Pneumothorax affords a striking example of the adjustment of the organism to respiratory disturbances of high grade. The sudden interference with respiration causes intense objective and subjective dyspnœa, which gradually subsides and in many cases wholly disappears so long as the patient is at rest.

Dyspnœa is of much less unfavorable prognosis when the cyanosis is slight than when it is deep and persistent.

Forms of Dyspnœa.—DYSPNŒA AS THE RESULT OF PAIN occurs in pleurisy and especially in diaphragmatic pleurisy, peritonitis, inflammation of the diaphragm, and in affections of the intercostal muscles, as myalgia and trichinosis. Deep breathing is impossible; the respirations are shallow and hurried. The difficulty is not mechanical but functional.

DYSPNŒA FROM DIMINUTION OF THE RESPIRATORY SURFACE OR LIMITATION OF THE RESPIRATORY EXCURSUS.—These conditions are commonly associated. They are present in diseases involving the parenchyma of the lungs, such as croupous and bronchopneumonia, large infarcts, congestion, and the like; also in those affections in which the capacity of the thorax is decreased, as pleural and pericardial effusion, pneumothorax, tumors, massive hypertrophy of the heart, and kyphoscoliosis; and finally when ever the movements of the chest are hindered, as in emphysema, severe istration of this drug. The prognosis of the underlying condition is always grave, and this form of respiration is seen in profound illness from which, exceptionally, the patient rallies in a short time or, and this is the general rule, presently dies. In some cases of cardiac or renal disease, however, Cheyne-Stokes respiration recurs from time to time for months.

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Dyspnœa in Circulatory Derangements.—Valvular lesions cause dyspnœa when the compensation fails. Myocardial changes act in the same way. There is a transference of blood-pressure from the arterial to the venous side of the circulation. The organs receive less arterial and retain more venous blood than normal. The circulatory derangement affects the respiratory centre, with the result that the breathing is increased both in frequency and depth. Lesions of the left side of the heart cause overfilling not only of the veins of the general circulation but also of those of the pulmonary circuit. The overfilling of the pulmonary capillaries, which ultimately gives rise to brown induration, is an additional cause of dyspnœa, not so much because of the space occupied by the blood as by reason of the impairment of elasticity in the congested tissue of the lung. The alveolar distention remains near the inspiration point and the respiratory excursus is correspondingly diminished. The loss of elasticity acts as a direct hindrance to breathing. The paroxysmal attacks of dyspnœa in such cases constitute so-called cardiac asthma. This term is frequently used to describe any shortness of breath occurring in disease of the heart. It is preferable to restrict it to the attacks which bear a close resemblance to true bronchial asthma. Such attacks often come on at night after the first sleep. In both conditions the form of dyspnœa is the same. There is a tendency to prolongation of the respiratory act with difficult and prolonged expiration attended by stridor. To the habitual overfilling of the pulmonary capillaries in mitral disease must be ascribed the dyspnœa upon exertion which is so common in this condition in the absence of impaired compensation. A further cause of dyspnœa in circulatory disturbances is the bronchial catarrh which is present to some degree in most of the cases.

Dyspnœa in Obstruction of the Upper Air-passages.—The stress upon the inspiratory muscles is proportionate to the degree of obstruction. The respiration tends to become prolonged and deep. In many cases, however, it is increased in frequency and correspondingly superficial. This form of dyspnœa is present in the marked stenosis of the pharynx which occurs as the result of hypertrophy of the tonsils or retropharyngeal abscess, in spasmodic and membranous laryngitis, in œdema or spasm of the glottis, in paresis of the abductor muscles of the larynx (posterior crico-arytenoids) and in narrowing of the pharynx and trachea by tumors, foreign bodies, and compression from outside, as in the case of aneurism or mediastinal tumor.

As the obstruction reaches a high grade the volume of air in the lungs is progressively diminished and the less rigid portions of the thorax yield to the pressure of the external atmosphere. The depressions are especially marked in the epigastrium and the suprasternal and postclavicular regions.

In young children, in consequence of repeated attacks of laryngitis or bronchitis the cartilaginous portions of the wall of the thorax yield and more or less persistent deformities of the chest arise. Among these are the wide, shallow, oblique depressions at the base of the chest known as Harrison's furrows, and the prominence of the sternum, known as chicken-breast.

The stridor in this form of dyspnœa is characteristic. It is commonly loud, prolonged, and hissing or whistling in character and usually much more marked upon inspiration than upon expiration,—a fact that finds explanation in the lateral drawing together of the tissues below the seat of obstruction in consequence of the tendency to vacuum caused by the powerful inspiratory effort and the greater force of the inspiration as compared with the expiration. In fact stridor may be wholly absent during the expiration. When, however, patients suffering from stenosis of the upper air-passages in increasing obstruction become obliged to use the abdominal muscles in active expiration, expiratory stridor becomes marked or predominant.

Dyspnœa in Bronchitis.—Dyspnœa arises, as a rule, only in those cases in which the catarrhal inflammation involves the finer tubes. The lumen is narrowed by swelling of the mucosa and the presence of secretion or exudate. If the narrowing involves a limited number of bronchial tubes the difficulty in breathing is not urgent and compensation takes place by increase in the respiration frequency. When the obstruction involves a great number of bronchial tubes differences in type of the dyspnœa arise which depend upon the degree of obstruction. In so-called capillary bronchitis the respiratory surface is diminished to an extent corresponding with the number and distribution of the lobules involved; dyspnœa with hurried respiration then results. In the dry bronchitis of the middle-sized tubes it is yet possible for a sufficient quantity of air to be drawn into the lungs. This can generally be accomplished best by respiration of diminished frequency and abnormal depth, just as in stenosis of the larynx.

Dyspnœa in Bronchial Asthma.—The breathing is slow, the expiration usually prolonged and accompanied with stridor—*expiratory dyspnœa.* The râles can be heard at a considerable distance from the patient. The difficulty is not to get the air into the lungs but to get it out. Acute emphysema occurs and the respiratory excursus is greatly diminished; hence the "air hunger."

Dyspnœa in Emphysema.—The chest tends to assume permanently the inspiratory form. The elasticity of the lung parenchyma is impaired. The respiratory excursus is correspondingly diminished. The alveolar septa are in many places destroyed, together with the intra-alveolar blood-vessels. The breathing is shallow and frequent. The patient is distressed for breath, especially upon the slightest exertion. The dyspnœa is increased by the bronchitis which is so common in emphysema. Modifications arise in consequence of the frequent occurrence of bronchial asthma in emphysema.

So-called Uræmic Dyspnœa.—This form may occur as a true uræmic bronchial asthma. There is slowing of the respiration with prolonged expiration and expiratory stridor. The condition is not common. The dyspnœa in the majority of the cases is not actually uræmic but rather a manifestation of cardiac derangement, bronchial catarrh, or beginning pulmonary œdema.

The Dyspnœa of Fever.—Rise of temperature is usually associated with increase in respiration frequency. Artificial elevation of temperature also causes hurried breathing. The dyspnœa is doubtless due to the action of heated blood upon the respiratory centre. As it bears no constant relation to the height to which the temperature rises, it is probably due, in part at least, to the action of the fever-producing toxins. Experience has shown that febrile diseases in which the respiration frequency, in the absence of lung complications, is very high, are as a rule of serious import.

The Dyspnœa of Anæmia.—When the hæmoglobin is diminished, the oxygen requirement of the organism demands the most complete performance of the respiratory function. There is no hindrance to respiration and the breathing is quickened and increased in depth. This form of dyspnœa is characteristic of high grades of secondary anæmia such as occur in hemorrhage, in advanced pernicious anæmia, and in chlorosis, following exertion. It is a symptom of internal hemorrhage and occasionally of hemorrhagic pancreatitis.

COUGH AND ALLIED PHENOMENA.

The normal rhythmical expansions and contractions of the thorax serve the physiological purposes of respiration. Certain other movements which are respiratory in character serve other purposes. Of these some are voluntary, others involuntary, some purposeful, others spasmodic. Among such movements are the following:

Cough.—A more or less deep inspiration is followed by an expiratory act which is interrupted by repeated partial closure of the glottis and the production of a series of characteristic sounds. The air is expelled through the narrowed glottis with some force so that foreign bodies, such as a crumb, mucus in the respiratory passages, and the like, are swept from the upper air-passages into the mouth. In the great majority of cases cough, whether in consequence of lesions of the respiratory organs or disease or irritation in distant organs, is reflex and spasmodic. It may be voluntarily produced.

Hawking is a voluntary act, the result of irritation in the pharynx. It resembles cough except that the glottis is open and the expiration continuous.

Sneezing consists in a deep inspiration followed by a forcible expiratory blast through the nose; the glottis is open and the mouth usually but not always closed. Sneezing is excited by irritation of the terminal fibres of the nasal branches of the fifth pair of cranial nerves and is often preceded by peculiar sensations in the nose.

Laughing is an emotional act characterized by deep inspiration succeeded by repeatedly interrupted expiration with an open glottis and vibrating vocal cords. The mouth is wide open, the expiration is much less forcible than in coughing, and the muscles of expression give a characteristic appearance to the face. Laughing may be voluntary or involuntary. When very violent and repeated it may be spasmodic and accompanied by tears.

Crying closely resembles laughing. It cannot in fact always be distinguished from laughing and the one may readily alternate with the other in young children or patients suffering from hysteria. The rhythm and the facies are different. Crying is involuntary and accompanied by tears.

Sobbing follows long spells of crying or is the expression of deep grief. It is characterized by interrupted inspirations with a partially closed glottis, followed by a prolonged quiet expiration, and is usually involuntary.

Sighing consists in a prolonged inspiration attended by a characteristic soft sound. The mouth is closed or the lips but slightly parted; it is largely voluntary.

Yawning consists in a prolonged deep inspiration through the widely opened mouth accompanied by a peculiar sound. The glottis is open and the expiration short. The arms are thrown out and the shoulders back. It may be either voluntary or involuntary but is not spasmodic.

Snoring occurs during sleep. The mouth is open and the relaxed palate is thrown into vibration by the in- and outflowing air. The sound is louder during inspiration. It is much more liable to occur when the sleeper is on his back.

Stertor or stertorous breathing resembles snoring. It occurs in apoplectic and other comatose states, as cerebral concussion, fracture of the skull, epilepsy, deep anæsthesia, alcoholic stupor, poisoning by opium, illuminating gas, and other narcotics, pulmonary œdema, and all conditions in which excessive amounts of mucus or fluid are accumulated in the bronchi, and frequently in the death-agony. Among the varieties of stertor are *buccal,* characterized by vibrations of the lips and puffing of the relaxed cheeks during expiration; *palatine,* in which the soft palate vibrates with the in-going and out-going air; *pharyngeal,* caused by the sinking back of the base of the relaxed tongue into near relation with the posterior wall of the pharynx; *mucous,* the coarse snoring sound produced by the churning of the respiratory air through fluid, such as mucus or blood in the trachea or larger bronchial tubes.

Stridor or stridulous breathing is that noisy form of breathing caused by obstruction in the larynx or trachea. This symptom may be present in croup and diphtheria, œdema of the glottis, laryngeal tumors, mediastinal new growths, and aortic aneurism. Stridor due to laryngeal obstruction is commonly accompanied by aphonia. It varies greatly in character, being harsh, musical, or crowing.

Hiccough is caused by a sudden spasmodic contraction of the diaphragm accompanied by closure of the glottis. There is a peculiar abrupt sound and a distressing sensation of jerking in the epigastrium. It is due to irritation of the terminal filaments of the phrenic nerve, which may be direct or reflex. It may occur as the result of gastric or peritoneal irritation or may be the manifestation of a derangement of the nervous system. Hiccough is occasionally observed after excessive or injudicious eating or drinking, in gastric disorders, peritonitis, the so-called typhoid state, and uræmia. It occurs also in hysteria and may constitute a pure neurosis. The hiccough of cerebral disease, as hydrocephalus or meningitis, is doubtless the result of irritation of the central origin of the phrenic nerve. The writer knows a gentleman in whom certain kinds of tobacco invariably produce distressing hiccough, while others can be smoked with impunity.

Persistent and intractable hiccough occasionally attends the closing days of fatal illness and in rare instances, occurring as a neurosis, has caused death by exhaustion.

Of all the special or modified respiratory movements cough has the most important bearing upon diagnosis and therefore requires more extended consideration.

SIGNIFICANCE OF COUGH IN DIAGNOSIS.

Etiological Considerations.—Reflex Cough.—Cough in the vast majority of instances is the result of reflex irritation of the terminal nerve filaments of the vagus distributed to the respiratory tract. Irritation of the mucous membrane of the larynx above the vocal cords does not produce cough but causes gagging, while irritation below the cords gives rise to cough. Especially sensitive areas are the interarytenoid space and the region of the bifurcation, while the general mucous membrane of the trachea and bronchi shows a scarcely inferior irritability. Lesions of the lung parenchyma probably do not cause cough, though they are usually connected with pathological conditions of the bronchi. Pleural irritation is commonly attended by this symptom. The aspiration of a pleural exudate is frequently followed by prolonged and violent cough.

Irritation of the nasal mucosa, which is supplied with sensory nerve-twigs from the trigeminus, may in neurotic individuals produce coughing with lachrymation, as in rose cold, hay fever, and similar conditions. In such persons the slightest touch of the probe in the sensitive areas may provoke violent attacks of coughing. Less common is cough as a symptom of hypertrophic, atrophic, or vasomotor rhinitis or of polypi or deviations of the septum. The inhalation of dust or smoke, irritating chemical fumes, as those of ammonia, bromine, or pungent substances, as pepper, produces cough in a normal respiratory mucous membrane. Violent paroxysmal cough is excited by the insufflation of a foreign body, as a crumb or a drop of liquid, into the larynx or through the glottis. The common source of irritation is to be found in a morbid condition of the mucous membrane of the larynx, trachea, or bronchi. There may be merely inflammation and hyperæsthesia with altered or deficient secretion; an exudate of varying consistence, from the thin fluid of bronchorrhœa to the tough masses of tenacious mucus in the early stages of acute bronchitis; or the solid bronchial casts of the terminal tubules in croupous pneumonia or fibrinous bronchitis; or, finally, the material in the bronchi may be derived from adjacent structures and consist of blood, as in bronchopulmonary hemorrhage or an aneurism, or pus from an empyema, a subphrenic abscess, or an abscess of the liver.

Cough Occurs as a Symptom in All Forms of Respiratory Catarrh.—In acute or subacute rhinitis it is often associated with sneezing; in laryngitis with hoarseness or aphonia; in tracheitis or tracheobronchitis with substernal pain; in bronchitis of the larger tubes with tickling sensations in the early stages and a mucopurulent expectoration later; in bronchitis of the smaller tubes with dyspnœa and a tendency to cyanosis; in pneumonia with fever and other indications of acute illness; in pleurisy with a stitch in the side. Cough is a prominent symptom in bronchiectasis and in all diseases, both acute and chronic, in which the respiratory mucous membrane is primarily in a morbid condition or is irritated by the presence of exudates or discharges from the alveolar tissues or other sources. The irritation is always mechanical, often also chemical. Cough is therefore a constant and suggestive symptom in pulmonary tuberculosis in all its forms and at all stages of its progress. Cough is at once the reflex response to the irritation and the effort to remove the cause of the irritation, and ceases when the effort is successful. The offending substance ejected is known as expectoration, phlegm, or sputum, or, in the plural, sputa.

Exceptionally there are cases in which, with the most pronounced symptoms and signs of disease of the lungs, cough is wholly absent. This may occur in the low fevers, the pneumonia of drunkards, the cachectic, or the aged, in cerebral disease, and shortly before death. The reflexes are obtunded and bronchial secretion or an exudate, the presence of which is manifested by râles, fails to excite cough. The sudden cessation of cough in grave cases of pneumonia or advanced phthisis is an ominous sign. Cough is sometimes absent because the bronchial secretion is swept onward by the ciliated epithelium to the larynx and removed by hawking. If it is then swallowed, as is a frequent occurrence, not only is cough absent but also expectoration.

Much less common is cough due to extrarespiratory irritation. The sufferers are usually neuropathic.

Pharyngeal Cough.—Tickling of the wall of the pharynx or the base of the tongue, which in most persons is resented by gagging, in some is followed by cough. Lymphoid growths in the nasopharynx and collections of thick mucus, or the presence of inflammatory exudates, may be the cause of cough. Elongation of the uvula and paresis of the palate may excite cough by producing irritation of the posterior wall of the pharynx, especially during recumbency.

Ear Cough.—Not infrequently paroxysmal cough is produced by the presence of a foreign body in the external auditory meatus or by disease of that passage. The mere introduction of the speculum may cause cough so violent as to make the examination most difficult. The afferent nerve is the auricular branch of the pneumogastric or, according to others, the auriculotemporal branch of the fifth nerve.

Stomach Cough.—The popular explanation of certain forms of cough as a manifestation of disorders of the stomach is sustained neither by pathological nor experimental investigation. The morning cough of the drunkard is to be accounted for by the pharyngeal catarrh which accompanies chronic alcoholic gastritis; of the consumptive, by lesions of the lungs or larynx, with which secondary gastric disorders are commonly associated. The cough occasionally observed in subacute catarrhal gastritis and which disappears as the gastritis improves is due to the associated pharyngitis. Bronchitis is very common in chronic alcoholism and other forms of ill health with derangement of the gastro-intestinal tract, and a careful investigation of the cases of so-called stomach cough will almost always demonstrate, with the gastric condition, associated lesions of the respiratory tract which account for this symptom.

Liver Cough.—This symptom is doubtless due to irritation of the diaphragmatic pleura. It is met with in certain cases of hypertrophy of the liver, perihepatitis, hydatids, and hepatic and subphrenic abscess.

Disease or enlargement of the spleen may also in rare cases be the cause of cough.

Cough may exceptionally be provoked by pressure in the region of the liver or spleen.

For some years the writer had under observation a case of ventral hernia midway between the tip of the ensiform cartilage and the umbilicus in the median line, in which violent paroxysmal cough attended the presence of the tumor and immediately subsided upon its reduction.

Dentition.—Cough is not uncommon during the first dentition, without manifestations of disease of the respiratory tract. It appears before the eruption of the successive groups of teeth and disappears with the completion of the process.

Mediastinal Cough.—Mediastinal tumor or abscess, thoracic aneurism, enlarged bronchial glands, and caries of the dorsal vertebræ are occasional causes of persistent and troublesome cough. Massive hypertrophy or great dilatation of the heart is also in some instances accompanied by cough.

Nervous Cough.—The diagnosis of nervous cough is only to be made when, in default of direct signs or symptoms or by exclusion, the absence of disease of the respiratory organs or other lesions recognized as the cause of this symptom can be established. Not rarely cough is the only direct manifestation of a bronchitis or pulmonary tuberculous process in which, for the time being, the ordinary physical signs are lacking. It often happens that the diagnosis of nervous cough is made when the intensity and persistence of the cough is altogether out of proportion to its actual physical cause, as is common in neurotic individuals. Nevertheless in some cases cough must be recognized as a purely nervous phenomenon. There are persons who cough whenever their feet are chilled or a cold air blows upon an exposed part of the body. Paroxysmal cough of purely nervous nature is not uncommon in both sexes at puberty. Cough is one of the multitudinous symptoms of hysteria. Under certain conditions cough may occur in neurotic individuals in consequence of disease or irritation of the mammæ or of the genital organs in either sex. It has been shown that cough may be excited by irritation of the floor of the fourth ventricle above the centre for respiration. Whether, under pathological conditions, a true "centric cough" occurs is open to question. Irritation of a "cough centre" has been invoked to explain hysterical and other coughs of purely nervous origin.

Clinical Varieties of Cough.—The *character* of the cough is of importance in diagnosis. It is modified according to the seat of the irritation, whether respiratory or extrarespiratory; by the anatomical structure involved, as the larynx, bronchi, pleura; by the amount and consistence of the irritating substance; and by the constitutional peculiarities of the patient. The following forms demand especial consideration:

Dry Cough.—Patients themselves recognize the distinction between dry and moist cough. Cough due to irritation of the respiratory mucous membrane is dry when it occurs in the absence of secretion or if the secretion is tough, tenacious, and not readily dislodged. Extrarespiratory cough—so-called "reflex cough"—is dry. The sound is hacking, barking, or ringing and is not accompanied by expectoration. Dry cough is frequently spoken of as "unproductive." It occurs in the early stage of acute bronchitis, bronchial asthma, influenza, pneumonia, and pleurisy, in affections of the upper air-passages, phthisis, and pertussis. This is the cough which is excited by the inhalation of foreign bodies, irritating fumes, or dust, and by extrarespiratory causes. It results from pleural irritation and is encountered in pleurisy with fibrinous exudate and upon the withdrawal of an effusion.

Loose or Moist Cough.—This cough is associated with sounds indicating the part played by fluid in the mechanism of its production. It differs from dry cough not only in its acoustic characters but also in the occurrence of expectoration. It is "productive." Loose cough occurs in the later stages of acute bronchitis, influenza, and pneumonia; toward the close of the paroxysms of whooping-cough and asthma; in chronic bronchitis, bronchiectasis, and pulmonary gangrene; in advanced phthisis; and in all conditions attended by moderate or abundant bronchial secretion.

Constant and Recurrent Cough.—Adjectives such as constant, persistent, recurrent, designate peculiarities of the cough dependent upon the persistence or recurrence of its cause. So-called nervous cough is usually persistent; also the cough which attends diseases of the upper air-passages and acute bronchitis and that of bronchorrhœa. On the other hand, in chronic bronchitis, especially when there is bronchial dilatation, the cough is apt to occur paroxysmally at varying intervals. The expectoration of a large amount of matter is followed by relief. After a time the secretion reaccumulates, a mere overflow into the bronchi on change of posture excites cough, and the process is repeated. This form of cough attends the later stages of phthisis with large vomicæ and occurs in some cases of empyema with bronchopulmonary fistula. Recurrent cough is very common in chronic bronchitis and phthisis; it constitutes the "morning cough" of these conditions. The secretion accumulates slowly during sleep without exciting irritation. On waking, the patient moves, the accumulated material shifts its position a little, the bronchial reflex is brought into play, cough results and continues until the offending mass is expelled.

Paroxysmal Cough.—Recurrent cough is not necessarily paroxysmal, and paroxysmal cough for the time may be constant. The word paroxysm conveys the idea of suddenness and intensity. Such is the cough of acute inflammatory conditions; that caused by foreign bodies in the air-passages, the insufflation of saliva, and the like; by the periodical flooding of the bronchi with the abundant contents of the cavities in phthisis, bronchial dilatation, pulmonary abscess, or gangrene; and finally that of pertussis. The paroxysm recurs at intervals varying from an hour or less to once a day or longer. In the case of cavities or a bronchial fistula in empyema the interval is determined by the accumulation; in pertussis by the intensity of the neurosis. In the latter condition the cough is characteristic. Into its production two factors enter: an abundant tenacious mucus and a pathological nervous excitability. After a long inspiration, the expiratory cough-efforts succeed each other with such rapidity that inspiration is partial or absent until at last a prolonged inspiration takes place which, by reason of the spasmodic contraction of the glottis, is attended by a harsh, crowing sound or whoop; hence the common term whooping-cough. A somewhat similar inspiratory whoop sometimes attends the paroxysmal cough of other diseases, but so infrequently that in the vast majority of cases the phenomenon justifies the diagnosis of whooping-cough. The differential diagnosis involves consideration of the age of the patient, the presence or absence of an epidemic, history of exposure, the duration and course of the attack, and the presence or absence of lesions capable of causing violent paroxysmal cough other than that of pertussis. The cough which attends enlargement of the bronchial glands, mediastinal tumor, caries of the dorsal vertebræ, enlargement of the heart, and pericardial effusion is paroxysmal and dry. Quite often it has the laryngeal character. Very violent paroxysms of cough frequently result in retching and vomiting and, as a result of the venous congestion from intrathoracic pressure, in hemorrhage from mucous surfaces or into the skin.

Croupy Cough.—A dry cough, described as metallic, ringing, or croupy, is characteristic of laryngeal irritation. The voice is usually hoarse or aphonic, though it may be unimpaired. The laryngeal cough occurs in simple or exudative laryngitis, spasm of the larynx, from the inhalation of smoke or dust, as the result of the irritation produced by foreign bodies in the larynx, and in tuberculous, syphilitic, or cancerous ulceration. The cough of hysteria is usually laryngeal in character, though upon laryngoscopical examination neither swelling nor paralysis may be present. It is described by such adjectives as barking or croaking and resembles other hysterical manifestations by the readiness with which it may be voluntarily produced. A barking laryngeal cough, in the absence of swelling of the laryngeal mucosa or paralysis of the vocal cords or of lesions directly or indirectly involving the recurrent laryngeal nerves, is commonly hysterical.

Suppressed Cough. — Voluntary efforts to suppress cough are made under circumstances in which the sound of the cough is likely to annoy others and when the act is attended by pain, as in pleurisy, acute peritonitis, and some forms of acute bronchitis. The suppressed cough is usually lacking in tone, and is explosive and persistent.

Undeveloped Cough.—Incomplete efforts at cough, unattended by the characteristic sound, are observed in cases of destructive ulceration or paralysis of the vocal cords or of partial paralysis of the expiratory muscles. This form of cough is encountered in laryngeal phthisis, in patients suffering from bulbar paralysis, in enormous ascites or abdominal tumors, and in conditions attended with extreme debility, especially the later stages of croupous and bronchopneumonia, chronic bronchitis, pulmonary œdema, and consumption.

In the majority of instances the diagnostic significance of the symptom cough is direct and obvious. It is the indication of disease affecting the respiratory organs, manifested more or less fully by concurrent signs and symptoms; in a far smaller proportion of cases its significance is remote and obscure and only to be learned by close and systematic study of the various organs or parts to derangements of which it may be due. Important among these derangements are diseases of the intrathoracic circulatory organs, mediastinum, ears, teeth, and nose, some nervous affections, and the neurotic constitution. To this list must be added malingering, since cough may be a voluntary act.

THE EXPECTORATION OR SPUTUM.

These terms are applied to material voided by coughing or hacking. The expectorated substance is usually a secretion or exudate derived from the mucous membrane of the nose, pharynx, larynx, or bronchial tubes, or from the alveoli. It may consist of pus, which finds its way into the air-passages from an abscess or an empyema, or of blood from the pulmonary vessels or an aneurism. With these substances are frequently admixtures of food, drink, and the secretions of the mouth. Macroscopic and microscopic foreign bodies which have found their way into the respiratory passages are usually voided in the sputa.

Any of these substances may be present and not expectorated. Infants and young children almost always swallow the sputa and older persons frequently do so as a habit or from inability to expectorate or in abnormal mental states.

The naked-eye examination of the expectorated matter is frequently of great use in diagnosis; the microscopic examination is often essential. For the ordinary bed-side examination a considerable quantity of the sputum should be collected, preferably in a transparent glass spit-cup.

The quantity of the sputum varies according to the nature of the pathological process. Persistent and distressing cough may yield only an occasional small tough mass of tenacious material, as in dry bronchitis or beginning phthisis. In other patients an occasional spell of coughing may bring up enormous quantities of material, as in some forms of chronic bronchitis, bronchiectasis, advanced phthisis, pulmonary œdema, and hæmoptysis. The amount of pus expectorated in empyema with bronchopulmonary fistula may exceed 1000 c.c. in twenty-four hours.

The consistence bears some relation to the amount. An abundant expectoration is usually more fluid than a scanty one. Sputum composed of blood, pus, or a serous fluid is always thin; that consisting of mucus or mucopus usually thick and frequently tough and tenacious.

The reaction of fresh sputum is commonly alkaline. After standing for some hours in the cup, the sputum yields an acid reaction—a change due to decomposition processes caused by bacteria.

The color and translucency vary with the nature of the disease. Mucous expectoration may be transparent and thin, resembling saliva in consistency, or much thicker and still transparent. In proportion as cellular elements are present the sputum becomes thick and opaque, assuming the yellowish or greenish-yellow hue of pus. The gradations are expressed by the terms mucous, mucoid, mucopurulent, and purulent expectoration. Serous expectoration is usually clear and transparent, sometimes slightly tinged with blood. It is thin, frothy, and abundant, and occurs in œdema of the lungs and in the rare cases of perforation of a serous pleural exudate. The albuminous expectoration which exceptionally follows the aspiration of a pleural exudate is also thin, colorless, and abundant. The color is red when the sputum is admixed with blood. The proportion varies from pure blood to a mere trace sufficient to impart a faint pink tinge. Hemorrhagic sputum occurs in traumatism of the lungs, in the blood-spitting of tuberculosis, in pulmonary infarct, and in croupous pneumonia. It is also present in cases of gangrene of the lung, tumor of the lung, and intense pulmonary congestion. The "rusty sputum" of pneumonia owes its varying shades of color to derivatives of the blood-coloring matter. In rare instances the sputum of pneumonia is lemon-yellow or grass-green. These variations suggest the changes in color that take place in the subcutaneous blood extravasations following a bruise. In the adynamic and septic forms of croupous pneumonia and, more rarely, in gangrene of the lungs the expectoration is fluid and dark colored. This form of sputum is described as "prune-juice" expectoration. The sputum in malignant disease of the lungs is often viscid, tenacious, and of a bright red color. This is the "currant-jelly" sputum of authors. A still more objectionable term is "anchovy-sauce" sputum—a term applied to brownish-red sputum such as is seen in rupture of a liver abscess through the lungs, the peculiar appearance of which is due to the mixture of altered blood, pus, and bile.

BLOOD-STREAKED SPUTUM may occur in the following conditions: violent cough, acute bronchitis, or disease of the mitral valves. It may result from the admixture of blood from the mouth, as in the case of scurvy and other forms of inflammation of the gums with bleeding, or of ulceration of the tonsils or pharynx, or from the oozing of blood from an aortic aneurism into a bronchus. It occurs also in acute bronchopneumonia and plastic bronchitis. It is very often observed a day or two after an attack of hæmoptysis. Under these circumstances the streaks or masses of blood are clotted and dark. Blood-streaked sputum is not uncommon during the course of pulmonary phthisis.

YELLOW OR GREEN SPUTA can only be regarded as deriving their color from altered bile pigment when icterus or at least yellowness of the conjunctiva and biliary pigment in the urine are actually present. Icteric sputum may occur not only in pneumonia complicated with jaundice but also in any form of lung disease in a patient suffering from jaundice. A peculiar brownish tint is sometimes seen in the sputum in cases of chronic valvular disease. It is due to the presence of amorphous pigment in the epithelial cells. The brownish sputum sometimes seen in pulmonary abscess and other destructive processes involving the lung owes its color to the presence of hæmatoidin crystals, which are also the source of the coloring matter in the ochre-yellow purulent sputum of liver abscess with perforation into the lung. Greenish sputum is sometimes encountered in sarcoma of the lungs and very rarely in carcinoma. Remarkable coloration follows the habitual inhalation of certain dust-particles. Black sputum is common in those who breathe an atmosphere laden with coal-dust or soot. The pigment particles are only to a limited extent free in the sputum; much more commonly they are enclosed in round or oval cells which are in part epithelial, in part leucocytes.

The color of the sputum varies in different forms of pneumonoconiosis. In anthracosis it is often of an intense black; in the siderosis of mirror polishers it may be ochre-red; workers in lapis lazuli may have a blue sputum, and so on. The dust particles which are expectorated are those recently inhaled which have not yet penetrated to the lung parenchyma, as is shown by the fact that the color disappears from the sputum in the course of a short time after the workman has abandoned his occupation. If however the color persists or returns after a time, it is the sign of a destructive process, usually tuberculous. Various colors may be imparted to the sputum by articles of food or drink, as milk, wine, coffee, or medicines. Finally after the sputum has been ejected it may undergo color changes in consequence of the growth of chromatogenous bacteria and thus become blue, green, yellow, or red. The Bacillus pyocyaneus may be the cause of a blue discoloration of the sputum.

Air.—Air in the sputum is shown by the presence of minute bubbles. The quantity depends upon circumstances. It is greater in sputum from the finer than in that from the larger tubes, in sputum of thin than in that of thick and tenacious consistency, and in the sputum which is largely composed of mucus than in that which is chiefly pus. A little water in the spit-cup enables us to estimate the relative amount of air, as it affects the specific gravity; sputa which float contain air; those which sink do not. The sputa of phthisis and bronchitis often present the appearance of flat circular or coin-shaped masses—the so-called "nummular sputa"—or the masses may be globular; they are commonly grayish-white and sink in water; sometimes they are buoyed up by the small bubbles of air which they contain.

Stratification.—Layer formation takes place in the collected sputa of chronic bronchitis with abundant expectoration—bronchorrhœa—of bronchiectasis, putrid bronchitis, and gangrene of the lungs. The material is of thin consistence and abundant. As a rule it collects in three well-defined layers which can be studied by the use of a glass spit-cup. The upper stratum contains air and is often frothy, the middle is fluid and consists of mucus or pus-serum, and the lower is sedimentary and made up of pus corpuscles, molecular lung detritus, and shreds of necrotic tissue.

Odor.—The odor of fresh sputum has, under ordinary circumstances, nothing characteristic. Speedy decomposition renders it offensive. The sputum of putrid bronchitis, bronchiectasis, gangrene of the lung, and perforating empyema is always heavy and fetid; frequently horribly offensive. In abscess of the lung and in many cases of advanced phthisis also it is offensive. The foulness is imparted to the expired air, which not infrequently is even more obnoxious than the sputum. It is probable that in the cases of pulmonary consumption in which the sputum and breath are foul there is already cavity formation, though too small in some instances to be recognized by the methods of physical diagnosis, in which the secretion collects and undergoes decomposition. Very often the odor is imparted to the breath by offensive material in the crypts of the tonsils or by decaying teeth or other necrotic material in the mouth—a fact that cannot in all cases be established by the use of deodorizing mouth washes, since they act only upon the surfaces with which they come in contact and cannot reach deeply-seated tissues from which the odor may proceed.

Other Macroscopical Characters of the Sputum. — Very often the expectorated material presents a homogeneous appearance, as is the case with mucus, pus, blood, etc. Occasionally, on the other hand, the matter expectorated at different times varies in appearance and not infrequently a single mass consists partly of mucus and partly of pus, or of these substances with masses of blood. The purulent expectoration of an empyema or a pulmonary abscess is sometimes flaky or thready, best shown when the sputum is suspended in water. The naked-eye characters of the sputum may be conveniently studied by pouring a small quantity upon a plate or slab of which half is black, the other half white, or placing a specimen between two glass plates and examining it over a white and black background alternately. A hand lens may be used and particular objects removed for microscopical examination. Minute, dirty gray masses of necrotic lung tissue containing elastic fibres may be detected in the specimen in gangrene and abscess of the lung and in the later stages of phthisis; fragments of necrotic cartilage in destructive processes involving the bronchi, the trachea, or the larynx, and in rare cases shreds of tissue from tumors of the bronchi or lungs. Minute, dirty white or yellowish masses, in some instances constituting casts of the smaller bronchial tubes, are seen in fetid bronchitis and gangrene of the lungs. These masses consist of aggregations of bacteria and crystals of the fatty acids. They have an intensely disagreeable odor. Similar masses may be expectorated in lacunar tonsillitis and are sometimes present in the crypts of the tonsils in the absence of inflammation. Curschmann's spirals are visible to the naked eye and may be studied with the lens. They consist of twisted masses which may reach 1 or even 2 cm. in length and have a diameter of about 1 mm. These masses are made up of a highly refractive central undulating core or thread around which are coiled spiral filaments which are sometimes branching. The central core was at one time thought to be fibrinous, but has more recently been shown to consist of a substance analogous to mucin. These spirals are formed in the finest bronchial tubes as the product of an exudative bronchiolitis, and as this pathological process is frequently associated with bronchial asthma the spirals are very often found in that disease and in well-marked cases are sometimes present in great numbers. The association, however, is by no means constant; cases of asthma are occasionally encountered in which no spirals can be found in the sputum, and the spirals are sometimes present in the expectoration of cases of bronchitis unattended by asthmatic symptoms. Curschmann's spirals occasionally appear also in the sputum of croupous pneumonia and are then seen to be in strong contrast with the fibrinous casts of the bronchioles which occur in that disease. They have also been encountered in the sputum of pulmonary phthisis. Microscopically, leucocytes, notably eosinophiles, epithelial cells, and Charcot-Leyden crystals are found entangled in the spirals.

Fibrinous coagula, recognizable by their white or grayish-white color, tough consistence, and characteristic form, are found in the sputum under varying pathological conditions. They are usually coughed up in masses surrounded with mucus and, when of great size, with difficulty. In diphtheria fibrinous pseudomembrane is expectorated, sometimes in irregular masses, sometimes as a fibrinous mould, more or less incomplete, of the larynx or trachea. When the diphtheritic exudate extends to the bronchi, branching casts are sometimes coughed up. These casts may be easily recognized in the sputum and are of great importance both in diagnosis and prognosis. Fibrinous casts are common in croupous pneumonia, in the sputum of which they are frequently present in great numbers. They can be readily seen when the sputum is shaken with water in a test-tube, or when the masses of mucus in which they are embedded are shaken out in water with a forceps. In pneumonic sputum the fibrinous casts are small. Similar casts consisting chiefly of mucus are characteristic of so-called fibrinous or croupous bronchitis and provoke the intense paroxysmal cough of that disease.

Foreign bodies that have found their way into the air-passages by aspiration are usually expectorated promptly. They may, however, remain in a bronchus for a long time and give rise to symptoms of varying intensity. Instances are recorded in which a tooth, cherry-pits and other seeds, a beard of wheat, etc., have been expectorated after periods of months or years. Bronchial concretions, consisting in the main of lime salts and sometimes of considerable size, are in rare instances found in the sputum. They occur only in chronic conditions and are formed in the cavities of phthisis and bronchiectasis, or consist of fragments of bronchial glands that have undergone calcareous degeneration and found their way into the bronchial system. Even more rare is the presence in the sputum of echinococcus daughter cysts, membranes, or hooklets, which have found their way from the lung, pleura, or the liver into the bronchi.

The Sputum in Different Diseases.

Bronchitis.—The sputum is usually mucoid and mucopurulent. As a rule, at the beginning of an acute bronchial catarrh the bronchial secretion is diminished and the sputum scanty. In the course of some days, as the symptoms ameliorate, the expectoration becomes more abundant, less tenacious, and distinctly purulent. As the general symptoms improve there is a gradual diminution in the quantity of the sputum. In chronic bronchitis the expectoration varies greatly; sometimes it is more, sometimes less purulent. The subjective sensations of the patient are usually better when the sputum is of moderate amount, worse when the expectoration is suppressed or greatly increased in quantity (see bronchitis).

Fibrinous or Croupous Bronchitis.—The sputum differs from that of ordinary bronchitis in that from time to time it contains fibrinous casts associated with blood. Charcot-Leyden crystals are also present. The expectoration of the larger casts very often takes place after distressing cough, recurring in paroxysms which are separated by periods of urgent dyspnœa.

Pulmonary Tuberculosis.—The sputum of tuberculosis presents to the naked eye nothing characteristic. All varieties of sputum that occur in ordinary bronchitis, from mucous to purulent, occur in phthisis. In advanced ulcerative phthisis purulent expectoration is often constant and abundant. For the provisional diagnosis the presence of the minute grayish masses which frequently contain colonies of tubercle bacilli is important. Very often the sputum has an offensive odor; this is especially the case when there are cavities, the contents of which undergo stagnation and decomposition. A positive diagnosis rests upon the presence of tubercle bacilli and, in the absence of other destructive pulmonary lesions, the presence of elastic fibres. It is important for the student to bear in mind that there is no constant relation between the abundance of these morphological elements and the intensity of the process, therefore the gravity of the prognosis. There are cases of pulmonary tuberculosis of the gravest character in which neither tubercle bacilli nor elastic fibres are found. Very often these are cases of *phthisis florida* or of *disseminated miliary tuberculosis* in which the constitutional symptoms develop in advance of the local manifestations. The abundant catarrhal secretion, so common in unfavorable cases, proportionately diminishes the number of tubercle bacilli present in single specimens. On the other hand tubercle bacilli and elastic fibres are frequently found in the early stages at a period when the physical examination of the lung yields vague and uncertain signs. The diminution or temporary disappearance of tubercle bacilli and elastic fibres from the sputum cannot be regarded as indicating a favorable progress of the case in the absence of the general clinical indications of an arrest of the process, such as diminished cough, improved appetite, gain in weight, and disappearance of fever. In a suspected case the presence of tubercle bacilli in the sputum justifies a positive diagnosis. Their absence cannot be regarded as conclusive until repeated examinations have been made.

Acute Miliary Tuberculosis.—The sputum is that of ordinary catarrhal bronchitis and does not contain tubercle bacilli except when there is an associated ulcerative phthisis. In a large proportion of the cases there is no expectoration.

Croupous Pneumonia.—Hemorrhagic sputum is characteristic. Blood-spitting may be the initial symptom. At first the sputum is commonly mucoid, transparent and homogeneous; after twenty-four hours it is blood-tinged and viscid so that it adheres to the bottom of the spit-cup when turned upside down, and sometimes has to be wiped from the lips or face of the patient. At first red from unchanged blood-coloring matter it gradually becomes rusty or orange-yellow in color. Occasionally the sputa are variable; sometimes mucoid, sometimes blood-streaked, at other times pure blood. When jaundice is present the sputum may be green or yellow from the presence of bile pigment. Very commonly the sputum contains fibrinous casts of the smaller tubes. If there is an associated bronchitis of the smaller tubes the typical pneumonic sputum may be modified by the presence of mucus or mucopus. Fluid sputum of a dark brown color—the so-called "prune-juice" expectoration—is an unfavorable sign since it may indicate a beginning œdema of the lungs. In some instances a diminished consistency of the sputum marks the beginning of resolution. The amount of sputum in croupous pneumonia is very variable. In children and the aged, and in adynamic cases, there may be none, and exceptionally it may be scanty in classical cases in adults. A quantity amounting to 200–500 c.c. in twenty-four hours is not uncommon. The amount after the crisis, abundant at first, gradually diminishes.

In some cases there is at this period little or no expectoration. Under the microscope are seen leucocytes, erythrocytes, mucous corpuscles, epithelial cells, and occasionally hæmatoidin crystals. The pneumococcus of Weichselbaum and Fränkel is present in the vast majority of cases, and sometimes Friedlander's bacillus. Fibrinous casts of the bronchioles and moulds of the alveoli are not uncommon. Chemically the expectoration is particularly rich in sodium chloride.

Bronchopneumonia, Including Aspiration Pneumonia and Hypostatic Pneumonia.—The sputum usually presents the appearance of the ordinary forms of bronchitis; exceptionally that of croupous pneumonia. The latter is intelligible, since not only in the clinical phenomena but also in the histological findings there are cases of bronchopneumonia which are difficult to distinguish from croupous pneumonia. In these cases the sputum is hemorrhagic and contains fibrinous exudate. Bacteriologically a mixed infection is the rule. The pneumococcus and Friedlander's bacillus are found in association with the ordinary pus-producing and other organisms. The Klebs-Löffler bacillus is present when the lesions are secondary to diphtheria. In the lobular forms the streptococcus is the common organism; in the lobar forms, the pneumococcus.

Gangrene of the Lungs.—The intensely offensive odor, abundance, fluidity, and dark, dirty, greenish-brown color are characteristic. Upon standing the sputum separates into three strata—an upper frothy layer, which may contain necrotic particles of lung tissue which float by reason of entangled air, a middle thin layer, and a greenish-brown sediment which consists in part of leucocytes, in part of gangrenous detritus. Shreddy fragments of lung tissue of considerable size and frequently showing the alveolar arrangement may be picked out if the sediment is spread upon a glass. Under the microscope are seen elastic fibres, pigment granules, crystals of the fatty acids, cholesterin, leucine and tyrosine crystals, bacteria, and leptothrix. Altered blood-corpuscles are also present. When the fluid is retained in the gangrenous cavity for some time, the elastic fibres may undergo solution owing to the action of a peptonizing ferment. The odor is the more intense in proportion as the communication between the gangrenous areas and the bronchi is more free. Cases occur in which, in the absence of odor during life, circumscribed areas of gangrenous lung have been found upon post-mortem examination.

Abscess of the Lung.—The sputum is essentially purulent. It is offensive, but less intensely so than that of gangrene. When placed in water it has a thready or granular appearance. When the perforation is small there is an accompanying catarrhal bronchitis and the sputa are mucopurulent. When, however, the abscess discharges abruptly, a large amount of pus commingled with masses of necrotic lung tissue and containing elastic fibres in abundance is discharged. Microscopically the sputum contains hæmatoidin, cholesterin and fat crystals and various bacteria.

Perforating Empyema.—The sputum resembles that of pulmonary abscess. It may be at first free from odor but in the course of a little time becomes offensive. It is voided in considerable quantities at varying intervals. Elastic fibres are wholly absent or are present in small numbers. Hæmatoidin and other crystals and pyogenic bacteria are present.

Putrid Bronchitis.—The expectoration presents characteristics similar to that of perforating empyema. It is purulent and foul-smelling, but does not contain elastic fibres. It is voided from time to time in moderate amounts; not in large bulk at intervals of some hours as is the case in empyema with bronchopulmonary fistula and bronchiectasis.

Bronchiectasis.—In saccular bronchiectasis the sputum is sometimes mucopurulent, sometimes purulent. It is brought up from time to time in severe paroxysms and in large quantities—mouthfuls. These paroxysms may follow change of posture, the cough reflex being excited by the shifting of accumulated secretion from the dilatation to the normal bronchial tube. A paroxysm usually occurs in the morning. The color of the expectorated matter may be gray or grayish-brown. It is usually fluid, acid-smelling, sometimes extremely fetid. Upon standing it separates into three layers, an upper consisting of brownish froth, a middle thin watery layer, and a lower, thick and granular. Microscopically the sputum consists of pus corpuscles, epithelial cells, erythrocytes, and large numbers of crystals of the fatty acids. Hæmatoidin crystals are sometimes seen. In the absence of bronchial ulceration, elastic fibres are not found, nor are tubercle bacilli present. Nummular sputa are uncommon. In many cases the sputum cannot be distinguished from that of a putrid bronchitis. Hemorrhage occasionally occurs.

Œdema of the Lungs.—The sputum is usually thin, frothy, colorless or slightly blood-tinged, and abundant. Upon standing it deposits a sediment consisting in part of red blood-corpuscles and in part of elements characteristic of the antecedent condition, as bronchitis or pneumonia. It is largely made up of blood-serum and is therefore rich in albumin. In the rare cases in which perforation of the lung occurs in serofibrinous pleurisy the expectorated matter resembles that of pulmonary œdema but is richer in albumin. A very abundant sputum, similar in character, is sometimes expectorated after paracentesis thoracis, beginning toward the close of the operation—the *expectoration albumineuse* of the French. This serous sputum is the result of an acute pulmonary œdema following the dilatation of the compressed lung.

Bronchopulmonary Hemorrhage—Hæmoptysis.—In the blood-spitting which follows traumatism, the rupture of an aneurism, the lesions of tuberculosis, or new growths involving the lungs the sputum consists of more or less abundant, bright red, frothy blood. The distinction between venous and arterial blood cannot be made, since the dark blood of the pulmonary arteries becomes oxygenized and frothy during its course through the bronchial tubes. The differential diagnosis between hæmoptysis and hæmatemesis rests upon the following facts: In bronchopulmonary hemorrhage the blood is coughed up. In gastric and œsophageal hemorrhage it is vomited, but the account of the patient or his friends is not always satisfactory; in the excitement and alarm the distinction may not be made. Moreover violent paroxysmal cough may on the one hand be followed by gagging and vomiting, while on the other hand some portion of vomited blood may be drawn into the larynx by aspiration and thus excite coughing. The examination of the blood itself is important. Bright red, frothy blood may usually be referred to a lesion of the respiratory tract; blood that is dark, clotted, and free from air-bubbles, to the digestive tract. But there are exceptions to this rule. In profuse hemorrhage from the stomach the blood is sometimes vomited so rapidly that it is bright red and fluid, while in abundant pulmonary hemorrhage, resulting from erosions of a large branch of the pulmonary artery, the expectorated blood may be dark in color and contain but little air.

The reaction of the blood in hæmoptysis is alkaline. In hæmatemesis which occurs during digestion, when the stomach contains a large amount of acid fluid, the reaction may be acid. Too great importance cannot be ascribed to the reaction of the blood in doubtful cases, since vomited blood is frequently alkaline. The presence of particles of food in the blood is of importance in diagnosis.

There are, however, cases in which the distinction between hæmoptysis and hæmatemesis cannot be immediately made.

The condition of the patient prior and subsequent to the bleeding is in doubtful cases of greater importance than the appearance of the blood. A history of gastric symptoms before the blood loss or the occurrence of such symptoms subsequently is common in bleeding from the stomach. The presence of altered blood in the stools after the hemorrhage points to bleeding from the stomach rather than from the lungs. On the other hand the mere fact that the patient has suffered for some time from cough and expectoration is suggestive of pulmonary hemorrhage, which is apt to be followed for some days by the occasional expectoration of small blood-clots or of sputum mixed with blood. When due consideration is given to these facts errors of diagnosis are not likely to occur.

Hemorrhagic sputum is occasionally encountered in acute bronchitis. This sputum is to be distinguished from pneumonic sputum by the fact that the blood is present in streaks rather than as a homogeneous mixture. Profuse hæmoptysis rarely has its seat of origin in the larynx or trachea, since the blood-vessels of these organs are of relatively small size. On the other hand, blood-streaked sputa are not uncommon in acute catarrhal inflammation of the trachea, larynx, or pharynx. There are forms of hemorrhagic bronchitis characterized by blood-tinged sputum which continue for some days or weeks. Such cases are not uncommon during epidemics of influenza. It sometimes happens, especially during sleep, that the blood in epistaxis trickles into the pharynx and is swallowed. If vomited, such blood may be regarded as due to gastric ulcer. If the blood in the pharynx under these circumstances excites cough and is ejected mingled with mucus, it may be erroneously regarded as coming from the lungs. If the trickling blood be seen upon the wall of the pharynx the diagnosis is at once established and the precise site from which it comes may be determined by means of the rhinoscope.

Infarcts.—The sputum in hemorrhagic infarct is commonly dark in color and resembles pure blood, from which it differs in its somewhat tenacious consistence, suggestive of pneumonic sputum. In point of fact the sputa in cases of pulmonary infarct may vary according to the amount of bronchial secretion present from pure blood to a tenacious blood-tinged mucus.

Chronic Valvular Disease.—Hemorrhagic sputum occurs in certain cases of valvular disease of the heart, particularly in mitral stenosis.

VI.

CIRCULATION; PULSATION; RADIAL PULSE; ANOMALIES OF THE PULSE; CAPILLARY PULSE; VENOUS PULSE.

CIRCULATION.

The term arterial pulse is used to designate the rhythmical fluctuations of the arterial pressure which correspond to the contractions of the ventricles of the heart. These rhythmic fluctuations depend upon the intermittent injection of blood from the ventricle to the aorta, upon the resistance to the arterial flow produced by friction, and upon the elasticity of the walls of the arteries. After the blood enters the capillaries the pressure is no longer intermittent, but is continuous, and pulsation under normal conditions disappears. The pulse may be affected by changes either in the force of the ventricular contractions, in the elasticity of the arteries, or in the peripheral resistance, and by varying combinations of these modifications. The examination of the arterial pulse is therefore obviously of great diagnostic importance. By this means conclusions may be reached in regard to a wide range of clinical facts, including the innervation of the heart, the power of the heart muscle, the blood-pressure, the blood loss in hemorrhage and anæmia due to other causes, the condition of the peripheral arteries, the action of fever-producing toxins upon the heart and blood-vessels, and finally, under certain conditions, in regard to the presence and nature of valvular lesions.

PULSATION.

ARTERIAL PULSATION may be studied in any of the superficial arteries. The methods employed in ordinary clinical work are palpation and inspection. Auscultation is of more limited application in the study of the blood-vessels. The results obtained by the use of the sphygmograph are of more value in clinical research and for purposes of record and comparison than for diagnosis.

The increase in the contents of the arterial system which causes the pulsation is accompanied not only by an increase in the tension of the artery at any given point but also by an increase in the length of the vessel. This increase in length results in a more or less marked lateral undulation and exaggeration of the curves of the vessel, normally not sufficient to attract attention, but conspicuous in the temporal arteries of emaciated persons and at various points in the course of the superficial arteries in conditions, such as aortic insufficiency, which are attended with cardiac hypertrophy and relaxation of the arterial walls. The arterial pulse, corresponding to a contraction of the ventricles, is not perceptible at the same moment at all parts of the body, an appreciable interval separating the cardiac impulse, the radial pulse, and that of the dorsal artery of the foot.

THE AORTA AND ITS BRANCHES.—Pulsation in the notch of the sternum is occasionally seen in aged persons in the absence of disease. It occurs in dilatation of the aorta and is a sign of aneurism of the transverse portion of the arch. In rare cases it is due to an anomalous distribution of the branches of the aorta in this region.

PULSATION AT THE ROOT OF THE NECK is common in cardiac hypertrophy and dilatation, in aortic insufficiency, and in neurotic and anæmic conditions, especially during periods of physical or mental excitement. It is a prominent symptom of exophthalmic goitre. Under these circumstances pulsation of the aorta is associated with a heaving impulse in the innominate and carotids, communicated to the overlying tissues, so that throbbing in this region becomes a sign of importance. It is often accompanied with distention of the veins and flushing of the face.

The differential diagnosis between dynamic dilatation of the arch of the aorta and aneurism cannot in all cases be made during life. Not rarely when the signs of dilatation of the arch and enlargement of the innominate and right carotid have been well marked clinically, the vessels have been found post mortem to be of normal measurement.

PULSATION OF THE SUBCLAVIANS occurs in the general pulsation at the root of the neck, above spoken of. It is usually less marked than that of the innominate and carotids. Visible pulsation of the subclavians is sometimes present in consolidation and retraction of the lung in phthisis.

PULSATION OF THE ABDOMINAL AORTA is very common. It may often be made out in quite thin persons under normal conditions both by inspection and palpation. Under these circumstances it is of very slight intensity. More vigorous pulsation in the line of the abdominal aorta, namely, in the median line or slightly to the left of it, and in the epigastric zone is an important sign of disease. Objectively the pulsation varies in degree. It is frequently violent and throbbing and may be demonstrated by the motion communicated to the stethoscope lightly pressed upon the surface. Subjectively the sensation of throbbing is annoying and frequently distressing. It often prevents sleep. Epigastric pulsation is not in all instances due to the movements of the aorta. It may be directly due to the heart. A faint pulsation in the region of the ensiform cartilage occurs in physiological over-action of the heart, in hypertrophy and dilatation of the right ventricle, and in displacement of the heart towards the right in consequence of left-sided pleural effusion or of emphysema. In the last named condition the epigastric pulsation is often marked, since the heart is displaced toward the median line and the right ventricle is hypertrophied. The pulsation is transmitted to the left lobe of the liver. It is more marked in the neighborhood of the ensiform appendix and costal cartilages than toward the umbilicus, and nice observation will show that it corresponds in time to the cardiac systole, whereas aortic pulsation is slightly postsystolic.

The most common causes of pulsation of the abdominal aorta are referable to the nervous system—simple dynamic pulsation. The throbbing may be a direct manifestation of neurasthenia or hysteria, or it may be a reflex manifestation of disorders of the gastro-intestinal tract. It is much more common in females and in early life. It occurs also as the result of diminution of the amount of blood and thus becomes one of the signs of anæmia due to hemorrhage or other cause. Marked epigastric pulsation frequently occurs as a sign of enlarged lymphatic glands, carcinoma of the stomach or pancreas, or other form of tumor overlying the aorta. In rare instances fecal accumulations in the colon transmit the aortic impulse to the surface. Thorough evacuation of the bowels is an imperative preliminary measure in the diagnosis of doubtful cases. Finally it may be due to an aneurism.

The diagnostic significance of this sign varies greatly and in some cases is only to be determined by careful study of the associated clinical phenomena. In simple dynamic pulsation the aorta may in thin persons frequently be felt to be somewhat dilated, especially during the paroxysm, but no distinct tumor formation can be recognized. The symptoms of neurasthenia or the stigmata of hysteria are present and these are often associated with gastro-intestinal symptoms. The throbbing is intense and distressing, sometimes diffused but never distinctly expansive. It can be felt when the patient is in the knee-elbow posture. The throbbing of anæmia is much less marked. Pulsation transmitted from the aorta through an overlying tumor communicates a lifting sensation to the hand upon palpation, is usually circumscribed, not expansile, and disappears when the patient is examined in the knee-elbow position, the mass falling away from the aorta under the action of gravity. The clinical phenomena of the primary condition are usually more or less well defined. Errors of diagnosis not infrequently occur under these circumstances, the tumor being mistaken for an aneurism. When well defined the pulsation of an abdominal aneurism is characteristic. If the aneurism be of large size there is dulness continuous with that of the left lobe of the liver. In thin persons a distinct tumor may be felt, the pulsation is expansile and forcible, and persistent rather than paroxysmal. A systolic murmur is very commonly heard in the absence of pressure of the stethoscope or the murmur may be audible in the back. In some cases a low-pitched soft diastolic murmur is heard. In many cases there is a distinct systolic thrill. Both the murmur and thrill may occur in other conditions which cause an abrupt narrowing in the lumen of the aorta, and may be produced by the pressure of the stethoscope. These signs are occasionally encountered in the epigastric pulsation of nervous diseases and in tumors of various kinds developing in relation with the abdominal aorta. The diagnosis of aneurism must therefore be made with extreme caution. It is justified in cases in which there is a distinct tumor with expansile pulsation persisting in the knee-elbow posture and when radiating pain, vomiting, and retardation of the femoral pulse are present. The pulsation of an abdominal aneurism may be manifest in the left hypochondrium or lumbar region. The X-rays furnish an important aid to diagnosis in doubtful cases. Epigastric pulsation must not be confounded with the purely subjective sensation of fluttering in the left hypochondrium of which hysterical women frequently complain. These two phenomena are entirely distinct, though they are frequently present in the same case.

RADIAL PULSE.

The pulse may be studied in any superficial artery. For this purpose the radial, because of its accessibility and convenience, is usually selected. This artery is palpated over the flat portion of the radius between the styloid process and the tendon of the radialis internus. In an anomalous distribution of the artery the radial pulse must be sought for elsewhere. It is a good plan to compare the pulse in the radials of both sides. It occasionally occurs that a small arterial twig occupies the usual position of the radial while the main branch has an anomalous course. In the absence of comparison with the other side an erroneous conclusion as to the volume and force of the pulse would be formed. In any case of doubt the pulse in the bend of the elbows or in the brachial or axillary arteries upon the two sides may be compared. Pathological differences in volume, force, and time, that is to say, retardation upon one side, are due to the interference with the flow of blood in the artery caused by endarteritis and aneurism, or the pressure of a tumor upon the wall of the vessel. Complete obliteration results from embolism or thrombosis. In traumatism from extensive crushing or laceration it is a sign of destruction of the artery. Retardation of the femoral pulse upon both sides may occur in aneurism of the thoracic or abdominal aorta. On one side it is commonly the sign of aneurism of the common iliac artery. Under certain circumstances it is convenient to study the pulse in the temporals, carotids, or even in the posterior tibials.

The best method of feeling the pulse consists in the application of the tips of three adjacent fingers, that of the index finger being, according to an old rule, nearest the heart of the patient. Under changing pressure the distention of the artery which constitutes the pulse is recognized and studied. The value of the pulse in diagnosis depends largely upon the experience and judgment of the physician. In the study of the pulse the following points require especial consideration; (a) condition of the arterial wall; (b) frequency; (c) rhythm; (d) volume; (e) celerity; (f) tension; (g) dicrotism.

The condition of the arterial wall enables us to form conclusions as to the presence or absence of general arteriosclerosis, and to recognize the modifications of the pulse-wave caused by changes in the elasticity of the artery. It is of much greater diagnostic importance than the pulse-rate. Empty the artery by pressure and roll it to and fro upon the underlying bone. In healthy individuals in early life the artery is felt as a strand of soft elastic tissue. In arteriosclerosis and in those conditions in which the blood-pressure is habitually high, such as chronic nephritis, gout, and lead poisoning, the increased resistance of the artery may be readily recognized. It feels like a whip-cord under the fingers. In advanced arteriosclerosis calcareous deposits in the wall of the artery—atheroma—can be distinctly felt, and in some cases these deposits are so coarse and irregular as to warrant their comparison with a string of wampum. Such arteries are often tortuous. These changes can be best recognized by passing the palpating finger gently along the course of the artery. Important as is the study of the condition of the walls of the peripheral arteries for the diagnosis of arteriosclerosis, it is nevertheless necessary to call attention to the fact that there are cases of very advanced sclerosis of the aorta and even of the coronaries, and indeed of other deeply situated vessels, in which the superficial arteries upon palpation yield no indication of changes in their walls. To arteriosclerosis, which is often unequally distributed, the radial shows no special liability. It is therefore necessary in suspected cases to examine carefully the superficial arteries in various parts of the body. Increased arterial tension and an accentuated aortic second sound are important signs of arteriosclerosis.

Frequency of the Pulse.—By this term is indicated the number of beats in a minute. It is convenient to count the radial pulse for 15 seconds and multiply the result by 4. If the pulse is irregular or extremely rapid it becomes necessary to count for an entire minute and to repeat the counting in order to avoid error. If after repeated observation wide variations in the frequency are found, the extremes may be recorded. Various devices have been suggested for the counting of very rapid pulses. If regular. every second or third beat may be counted and the result multiplied respectively by 2 or 3; or a dot for each beat may be made with a pencil upon a sheet of paper. These methods are liable to error, and variations in the pulse-frequency uncountable by ordinary methods, that is, exceeding 200, are without clinical importance.

The pulse-frequency is modified by a great variety of physiological influences. The pulse should therefore be counted regularly under similar conditions. When this is impracticable any circumstance liable to influence the frequency should be noted.

Mental excitement in nervous individuals exerts a marked influence upon the frequency of the pulse. The approach of the physician to the bedside or the entrance of the patient to the consulting room is often followed immediately by a rapid increase. It is therefore wise to postpone the taking of the pulse until after some general conversation sufficiently prolonged to enable the patient to regain his equanimity.

The effect of muscular effort in increasing the pulse-frequency is well known. Athletic sports, running, boxing, stair-climbing, and similar effort may be followed by a very rapid pulse-rate which is nevertheless physiological. During convalescence from disease and in feeble and delicate persons slight movements of the body increase the pulse-frequency, which falls again after a period of rest. If, however, the effort be prolonged the return to the normal frequency is delayed.

The pulse-rate is modified by the posture of the body. It rises immediately upon change from the recumbent to the sitting and again from the sitting to the standing position. The frequency attained immediately after these changes falls again in a little time but not to the normal of the previous posture. The pulse-rate for the same individual is relatively higher while each of these positions is maintained. The figures in healthy individuals, in the absence of other modifying conditions, are approximately in the recumbent posture 66, in the sitting 70, in the standing 80 beats per minute.

The pulse-frequency is increased during the digestion of food. Hearty meals and alcoholic beverages render the increase more marked. The diurnal modifications of the pulse bear a definite relation to the periods of taking food. They occur, however, in those who are fasting and bear some relation to the diurnal variations of the temperature. The pulse's frequency is to some extent modified by respiration, being slightly increased upon inspiration and diminished upon expiration. It is higher after paroxysms of cough. It varies greatly at different periods of life.

PULSE-FREQUENCY AT DIFFERENT AGES.—ROLLET.

At birth	144-133 per minute
To end of 1st year	143-123 per minute
10th to 15th year	91- 76 per minute
20th to 60th year	73- 69 per minute

PULSE-FREQUENCY IN CHILDHOOD.—VIERORDT.

0- 1 year	134 per minute
1- 2 years	110.6 per minute
2- 3 years	108 per minute
3- 4 years	108 per minute
4- 5 years	103 per minute
5- 6 years	98 per minute
6- 7 years	92.1 per minute
7- 8 years	94.9 per minute
8- 9 years	88.8 per minute
9-10 years	91.8 per minute
10-11 years	87.9 per minute
11-12 years	89.7 per minute
12-13 years	87.9 per minute
13-14 years	86.8 per minute

In general terms the frequency declines with advancing years. The pulse in women is about 7 beats per minute more rapid than in men of corresponding age. In large individuals it is slightly slower under similar conditions than in those of smaller size.

Cases are occasionally observed in which the radial pulse is less frequent than the impulse of the heart. This discrepancy arises in consequence of the feebleness of certain contractions of the heart, the pulse-wave not reaching the radials. Under these circumstances the pulse is commonly but not always irregular. In every case of irregularity of the pulse it is desirable to count the contractions of the heart as manifested in the precordial impulse.

In general, departures from the normal pulse-rate, either in the direction of increased or diminished frequency, arise in consequence of derangement of the nervous mechanism of the circulation. Increase may be due to paresis of the pneumogastric or irritation of the sympathetic nerves or the intracardiac ganglia; decrease to irritation of the pneumogastric or paresis of the cardiac sympathetic nerves and ganglia. Derangements of the pulse-rate arise in consequence of causes affecting the myocardium itself.

Increased Frequency—Rapid Heart.—Perhaps the most common cause of an increase in the pulse-rate is the action of the fever-producing toxins. We find it therefore in the febrile infections, the increase in the pulse-frequency bearing a general relation to the elevation of the temperature. The prognosis in severe febrile disease is more favorable where this parallelism is maintained than in those cases in which the pulse-rate is increased out of proportion to the rise of temperature; the very rapid pulse being the sign of special implication of the heart or vasomotor system. In the acute febrile diseases a pulse-rate of 140–160 in the adult, if maintained for any length of time, is of itself ominous. In children even higher pulse-rates are not uncommon in cases that run a favorable course. The effect of the specific toxins upon the mechanism of the circulation is by no means constant. A knowledge of the variations is of diagnostic importance in doubtful cases. In scarlet fever the pulse-rate is high—120–160—throughout the whole course of the attack; in diseases to which it bears some resemblance, such as angina tonsillaris, diphtheria, rubella, and measles, the pulse-rate of the period of invasion is slower. The pulse-rate in acute miliary tuberculosis and in septicopyæmic conditions is high, out of proportion to the temperature. In malignant endocarditis the pulse is rapid both during the febrile paroxysms and in their intervals. In puerperal sepsis a high pulse-rate is more constant than elevation of temperature. Increased pulse-frequency is common in the early stages of phthisis and usually persists throughout the whole course of the disease, alike in afebrile periods and when the temperature is moderate or excessive.

On the other hand the pulse-frequency of enteric fever is low in proportion to the temperature. In cases of average severity it frequently does not exceed 100–110 with a temperature range during the fastigium of 102° F. (38.9° C.) A.M. to 104° F. (40° C.) P.M. This fact is not without importance in the differential diagnosis between enteric fever and septic infections, the so-called typhoid form of malignant endocarditis and acute miliary tuberculosis. A very rapid pulse in enteric fever is usually the sign of an inflammatory complication or secondary infection.

A frequent pulse occurs in acute affections of the heart, endocarditis, pericarditis, and myocarditis, and in chronic valvular disease in the stage of failure of compensation. Increased pulse-frequency after slight exertion occurs in most forms of chronic myocarditis, in general muscular asthenia, in anæmia, during convalescence from acute diseases, and in conditions of the neighboring organs which subject the heart to abnormal pressure, as pleural effusion, thoracic aneurism, massive enlargement of the liver and spleen, tympany, and ascites. The frequency of the pulse is increased in cardiac palpitation from any cause.

The pulse-frequency is greatly increased in many nervous diseases. A rapid pulse with subnormal temperature is characteristic of shock and collapse. Acceleration of the pulse is a constant symptom of exophthalmic goitre; during the paroxysms of palpitation the pulse is often uncountable. In neurasthenia, Addison's disease, the primary and secondary anæmias, arthritis deformans, and locomotor ataxia the pulse-frequency is likewise habitually increased. In these conditions the rapidity of the pulse may be continuous or show itself only after moderate exertion. In general terms it is proportionate to the severity of the disease. Pain often causes increase in the pulse-rate. Exceptionally slowness of the pulse occurs in connection with very intense pain. In either case the derangement is reflex.

Excesses in alcohol, tobacco, coffee and tea, disorders of digestion, lack of sleep, other exhausting influences, and lowered blood-pressure not rarely produce abnormal pulse-frequency. Certain drugs, as atropine, have the same effect.

Tachycardia—Pycnocardia—Heart Hurry.—The extreme rapidity which follows violent exercise or fright may persist for days or weeks; the rate may reach 160–220. The condition may occur as a pure neurosis. Palpitation and dyspnœa are not always present. The patient is often able to attend to his ordinary duties. Tachycardia is one of the symptoms of the neurasthenic at the menopause and has been attributed to reflex irritation from ovarian or uterine disease. This symptom may be due to lesions such as a tumor or clot in or about the medulla or pressure upon the pneumogastrics.

Paroxysmal tachycardia is a neurosis characterized by attacks of greatly increased action of the heart occurring at irregular intervals and without obvious cause. The attacks usually begin abruptly and are of varying duration, frequently not exceeding an hour or two. The pulse-rate exceeds 200 and is sometimes uncountable. Subjective symptoms may be absent. In many of the cases there is much distress and oppression.

Diminished Frequency—Slow Heart.—In many cases the normal pulse-rate does not exceed 60. In some individuals the pulse may be slow under conditions in which in others it is rapid. This is often the case during the period of convalescence from pneumonia, enteric fever, rheumatic fever, and diphtheria. The pulse is slow while the patient is at rest but is accelerated by slight exertion. It is the slow pulse of exhaustion and occurs in young persons and at the close of uncomplicated cases. Transient slowing of the pulse is a postcritical symptom in certain febrile diseases, as pneumonia. If the pulse-frequency remains high during an abrupt fall of temperature in the course of croupous pneumonia, pseudo-crisis is to be thought of. Slow pulse is encountered in chronic gastritis and ulcer and cancer of the stomach. It occurs in emphysema but is not common in other affections of the respiratory system. It is not rare in aortic stenosis but is infrequent in other valvular diseases of the heart. It is an occasional but by no means constant symptom in chronic myocarditis. Toxic agents, as lead, alcohol, tobacco, coffee, digitalis, and opium, produce slowing of the pulse, and it occurs in some cases of primary and secondary anæmia, diabetes, and myxœdema, especially while the patient is at rest.

Bradycardia—Brachycardia.—The pulse-rate falls as low as 40 and may be persistently slow. It is important to see that the arterial pulse corresponds in frequency with the cardiac contractions. Bradycardia may be physiological or pathological. In rare instances it is a peculiarity of normal individuals. During labor, whether premature or at term, the pulse may fall to 40 or below it. Slow pulse is one of the symptoms of hunger and exhaustion. Cachectic individuals have usually not only subnormal temperature but also a slow pulse-rate. Slowing of the pulse occurs in gall-stone colic, in renal and hepatic colic, and in lead colic. It is associated with acute but not necessarily with chronic jaundice. Either the circulatory mechanism becomes habituated to the bile intoxication or the bile salts are diminished in amount. Bradycardia occasionally occurs in disease of the genito-urinary tract, especially in nephritis and in uræmia. It is of special diagnostic importance in acute cerebral disease associated with intracranial pressure. It occurs in various forms of meningitis, especially tuberculous meningitis, in which considerable elevation of temperature is sometimes associated with a slow pulse. Chronic cerebral compression, such as results from tumor or hydrocephalus, is not attended with bradycardia except during acute exacerbations. Apoplexy, the postepileptic state, disease of the medulla and diseases and injuries of the cervical cord may be associated with a very slow pulse. Bradycardia occurs in general paresis, mania, and melancholia. It constitutes the essential sign of heart block. A very slow pulse is occasionally associated with shock and may follow the rapid evacuation of large peritoneal or pleuritic effusion. Certain drugs, as opium and digitalis, cause slow pulse.

Rhythm.—Under normal conditions the pulse is regular or rhythmic, that is to say, the individual pulse-waves are of like volume and follow one another at equal intervals of time. Physiological derangements of rhythm are slight and transient and occur under those physiological conditions which are attended by changes in the pulse-frequency. Marked disturbances of rhythm—*arrhythmia*—are always pathological and have their source either in functional derangements of the heart or demonstrable lesions of that organ.

The causes of the various disturbances of rhythm are, (a) psychic or emotional, (b) organic cerebral disease, as endarteritis, hemorrhage, concussion, or compression, (c) reflex, such as produce the cardiac irregularity in gastro-intestinal derangements and diseases of the liver, kidneys, or genito-urinary organs, (d) toxic, the common agents being tea, coffee, tobacco, and alcohol, and finally (e) changes in the heart itself.

Arrhythmia. The following types are to be considered:

1. **Respiratory arrhythmia: Sinus Irregularity of Mackenzie; Pulsus Respiratorius.**—Variation in the length of the diastolic period is the chief characteristic, the systolic period remaining constant. The normal inspiratory increase and expiratory decrease of the cardiac action are exaggerated. It is easily recognized, pulse beats of equal strength but frequency continuously changing with the phases of the respiration being recognized by the finger and upon auscultation. With marked slowing of the inspiration, the pulse shows this irregularity with great constancy. This form of arrhythmia is of vagus origin and is attributed to an exaggeration of the normal respiratory reflex. Jugular tracings show that the right auricle and ventricle contract with the same irregular rhythm as the radial pulse. It has been observed in infants, healthy adolescents and less frequently in healthy adults and is not uncommon in the convalescence from the acute febrile infections, in cerebral disease, especially tuberculous meningitis, neurasthenia and anæmic states. It is of no great diagnostic significance.

2. **Extrasystole; Pulsus Extrasystolicus.**—According to Mackenzie this term should be limited to "premature contractions of the auricle or ventricle in response to a stimulus from some abnormal point of the heart, but when otherwise the fundamental or sinus rhythm of the heart is maintained." As the primary automatic stimulus for contraction arises normally in the remains of the sinus venosus at the mouths of the great veins—sino-auricular node—and since this primitive cardiac tube is represented in the auricle—atrio-ventricular node—and in the bundle of His and its extension to the ventricles, and each section of the heart is capable of independent automatism, the following forms of extrasystole occur:

a. Ventricular Extrasystole.—The origin of the extrasystole is assumed to be in the atrio-ventricular bundle beyond the atrio-ventricular node. The ventricle contracts in advance of its normal time. This contraction is followed by the usual refractory period during which the ventricle fails to react to the normal stimulus from the sino-auricular node with the occurrence of a compensatory pause which is longer than the normal diastolic pause by so much as to make up the loss in duration of the preceding diastole and is followed by a forcible postcompensatory systole in response to the succeeding rhythmic stimulus from the sino-auricular node. It is characteristic of the ventricular extrasystole that the compensatory pause plus the diastole preceding the extrasystole = two normal diastoles.

b. Auricular Extrasystole.—The stimulus arises in the remains of the primitive cardiac tube incorporated in the auricle below the level of the sinus. The premature auricular contraction is followed by ventricular contraction and a long pause after the extrasystole due to the fact that the normal rhythmic stimulus from the sinus occurs during the refractory period of the auricle. The auricle and ventricle are quiescent until the occurrence of the next sinus stimulus. In auricular extrasystole the pause is not usually fully compensatory. The sounds of the heart and the radial pulse do not differ from those of ventricular extrasystole from which auricular extrasystole can only be differentiated by simultaneous tracings of the jugular pulse.

c. Auricular Ventricular Extrasystoles; Nodal Extrasystoles. —This form of extrasystole is attributed by Mackenzie to an abnormal stimulus originating in the auriculo-ventricular node of Tawara and causing premature and simultaneous contractions of auricles and ventricles. Other observers regard it as a ventricular extrasystole in which the stimulus has passed back and prematurely stimulated the auricle—"a retrograde auricular systole."

Extrasystoles constitute the most common causes of arrhythmia. They may occur at regular or irregular intervals, during more or less prolonged periods or continuously, and these three forms of extrasystole may be present in varying combination or in association with other forms of arrhythmia. They are attributed to an undue excitability of the remains of the primitive cardiac tick. Most patients experience no subjective symptoms in connection with the occurrence of extrasystoles. When, however, their attention is called to the irregularity, they are often unnecessarily annoyed by various precordial sensations described as "fluttering," "heart dropping" or the "strong beat" which often follows the long pause. Extrasystoles are of common occurrence in neurotic individuals under various circumstances, especially excitement or exertion, in advanced life, at puberty, the climacteric and during pregnancy; in habitual over-indulgence in tobacco, tea, coffee or alcohol; in the convalescence from the acute febrile infections and in arterio- and cardio-sclerosis. They are essentially of minor clinical importance but the conditions of which they are symptomatic are often grave. Pulsus bigeminus is that form of irregularity in which every second beat is an extrasystole and is usually smaller than the normal beat. The smaller beat is invariably followed by a pause longer than the pause preceding it. The extrasystole may appear after every 1, 2, 3, 4 or more normal beats and on account of the long compensatory pause is spoken of as an intermission or the "dropping of a beat."

3. **Nodal Rhythm—Continuous Irregularity; Rhythm of Auricular Fibrillation—Ventricular Rhythm; Pulsus Irregularis Perpetuus.**—According to Mackenzie the automatic impulse starts at the auriculo-ventricular node and the auricles and ventricles contract nearly at the same time; according to Cushny the arrhythmia is due to fibrillation of the auricle which ceases to contract as a whole. The condition is characterized by extreme and continuous irregularity of the action of the heart—perpetual irregularity; delirium cordis—by the absence of the sinus rhythm, the presence of a ventricular or positive venous pulse and the absence of an auricular wave in the jugular tracing. The nodal rhythm is present in the majority of cases of severe heart failure and in a great many the immediate breakdown is directly attributed to the inception by the heart of this abnormal rhythm. (Mackenzie.) The heart's action may be (a) not greatly increased and the evidences of cardiac insufficiency may not be marked for a long time, or (b) it may be greatly increased. Sensations of fluttering in the left chest and rapidly developing symptoms of cardiac insufficiency occur. The pulse is small, rapid and except when extremely rapid very irregular in tachycardia; and (c) the nodal rhythm is transient and recurrent; see Paroxysmal Tachycardia. The significance of the nodal rhythm is grave. Improvement in the underlying cardiac condition may occur under rest and the administration of digitalis, but it is transient and exertion is almost always followed by a recurrence of the symptoms.

4. **Partial or Complete Heart Block; Ventricular Rhythm; Pulsus Transmissionus.**—This form of arrhythmia is attributed to partial or complete impairment of the conductivity of the bundle of His. The ventricle fails to contract after the auricular contraction. The stimulus may be delayed or at times prevented from passing over, or finally it may be completely blocked beyond the auricular ventricular node and the contraction of the ventricle then arises in response to a stimulus originating in the functionating remains of the bundle of His—Heart Block; Ventricular Rhythm. See p. 1235.

5. **Pulsus Alternans; Exhaustion of Contractility.**—This form of arrhythmia is characterized by an alternating succession of large and small beats, the rate of the heart remaining perfectly regular. It indicates weakness of the heart muscle. The large contraction encroaches upon the period of rest so that contractility has not sufficiently recovered to fully react to the next stimulus, hence the smaller beat due to diminished energy of the heart muscle at the moment. The smaller contraction is in turn followed by a longer period of rest and a correspondingly larger pulse beat. Pulsus alternans is not to be confounded with extrasystole.

Volume.—The volume is the size of the artery under the influence of the pulse-wave. It depends upon the degree of relaxation of the muscular coat. If the expansion is marked the volume is correspondingly great and the pulse is said to be large or full—*pulsus magnus*. If the expansion is slight the pulse is said to be small—*pulsus parvus*. The large pulse is commonly a pulse of low tension. The small pulse varies in tension. If low it is the sign of feeble action of the heart or diminished amount of blood. The pulse is small and of low tension in valvular disease of the heart with ruptured compensation, and small and usually of good tension in aortic and mitral stenosis.

Celerity.—There is an important distinction between the frequency of the pulse, that is, the number of beats in a minute, and the mode in which the pulse-wave develops under the finger. The pulse is said to be quick—*pulsus celer*—when it is characterized by a wave of rapid ascent and equally rapid recedence. The quick pulse is a pulse of low tension. It is encountered when the peripheral vessels are relaxed, as in the fevers and in various forms of anæmia. Celerity is characteristic of the water-hammer pulse of aortic insufficiency. This pulse occurs also in consequence of the extreme relaxation of the peripheral arteries in many cases of neurasthenia. In these conditions there is often a visible pulsation in the superficial arteries associated with capillary and, in some instances, with venous pulsation.

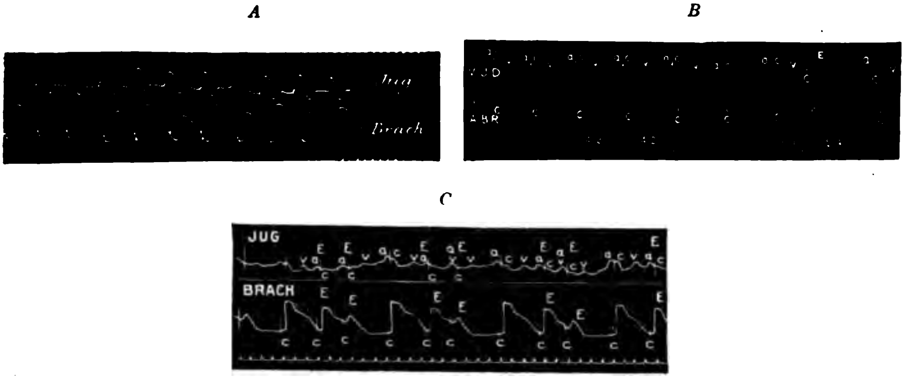


Fig. 198.—*A*, alternating pulse of a case of paroxysmal tachycardia; *B*, pulsus bigeminus. Tracing from jugular vein (V.J.D.) and brachial artery (A.B.R.) showing ventricular extrasystoles; *C*, pulsus trigeminus. Tracing from jugular vein and brachial artery showing two auricular extrasystoles (E,E) after regular systole.

The tardy pulse—*pulsus tardus*—is characterized by the gradual rise and equally gradual descent of the pulse-wave. It is a pulse of high tension and is encountered in arteriosclerosis, advanced age, chronic interstitial nephritis, and in some instances during the attacks of angina pectoris. The pulse in aortic stenosis and in arteries peripheral to an aneurism is commonly tardy.

Tension.—This term includes those qualities of the pulse which indicate the arterial blood-pressure. On the one hand the adjectives hard and tense are sometimes used interchangeably in regard to the pulse,—*pulsus durus*,—while on the other hand the adjective soft is used synonymously with relaxed—*pulsus mollis*. The clinician must, however, be constantly on his guard against confounding rigidity of the arterial wall with intra-arterial tension or blood-pressure. It is important also to distinguish between the tension corresponding to the ventricular systole and that corresponding to the ventricular diastole.

High tension occurs in chronic interstitial nephritis, gout, lead poisoning, and in the diabetes of advanced age. The pulse is small and tense in the early stages of acute peritonitis. The pulse tension is increased in pregnancy and in some forms of anæmia.

The pulse of low tension is soft and compressible. It is a sign of cardiac and general asthenia and occurs in all forms of depression and exhaustion. The pulse of obese persons is very often of low tension. Temporary diminution of arterial tension may follow hot drinks, alcoholic beverages, the hot bath, and accompany the period of reaction following great physical exertion or mental excitement. The statement is very often made that arterial tension increases with age. The clinician will do well to recognize the distinction between histological changes in the arterial walls and increase of intra-arterial pressure. When the diastolic pressure is relatively high and the artery remains well filled between the beats, the pulse is said to be full—*pulsus plenus.* When the pulse-wave is very full and quick and the vessels are soft and compressible, the pulse is sometimes spoken of as gaseous. When the artery is collapsed between the beats, the pulse is said to be empty—*pulsus vacuus, vel inanis.* When the pulse-wave is very small and the artery relaxed, the pulse is described as thready, running, or undulatory.

The blood-pressure—arterial tension—may be estimated by the fingers; but more exact and definite measurements are made by various forms of sphygmomanometers.

Dicrotism.—The occurrence of a secondary pulse-wave in each arterial beat is commonly shown in normal sphygmographic tracings. It is recognized by the finger only when fairly well marked. The conditions which favor dicrotism are: diminished arterial tension, relaxed capillaries,—both of which are due to diminished vasomotor tonicity,—a sudden forcible ventricular systole, and relaxation of the arterial walls. Dicrotism may be occasionally recognized by the finger in persons apparently in good health. Such individuals usually manifest a high degree of vasomotor instability, are easily fatigued and bear acute illness badly. The clinical condition in which dicrotism is most marked is fever. It is usually well developed in enteric fever from the beginning of the second week.

OTHER ANOMALIES OF THE PULSE.

The pulse should be examined not only in both radials but, under certain circumstances, in the superficial arteries elsewhere. Retardation, smallness, feebleness, or obliteration of the pulse on one side of the body or locally may be caused by deviations from normal anatomical standards or by traumatism, embolism, thrombosis, tumor pressure, and aneurism.

If the pulse be relatively feeble or small, or if it be absent *in the right radial,* it may indicate an aneurism of the ascending aorta or innominate; *in the left radial,* an aneurism of the transverse or descending portion of the arch; *in a radial of either side* it may indicate the presence on the same side of embolism, thrombosis, aneurism of the subclavian, axillary, or brachial arteries, cervical or axillary tumors exerting pressure upon the vessel, and if slight in degree may be suggestive of pneumothorax or large pleural effusion; *in one femoral, popliteal, or posterior tibial artery* the interference of the circulation may be due to aneurism, tumor pressure, embolism, or thrombosis; *in these vessels on both sides,* to abdominal aneurism or congenital obliteration of the aorta. Absence of pulse in the femorals is an occasional sign of aneurism of the abdominal aorta.

The Pulse in Different Conditions of the Heart—MYOCARDITIS.—In the various forms of sclerosis the pulse is usually feeble. It is sometimes, but not invariably, irregular. It is commonly slow, and not infrequently bradycardia is present, the pulse falling as low as 30 or 40 per minute. In fatty heart the pulse may show the same characteristic. Extreme fatty changes occur, however, without modification of the pulse, which may remain regular and of moderate strength.

MITRAL STENOSIS.—In the early stages the frequency of the pulse is not increased. It is small and rather tardy, the artery not well filled, the successive beats irregular in time and volume.

MITRAL INCOMPETENCE.—The modifications depend upon the extent of the lesion and the condition of the left ventricle. The frequency is increased, the volume and tension are diminished, the rhythm usually, but by no means constantly, irregular.

AORTIC STENOSIS.—In uncomplicated cases the pulse is slow, its volume diminished, its tension maintained, the artery being well filled during the ventricular diastole. The pulse of aortic stenosis is usually regular.

AORTIC INCOMPETENCE.—The large mass of blood propelled into the aorta by the dilated and hypertrophied left ventricle causes sudden distention of the arterial system, which is followed by an equally sudden collapse resulting from the failure of the base of support to the column of blood normally supplied by the aortic valves; characteristic and striking changes in the arterial pulse result, and are manifested in all the superficial arteries. The pulse is increased in frequency and usually regular. The artery is suddenly distended, the pulse being quick, jerking, visible, and abruptly receding. The pulse is locomotor, that is to say, the visible arteries are elongated and their curves accentuated. The jerking, visible, and collapsible character of the pulse, as observed in the arm and wrist, is intensified by elevating the member above the head. Very often there is pulsation at the root of the neck, and in well-developed cases there is distinct pulsation of the tissues of the hands and feet—capillary pulse—which may be felt by gently grasping the hand or foot, placing the thumb upon the palmar or plantar surface and the fingers upon the dorsal surface. With the ophthalmoscope pulsation of the retinal arteries may be seen. Capillary and the so-called penetrating venous pulsation also occur.

The pulse of aortic incompetence is frequently called the Corrigan pulse, after Sir Dominic Corrigan, who first systematically studied and described its peculiarities.

ARTERIOSCLEROSIS.—Modifications of the pulse in arteriosclerosis depend upon the peripheral resistance and the force of the ventricular systole. Sclerosis and high tension are usually associated. The pulse-wave is tardy, sustained, and subsides slowly, the vessel remaining full between the beats—diastolic tension. Pressure of the finger does not readily obliterate the pulse. An effort must be made to discriminate between the firmness due to intra-arterial tension and that due to thickening of the arterial wall. If, when the pulse-wave is arrested by the pressure of the finger, the artery can be felt beyond the point of compression, its walls are thickened.

CAPILLARY PULSE.

Normally the pulse-waves penetrate to the smallest arteries but are lost in the capillaries. Under certain circumstances, however, the pulse is manifest in the capillaries and shows itself upon inspection as a pulsatile flushing and fading of the surface. Conditions which especially favor the development of the capillary pulse are relaxation of the peripheral circulation and rapid discharge of a large amount of blood from the ventricle into the arterial system—*pulsus celer*. Capillary pulse is occasionally seen in areas of local hyperæmia and inflammation, as in whitlow, and patients often recognize this increased pulsation in the throbbing character of the pain. Of greater interest is the capillary pulse of aortic incompetence. It is seen in the pulsatile changing in the color intensity of the nail-bed—*subungual pulse*—a phenomenon which becomes more marked when the nail is slightly pressed near the edge so that the underlying tissue is momentarily pale. The border line between the pink and white advances and recedes with each cardiac revolution. The capillary pulse can be distinguished in the ear, lips, cheek, and especially distinctly upon the forehead at a point where it is reddened by light friction. The capillary pulse shows clearly through a piece of glass pressed upon the mucous surface of the everted lip. It is, however, not confined to aortic regurgitation, but occurs also in pyrexia, chlorosis, and other forms of anæmia, neurasthenia, and exophthalmic goitre. The capillary pulse of aortic incompetency is more marked in stages in which the compensation is good and disappears upon the failure of compensation.

VENOUS PULSE.

The modifications of intrathoracic pressure caused by respiration are of importance in connection with the venous circulation. Inspiration hastens, expiration retards the flow of blood in the veins. These modifications are not seen upon ordinary quiet breathing. On the other hand, in forced respiration and dyspnœa there is expiratory venous distention and inspiratory venous collapse, best seen in the veins of the neck, especially when they have been enlarged by previous congestion and are therefore more distinctly seen. Even more marked is the influence of the variations of intrathoracic pressure by violent cough and prolonged muscular effort. In individuals in whom these paroxysms of intravenous pressure recur through long periods, as in those who suffer from chronic paroxysmal cough, the veins, especially the jugular, become permanently enlarged, so that during the paroxysms the patient is not only cyanosed but manifests a distinct distention of the vessels at the root of the neck. The jugular bulb sometimes appears as a small, sausage-like swelling in the region of the insertions of the sternocleidomastoid muscle.

More rarely distention of the veins takes place during inspiration; retraction upon expiration. This reversal of the ordinary conditions is the result of mechanical interference by pressure or traction upon the great veins within the thorax, such as occurs in chronic mediastinitis, mediastinal tumors or a large pericardial or pleural effusion,—conditions in which Kussmaul's pulsus paradoxus is frequently observed.

The Forms of Venous Pulse.—Pulsation in the veins is due to the movements of the heart and has the cardiac rhythm. A pulsation communicated by the underlying carotids is sometimes seen in the external jugular veins. This is the so-called false venous pulse. The distinction between this and true venous pulsation is usually unattended with difficulty. The more extended superficial pulsation due to the greater width of the vein and the peculiar, prolonged, undulatory movement which is characteristic of the low intravenous tension are of importance. Upon light palpation the pulse is feeble and compressible and in strong contrast to that elicited upon palpation of the underlying artery. Upon compression of the vein the peripheral pulsation continues or may be increased in consequence of the distention; upon compression of the artery at the root of the neck the pulsation wholly disappears.

True Venous Pulse.—Three forms are encountered: the physiological or negative venous pulse, the regurgitant or positive venous pulse, and the penetrating or positive centripetal venous pulse.

(a) NEGATIVE VENOUS PULSE.—This form of pulsation in the veins is called normal or physiological because it is constantly seen in the exposed veins of animals and frequently in the jugulars of human beings in health. It is not observed in all persons, simply because the jugular veins in many individuals are difficult or impossible to distinguish. It is very obvious upon inspection in those persons in whom the veins are distended and plainly visible. The physiological venous pulse is readily distinguished from positive or regurgitant venous pulsation by compression of the vein with the finger. The pulsation peripheral to the point of compression ceases, and that central to it likewise disappears or becomes much fainter. The diminution or complete disappearance in the latter case makes it evident that the pulse-waves are not transmitted to the blood in the veins by the cardiac systole. It is thus apparent that the continuous blood stream from the veins is rhythmically restrained and hastened by the action of the heart. The negative venous pulse is observed in the external and internal jugulars. It is presystolic in time. The collapse of the vein at the time of the ventricular systole is attributed to the negative intrathoracic pressure caused by the diminution in the size of the heart at that moment in its revolution. During the ventricular diastole the aspiration influence ceases and the blood accumulates in the veins. It may be urged against this statement, however, that the venous pulse, central to the point of compression, though always reduced, does not in all cases wholly disappear; but the external jugular cannot be entirely emptied of blood, as there are tributary veins central to the point of compression. In some cases this form of venous pulsation can be still further reduced by simultaneous compression of the subclavian. In order to determine the time of the venous pulse, which is diastolic,—presystolic,—it must be compared with the carotid pulse, which is, of course, systolic. The negative venous pulse is without diagnostic significance. A knowledge of it is necessary, however, in order that it may be differentiated from the form about to be described.

(b) POSITIVE OR REGURGITANT VENOUS PULSE.—This form of venous pulsation is observed in tricuspid incompetence. During the ventricular systole the blood regurgitates into the right auricle and the pulse-wave is transmitted to the vein. The pulsation is presystolic-systolic rather than purely systolic, as in the case of the arterial pulse. When the valve in the jugular is competent the pulsation is more marked in the bulb, but it does not always cease at the level of the valve even when competent. The regurgitation is interrupted, but a positive pulse-wave of similar form, though weaker, is induced in the blood which accumulates above the valve. In some instances the closure of the valve under the influence of the regurgitant blood wave gives rise to a sound distinctly audible upon auscultation. In the majority of instances, as a result of the over-distention of the veins, the valve becomes insufficient, so that the positive pulse is equally perceptible over the upper portion of the jugular. The distinction between the positive and negative venous pulse rests upon the correspondence of the former with the carotid pulse and its persistence in the pulsating vein below the point of compression. As a rule, positive venous pulsation is observed only in the jugulars.

This form of pulsation is a sign of tricuspid incompetence. It has, however, been observed in two extremely rare conditions in which the lesions likewise favor the transmission of the systolic pulse-wave to the jugular veins, namely, mitral incompetence with persistent foramen ovale and aneurism of the aorta communicating with the descending vena cava.

The patient should be examined in the recumbent posture and during very quiet breathing. Before making compression in the course of the vein the finger-nail should be placed upon the vein at the root of the neck and lightly drawn upward to empty the vessel. In the absence of regurgitation the vein refills slowly, but if the tricuspid valves be incompetent the vein quickly refills from below and again pulsates.

Pulsation of the Liver.—In advanced cases of tricuspid incompetence the liver becomes enlarged and the hepatic veins dilated and engorged. In this condition the organ pulsates, the regurgitant wave being transmitted through the inferior vena cava. The pulsation may be recognized upon palpation, one hand being placed over the cartilages of the lower ribs to the right of the ensiform cartilage, and the other upon the side at the costal margin. An expansive pulsation of the entire organ can be felt with each cardiac impulse. In marked instances liver pulsation may be made out upon inspection. Pulsation of the liver must be distinguished from the jogging of the organ by a powerfully acting hypertrophied heart. It must also be distinguished from the epigastric pulsation of the abdominal aorta—dynamic pulsation—and from aneurismal pulsation. In very rare cases of aortic regurgitation, with good compensation and no sign of tricuspid incompetence, an arterial liver pulse has been noted, and local pulsation with double murmur has been observed in acute cholangeitis.

(c) PENETRATING OR POSITIVE CENTRIPETAL VENOUS PULSE.—This rare phenomenon is due to the fact that under certain conditions the pulse-wave is not lost in the capillaries but transmitted through them to the smaller veins. It has the same significance as the capillary pulse and occurs in cases of aortic incompetence or neurasthenia with great vasomotor relaxation. It has been observed in cases in which the capillary pulse has been faintly perceptible or absent altogether. It is associated with quick arterial pulse of large volume and is manifest not in the jugulars but in the small veins of the extremities, and disappears upon compression in the central, not in the peripheral, portion of the compressed vein.

In this connection diastolic collapse of the cervical veins, the so-called Friedreich's sign, may be mentioned. This sign occurs in chronic adhesive pericarditis but is of no great diagnostic value. The collapse of the veins is due to diastolic intrathoracic aspiration. The mechanism is the reverse of that in the physiological venous pulse.

VII.

THE DIGESTIVE SYSTEM: MOUTH; LIPS; TEETH; GUMS; TONGUE.

THE MOUTH.

The most important method of examination is inspection. The patient should be placed in a good light. The illumination is more satisfactory by light reflected from a head mirror. The mouth should be opened widely and, according to the part to be examined, the tongue should be protruded, drawn back, or moved from side to side. The soft palate and pharynx are best seen upon depression of the base of the unprotruded tongue with a spatula or the handle of a spoon. These instruments, if introduced too far, cause gagging. The examination of the posterior wall of the pharynx is facilitated when the patient pronounces the broad *a*, thus elevating the soft palate. In conditions of delirium or unconsciousness and in insane patients the examination of the mouth is often attended with great difficulty. In some instances holding the nose will cause the patient to open his mouth; in others, if necessary, the patient must be anæsthetized. In the case of children the examination is best conducted when the physician and mother or nurse sit viz-a-viz, the body of the child resting upon the knees of the latter, who holds his hands, the head upon the lap of the former, who opens the mouth and depresses the tongue with the spatula. The pharynx is best seen at the beginning of gagging. Palpation by means of the finger is very useful in detecting the presence and location of foreign bodies, the existence of retropharyngeal abscess, and especially adenoid vegetations in the nasopharynx and other similar conditions. This manœuvre, which is very annoying to the patient, must be executed rapidly. In the case of unruly patients or children the danger of being bitten is not to be overlooked. Against this accident a guard or shield may be employed, or the physician may, with the thumb and finger of the free hand, press the cheeks of the patient between the separated molars.

THE LIPS.

The lips are thick and coarse in habitual mouth-breathers, in cretinism, and in myxœdema. They are parted in conditions of great prostration and habitually in idiots and in some forms of insanity. They are pallid in anæmia and, like the nail-beds, early show cyanosis and the variations in its intensity. The lips are apt to be dry in dyspnœa and in obstruction to the nasal breathing. Dryness of the lips is associated with a diminution or perversion of the oral secretion, as in stomatitis, glossitis, and tonsillitis. The lips and mouth are dry and the latter open in the soporose condition preceding dissolution. There is drooling in dentition, mercurial salivation, diphtheritic paralysis, bulbar palsy, and idiocy. In these conditions the lips are apt to be loose and pendulous. Tremor or twitching of the lips occurs under intense emotion or may be a symptom of nervous disease. Convulsive retraction of the upper lip occasionally occurs as a sign of intense abdominal pain. Of great diagnostic importance is the occasional presence upon the lips of aphthous ulceration, mucous patches, sordes, rhagades—linear clefts or ulcerations at the corners of the mouth—or the scars resulting from them. The last, occurring in young children, are suggestive of hereditary syphilis. Herpes labialis is common in certain individuals in feverish colds, and occurs with such frequency in pneumonia, ague, and cerebrospinal fever as to have diagnostic value. It is very rare in enteric fever. This vesicular eruption develops rapidly upon an inflammatory base as a single lesion or in groups, most commonly upon the outer border of the lip, occasionally on other parts of the face, as the nose, the cheeks, or the ear. These positions are indicated by qualifying adjectives, as *herpes labialis*, *facialis*, *nasalis*, and the like. The contents of the vesicles are at first lymphoid, later purulent and scanty. Their efflorescence is attended by annoying burning or itching. They rapidly undergo desiccation with the formation of thick, tightly adherent scabs. The whole process is of short duration.

In paralysis of the seventh nerve the angle of the mouth on the affected side is lowered. In drinking, the liquid is apt to escape. In smiles or laughter the corner of the mouth is immobile and in attempts to show the upper teeth it is not raised. The mouth and lips are drawn toward the sound side. The labial sounds may not be fully formed. It is important to note that the displacement of the angle of the mouth may be due to loss of the teeth upon the opposite side or to retraction as the result of scar formation.

The lips are extremely sensitive and abscesses and acute inflammatory processes are attended with great pain. They are sometimes the seat of carbuncles. They undergo extensive necrosis in *cancrum oris*. The lip may be lacerated in the epileptic convulsion, but this is not common. It may be the seat of angioneurotic œdema or may be greatly swollen in consequence of the bites of insects. The lip is occasionally the seat of the initial lesion of syphilis. It shows more or less extensive superficial necrosis extending out upon the chin or cheeks after the taking of corrosive poisons and especially in carbolic acid poisoning. Epithelioma of the lip is common. It shows itself as an irregularly circular or oval ulcer with a swollen, infiltrated base, usually upon the lower lip, developing from a fissure or wart. At the beginning there are alternations of scab formation and open ulceration. After a time the submaxillary lymphatics become involved.

The differential diagnosis between chancre and epithelioma of the lip is usually unattended with difficulty. The chancre occurs, as a rule, early in life, epithelioma late. In chancre the lymphatics are involved early; in epithelioma late. Chancre is commonly circumscribed and densely indurated; epithelioma tends to spread and the induration is less dense. Healing of the chancre is progressive, especially under treatment; in epithelioma there is a tendency to extend, with alternations of scab formation and ulceration. In the former, constitutional symptoms and secondary rashes occur.

THE TEETH.

The teeth are of diagnostic interest. The time of their eruption and shedding in children and their state of preservation in adults are to be considered. Dentition and teething are terms used to describe the cutting of the teeth.

The First Dentition.—The temporary or deciduous teeth—the so-called milk teeth—are twenty in number; in each jaw two central incisors, two lateral incisors, two canines, two first molars and two second molars. They appear with considerable regularity as to order and time. Their eruption usually takes place in groups of four.

The first group—the lower and upper central incisors, 6 to 9 months. An interval of 1 to 3 months.

The second group—the upper and lower lateral incisors, 8 to 12 months. An interval of 1 to 3 months.

The third group—the four anterior molars, 12 to 15 months. An interval to the 18th month.

The fourth group—the four canines, 18 to 24 months. An interval of 2 to 3 months.

The fifth group—the four posterior molars, 24 to 30 months.

Healthy children usually have from four to eight teeth before they are a year old, and cut their first molars between a year and a year and a half, the canines before the end of the second year, and should complete dentition by the cutting of the second molars before the middle of the third year. The first teeth are usually the lower central incisors. The upper lateral incisors as a rule appear before the lower; the upper first molars usually precede the lower and not infrequently appear at about the same time with the lower lateral incisors.

Precocious dentition occasionally occurs. It is of no special significance. Delayed dentition occurs as the result of malnutrition either from improper feeding or disease. It is especially common in rickets.

The eruption of the teeth in healthy, well-nourished children commonly takes place without constitutional disturbance. At most transitory loss of appetite, fretfulness, disturbed sleep, a slight rise of temperature, 100–102° F. (37.7–38.8° C.), and derangement of the bowels are observed. In feeble and poorly nourished infants, especially in neurotic families, the perturbations caused by dentition may be more severe, the foregoing symptoms being aggravated and the temperature reaching higher levels, 103–104° F. (39.4–40° C.). The accidental coincidence of gastro-intestinal derangements, tonsillitis, laryngitis, and bronchial catarrh is very common, and the physician must be on his guard not to ascribe to dentition symptoms which are due to other causes. On the other hand there is danger that reflex symptoms due to the irritation of dentition will be erroneously interpreted. For example, annoying spasmodic cough, without fever, other constitutional disturbance or râles, and manifestly reflex, frequently accompanies the eruption of each group of teeth. Dentition may be the exciting cause of general convulsions in feeble, badly-nourished, rhachitic, or neurotic children. The process rarely causes eclampsia in well-nourished healthy babies. Tension, tumefaction, tenderness of the gums, and the bluish-red hue of deep congestion are indications for the use of the lancet.

The Second Dentition.—The permanent teeth in each jaw consist of two central and two lateral incisors, two canines, four bicuspids, and six molars. Their eruption takes place in the following order:

Anterior molars,	sixth to seventh year.
Central incisors,	seventh to eighth year.
Lateral incisors,	eighth to ninth year.
Anterior bicuspids,	tenth to eleventh year.
Posterior bicuspids,	tenth to eleventh year.
Canines,	eleventh to twelfth year.
Second molars,	twelfth to fourteenth year.
Third molars—wisdom teeth,	eighteenth to twenty-fifth year.

The milk teeth are gradually displaced by the permanent teeth and three additional molars appear on the sides of each jaw, so that the twenty milk teeth are replaced by the full set of thirty-two permanent teeth. The second dentition begins with the eruption of the anterior molars somewhere between the fifth and seventh years. Following these the milk teeth are gradually shed in the order in which they appeared, each tooth being forthwith or shortly replaced by a permanent tooth.

Shape and Structure of the Teeth.—Defects in the teeth are numerous, the most important being abnormalities of form, and especially the deficient development of enamel. In badly-nourished, feeble children the milk teeth are prone to caries.

The developing teeth are influenced by malnutrition, stomatitis, especially that produced by mercury, and constitutional diseases, as syphilis and rickets. The developmental defects show themselves in the permanent teeth. In rickets the teeth may be small and badly formed. As the result of infantile stomatitis the surfaces of the teeth are pitted, owing to deficient formation of enamel; the condition is sometimes improperly spoken of as *erosion*. These changes affect the incisors and canines, which are pitted by areas of default of enamel, and are of a bad color, showing a transverse furrow across all the teeth at the same level; the first permanent molars are also involved. These furrows are attributed, probably correctly, to severe illness in early life and are regarded as analogous to furrows on the nails which occur after serious disease.

Hutchinson Teeth.—In congenital syphilis the teeth are deformed and present appearances regarded by Hutchinson as specific and peculiar. The upper central incisors are affected. They are peg-shaped, short, and narrow, being smaller at the cutting edge than at the root. The enamel is commonly well formed and regularly developed, but the color is more yellow than that of the other teeth. At the edge of the teeth there is a single concave notch of varying depth in which the dentin is exposed. They are called Hutchinson, notched, or screw-driver teeth. These defects are not constant nor are they pathognomonic of syphilis, as they are sometimes found in other conditions, especially rickets. In the presence of other signs of syphilis—rhagades, keratitis, iritis, and nodes—notched teeth acquire positive diagnostic importance.

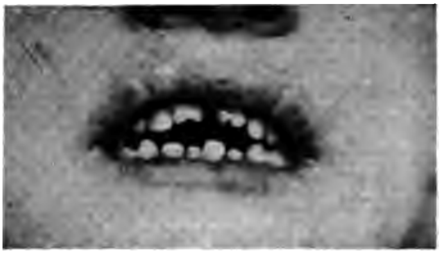


FIG. 199.—Hutchinson's teeth.

Caries.—Carious and neglected teeth play a very important part in the causation of derangements of digestion from imperfect mastication, and are themselves not rarely the result of constitutional disturbances. Extensive and rapid dental caries may occur after serious acute disease and in constitutional disorders as rickets and diabetes. It also occurs in pregnancy. The teeth become loose in forms of stomatitis associated with swollen and ulcerated gums, such as are encountered in mercurial ptyalism, scurvy, purpura, and phosphorus poisoning. Receding gums with exposure of the neck of the teeth and their ultimate loss occur from neglected salivary deposits, pyorrhœa alveolaris, and gouty conditions.

Sordes—literally filth—is a term applied to collections of dark brown foul matter upon the teeth and lips in low fevers. It consists of food, epithelial material, and altered blood, and contains micro-organisms in great numbers.

THE GUMS.

The gingival mucous membrane is pale in all forms of anæmia; it is red and spongy when the teeth are carious or ill-kept. A narrow red line along the margin is seen in some cases of tuberculosis, diabetes, and in cachectic states; also in alveolar disease. The gums are red, spongy, and ulcerated as a result of accumulated tartar and gangrenous and mercurial stomatitis. They are swollen, spongy, and bleeding in scurvy.

In lead poisoning a narrow bluish-black line is seen, although not invariably, at the margin of the gums. The color is not uniform, but, being due to lead sulphide deposited in the papillæ of the gums, is seen with the magnifying glass to be stippled. This line may form rapidly after exposure and disappear in the course of a few weeks under treatment, or it may persist for months. It is usually limited in extent. A similar line, due to the deposition of carbon particles, has been observed in miners. Such lines are to be distinguished from the deposits of black matter upon the teeth at the line of their juncture with the gums in untidy persons and smokers who neglect the mouth. The latter disappears upon the use of the tooth-brush, or the two lines may be differentiated by slipping the corner of a piece of writing-paper under the gum. If the pigment material is in the gum it stands out plainly against the white paper; that on the tooth is not seen. It is under certain circumstances also to be distinguished from cyanosis due to general disturbances of the circulation or local inflammatory processes. In cyanosis the discoloration is uniform and more intense at the edges of the gums and disappears under pressure.

frequently becomes elongated in angina and bronchitis. Under these circumstances it causes irritation of the base of the tongue and excites cough, especially in the recumbent posture; the mechanical violence of intense paroxysmal cough elongates the uvula and thus a vicious circuit is established. It becomes elongated and œdematous in cases of debility, anæmia, and anasarca. When greatly œdematous the uvula becomes globular and may attain the size of a cherry, interfering with swallowing and breathing and producing a constant disposition to hawk. In constitutional hemorrhagic states submucous extravasation of blood may occur in the uvula. In very rare instances crops of vesicles resembling herpes show themselves upon the palate.

Anæsthesia of the hard and soft palate and of the anterior two-thirds of the tongue occurs in lesions of the sensory division of the fifth nerve. The tactile sense is usually lost before the pain. The palate is innervated by the accessory nerve to the vagus. Paralysis of the soft palate occurs in bulbar palsy, basal tumors, and meningitis of the base. By far the most common cause is postdiphtheritic neuritis. Upon inspection while the patient pronounces the long *a* the palate and uvula are thrown back and elevated. Under normal circumstances the extent of this movement is the same on both sides. In unilateral paralysis movement upon the affected side is greatly diminished. In bilateral paralysis the whole palate remains relaxed and motionless, the voice has a nasal character, the pronunciation of certain consonants—gutturals—is impaired, and upon attempts to swallow, liquids are returned through the nose. Lesions involving the nerve-supply of one side cause unilateral paralysis.

THE TONSILS.

The tonsils, also called *amygdalæ* from their almond shape, lie at the side of the pharynx between the anterior and posterior palatine folds. They are larger in childhood than in adult life and early undergo senile involution. The greater part of their surface is exposed to inspection by ordinary methods. Upon gagging they are rotated forward. In inflammation the mucosa is reddened and swollen and the surface covered with a mucoid or mucopurulent secretion which may be tinged with blood. In follicular or lacunar tonsillitis this secretion develops in the crypts, producing whitish-yellow spots. These may by extension and coalescence form patches upon the tonsils presenting a superficial resemblance to diphtheria. The pseudomembrane thus formed is not usually distinctly marginate and corresponds in appearance to the points of exudate seen to occupy adjacent crypts. It is not developed in the mucosa but lies upon it, as may be seen upon removing it by wiping or gentle scraping. A pseudomembranous exudate frequently forms in the course of various infections, as scarlet fever, measles, pertussis, enteric fever, and variola. In a great majority of these cases the Streptococcus pyogenes is the active organism. As a rule the development of this form of pseudomembrane does not constitute a serious complication of the primary disease. It may, however, give rise to an intense angina with local sloughing and grave constitutional disturbance. A general streptococcus infection is by no means is impaired in paresis, diphtheritic palsy, progressive muscular atrophy, and some forms of hemiplegia. Slight deviation toward the paralyzed side may occur in cases of hemiplegia in which the face is affected. When the fibres of the hypoglossal are involved within the medulla after leaving their nuclei, there may be paralysis of the tongue on one side and paralysis of the limbs on the other, and the tongue when protruded deviates toward its sound side. Other causes of nuclear or infranuclear lesions of the hypoglossal are lead poisoning, basal meningitis, and tumors of the base.

Spasm of the tongue is very rare. It may be unilateral or bilateral —tonic or clonic. It is usually one of the manifestations of some other convulsive disease, as spasm of the facial muscles, tetanus, epilepsy, or chorea. Tonic spasm may occur in hysteria and as the result of reflex irritation of the fifth nerve. The tongue is contracted and rigid. Clonic spasm is much more common. Spasm of the lingual muscles occurs in stuttering. It is an occasional symptom in disseminated sclerosis, general paresis, and melancholia. There are cases of paroxysmal clonic spasm in which the tongue is thrust out and drawn in as often as forty or fifty times a minute. In this affection the spasm is usually bilateral; the attacks may occur during sleep.

The frænum of the tongue may be abnormally short—a congenital defect which, by limiting the movements of the tongue, interferes with nursing in the new-born and with articulation later.

(b) **Size of the Tongue.**—Variations in the size of the tongue are of diagnostic importance. The tongue is slightly enlarged and flabby in various conditions of ill health and especially in chronic gastritis, forms of anæmia, scurvy, and typhus fever. Under these circumstances the edges are indented by the teeth.

Enlargement of the tongue, or *macroglossia*, is usually congenital but may occur in later life. In the congenital form the tongue and very often the lips are greatly enlarged by an increase in all the tissue elements, an increase in the fibrous tissue alone, or from the development of tumor-like masses—true lymphangioma. The organ may become so large that it projects beyond the teeth, in some cases attaining twice its normal size. The surface is dry, fissured, or ulcerated from contact with the teeth, and deformity of the bony structures results from pressure. The lymph-vessels are dilated and in some instances there are actual cysts.

Great enlargement takes place in acute inflammation of the tongue, such as glossitis, inflamed ranula, erysipelas, angina Ludovici. The tongue is frequently much enlarged in actinomycosis. One side only may be involved in the inflammatory process—*hemiglossitis*. The tongue is somewhat enlarged in acromegaly and myxœdema. Localized swelling may be caused by tumors, as gumma or carcinoma. The tongue in rare instances becomes cyanosed and œdematous from obstruction to the return of the venous blood.

Diminution in the size of the tongue may be the result of a temporary shrinking or of atrophy. The tongue may be uniformly diminished in size after hemorrhage, during convalescence from enteric fever, or in conditions of advanced emaciation.

in intense congestion or hemorrhagic states the blood may be swallowed and accumulate in the stomach. If vomited the hemorrhage may be attributed to a lesion of the stomach. This error of diagnosis may be avoided by careful inspection of the pharynx.

Pharyngitis.—In acute inflammation of the oropharynx the mucosa is congested and reddened. The patient complains of tickling and dryness with a constant desire to hawk. The secretions are diminished and altered. There is dryness with thin flakes or a whitish exudate, to be seen only upon close examination. The constitutional symptoms are slight.

Rheumatic angina is characterized by sore throat and dysphagia referred to the pharynx. In the majority of the cases the signs upon inspection are not distinctive.

Chronic pharyngitis may develop insidiously or as the result of repeated acute attacks. The mucosa is at first reddened and shows distended vesicles; later it is relaxed and presents a granular or warty appearance—granular pharyngitis, due to hyperplasia of the lymph elements. The secretion is mucoid or purulent and undergoes desiccation, forming dry crusts or scales which very often communicate an offensive odor to the breath. The process extends into the nasopharynx. There is very often a free mucoid or mucopurulent secretion which gives rise to the sensation of dropping or trickling into the throat and causes hawking. In other cases the secretion is slight and the mucous membrane reddish-brown, dry, atrophic, smooth and glistening—*pharyngitis sicca*. The pseudomembranous exudate of diphtheria frequently extends into the pharynx; the exudate of pseudodiphtheritic, diphtheroid, or diplococcus inflammation commonly appears upon the tonsils and does not as a rule involve the pharynx.

Ulceration of the pharyngeal wall is not uncommon. Limited areas of superficial ulceration occur in chronic pharyngitis. Small round or oval ulcers upon the posterior wall are sometimes seen in enteric fever. Irregular superficial patches of ulceration are frequently seen in the later stages of consumption. The bases are necrotic and grayish-yellow. The ulceration may involve the greater part of the posterior pharyngeal wall and cause intensely painful dysphagia. Ulceration of the pharyngeal wall occurs also in syphilis. In the secondary stage it is very often superficial and associated with mucous patches. In the tertiary stage it results from the breaking down of gummata which heal satisfactorily under treatment, leaving white cicatrices.

Ulceration of the pharynx may occur in connection with the various forms of pseudomembranous inflammation and attends cancer and lupus. The etiological diagnosis of ulceration of the pharynx is frequently attended with difficulty. As in the case of the tongue, tubercle, cancer, and syphilis are to be differentiated. A careful anamnesis is important. The associated clinical phenomena are very often characteristic. In tuberculosis the presence or absence of tubercle bacilli and the inoculation test are important; in syphilis the therapeutic test.

Acute phlegmonous inflammation of the pharynx may result from traumatism or foreign bodies in the pharynx.

Acute infectious phlegmon, a rare condition, characterized by anginal symptoms, dysphagia, rapid abscess formation, swelling of the neck, and

Xerostomia—dry mouth—is a condition characterized by arrest of the salivary and buccal secretions. The condition is rare. It was first described by Jonathan Hutchinson. The tongue is red, dry, and fissured; the buccal mucous membrane is smooth and dry. Movements of the parts involved in articulation, mastication, and deglutition are attended with difficulty. In some cases the dryness extends to the nostrils and eyes and is accompanied by distressing itching. Slight enlargement of the salivary glands has been observed but is not constant; most of the cases occur in women of neurotic constitution. In a case under my observation in a woman aged thirty this condition developed during the convalescence from an attack of epidemic influenza. It has been suggested that the disease is due to involvement of a hypothetical centre controlling the salivary and buccal secretions.

Dryness of the tongue occurs in mouth-breathing, with thirst, after violent exertion, in febrile and septic states, conditions of profound prostration, and as the result of loss of fluid in diabetes mellitus and insipidus. It is an important symptom of atropine poisoning, and attends facial paralysis. Dryness of the tongue occurs under other conditions attended with extreme loss of fluid from the body, as in hemorrhage and cholera.

The papillæ of the tongue are often swollen, giving it a warty, granular appearance. This condition is seen in catarrhal and other forms of stomatitis, in some forms of chronic gastritis, and sometimes in the acute febrile infections. The enlarged fungiform papillæ of the tongue in scarlet fever have given rise to the unfortunate term "strawberry tongue," which by some teachers is understood to mean a tongue covered with a white fur through which the tip of the papillæ show, and by others to mean the rough bright red tongue which follows the separation of the coating. The latter is sometimes called the "raspberry tongue." In conditions of prostration, such as attend the later stages of infections or sepsis, and in some constitutional diseases, as diabetes, the tongue sheds its epithelium and the papillæ undergo atrophy. This condition is usually attended with dryness and glossing of the surface. The papillæ at the border of the tongue are sometimes greatly enlarged in gouty individuals. Patients are occasionally alarmed upon the discovery of the large circumvallate papillæ at the root of the tongue and hesitatingly accept the assurance that they are normal.

Coating of the Tongue.—This subject involves a consideration also of the general condition of the mucous membrane as regards color, dryness and moisture, and the condition of the papillæ. The presence or absence of coating is determined by local and constitutional conditions. It does not follow, as is very often assumed, that the condition of the tongue is directly dependent upon the condition of the mucous membrane of the stomach. On the contrary the diagnostic significance of coated tongue will be best understood by the clinician who realizes the fact, of which there is abundant clinical demonstration, that the condition of the tongue as regards coating and allied phenomena is largely dependent upon constitutional influences which are likewise exerted upon other mucous surfaces. Coating of the tongue occurs in many morbid conditions, especially dyspeptic states and in fevers, and is usually associated with loss of appetite; yet there are healthy individuals with good appetite whose tongue is constantly furred. A coated tongue is present in acute and chronic gastric catarrh, while on the other hand gastric ulcer is very often accompanied by a clean tongue and good appetite. The coating or fur is composed of accumulated epithelium and food detritus and contains great numbers of micro-organisms. The immediate cause of the extraordinary proliferation and accumulation of epithelial elements is not well understood. That the absence of coating is not merely dependent upon mechanical conditions associated with drinking and the ingestion of food is clearly shown by clinical experience. The coating of the tongue like its mucous membrane is very often stained by articles of food and drink or by drugs.

Coating of the Tongue in Local and General Conditions.—(a) LOCALIZED COATING of the tongue results from the irritation of a tooth and surrounds traumatic and other circumscribed lesions.

(b) UNILATERAL COATING of the tongue is sometimes seen in trifacial neuralgia involving the infra-orbital branch. It may occur also in unilateral palsy of the tongue.

(c) A UNIFORM THIN, WHITISH COATING is habitual to many persons in health, especially mouth-breathers, smokers, and those who are troubled by subacute catarrhal processes involving the pharynx and stomach. It occurs also in constitutional disturbances attended by slight fever.

(d) A THICKISH, PASTY, YELLOW-WHITE FUR is common in those addicted to excesses at table or in tobacco or alcohol. It is attended with a disagreeable taste. On rising it usually involves the greater portion of the dorsum of the tongue, but disappears in part or wholly during the day. In many persons this coating remains upon the back part of the tongue continuously. Its disappearance is to some extent due to movements of the tongue, friction against the teeth, the mechanical effects of food and drink, and increased flow of salivary and buccal secretions. A slightly enlarged, flabby, indented tongue covered with fur of this kind very often accompanies chronic gastritis.

(e) A THICK, UNIFORM, MOIST, WHITISH OR YELLOWISH-WHITE COATING with abrupt edges is seen in the early stages of the acute febrile diseases. In consequence of the diminished amount and altered character of the salivary and buccal secretions, this coating presently loses its moisture and becomes dry and darker in color. After a time it separates, leaving the tongue moist and of normal appearance if convalescence has begun, or dry, hard, red or brown, and denuded of epithelium if the fever continues and particularly if the patient falls into the so-called typhoid condition. Under these circumstances the tongue becomes fissured both longitudinally and transversely. In some cases a deep median fissure forms, on each side of which there is a thick, rough, dry, brownish fur, the tip and edges of the tongue being red and denuded; or again the tongue may be dry, red, and glazed. It is protruded upon request tremulously and slowly and, owing to the accompanying mental condition, is not immediately withdrawn. The disappearance of the crusty coating, the redevelopment of epithelium, and the return of moisture are favorable signs. The tongue may be dry, brown, and incrusted in the last stages of chronic diseases of the nervous system, and in cancer, nephritis, and pulmonary tuberculosis.

(f) THE THICK WHITE FUR of the acute febrile diseases is sometimes penetrated by the greatly enlarged filiform papillæ which appear as scattered bright red minute points. This constitutes one of the forms of so-called "strawberry tongue." It occurs with some frequency in scarlet fever, but is not diagnostic of that disease, since it may be present in other acute febrile infections.

(g) A DENSE, WHITE, FLAKY COATING is sometimes seen upon the tongue of patients who are fed upon an exclusive milk diet. A somewhat similar appearance may be presented by children suffering from thrush—a condition caused by *saccharomyces albicans*, which begins on the tongue in the form of slightly elevated pearly white spots which, by increase in size and coalescence, may cover the greater part of the dorsum of the tongue.

(h) GENERAL HYPERTROPHY OF THE PAPILLÆ gives rise to a peculiar appearance which suggests coarse plush. This is the *shaggy tongue*. It is seen in gastro-intestinal and constitutional diseases in advanced life, but is sometimes present in elderly people whose health is good. The shaggy tongue is frequently also fissured, the plush-like surface being divided by conspicuous deep longitudinal and transverse lines of separation. The color is usually deep red. Upon the supervention of acute illness it quickly becomes dry, hard, and full, usually remaining rough.

A red, dry tongue, denuded of epithelium, glistening and resembling raw beef—the *beefy tongue*—occurs in dysentery and chronic intestinal catarrh. It is seen also in hepatic abscess.

Other conditions of the tongue may be of diagnostic importance: fissures, ulcers, mucous patches and plaques, tumors, and cicatrices.

(a) FISSURES of the tongue are often seen in healthy persons in advanced life. They may be the signs of a superficial chronic glossitis caused by habitual use of tobacco or irritating food or drink. The median longitudinal fissure is commonly the most marked and readily becomes ulcerated. Transverse fissures are common. Sometimes the fissures are forked or curved. Fissures may be deep and inflamed, the result of extending glossitis—dissecting glossitis—or syphilis. Fissures are common in chronic hepatic disease, chronic colitis, and diabetes mellitus. Local fissures or notches at the edge of the tongue may arise from the irritation of a broken or carious tooth or from syphilitic ulceration.

(b) ULCERS OF THE TONGUE.—Simple excoriations occur as the result of slight traumatism or scalding, or spontaneously in dyspeptic conditions. Aphthous stomatitis is characterized by small, slightly depressed spots with grayish bases and bright red margins. They occur at the edges and tip of the tongue, on the frænum, and elsewhere about the mucous membrane of the lips and mouth. The ulcers are preceded by vesicles and are attended with great pain. The buccal secretions are increased. The ulcers may appear singly or in series or crops. They occur in transient gastric derangements and in women at the menstrual period. There is an individual predisposition to them.

A chronic, recurrent herpetic eruption of the buccal mucous membrane, sometimes associated with erythema multiforme, has been observed in neurotic persons.

Riga's disease is an affection occurring about the time of the first dentition and characterized by a pearly white pseudomembrane beneath the tongue and upon the frænum, with induration and ulceration. It is endemic and sometimes epidemic in Southern Italy.

Superficial ulcers with a red glazed surface occur upon the tongue in various forms of chronic glossitis. They are of irregularly round or oval shape with infiltrated edges and are usually extremely painful. Ulceration of the tongue is commonly attended with salivation. Tuberculosis of the tongue shows itself in the form of circumscribed, indolent, irregularly extending ulceration with a necrotic or caseous base. The edges are usually slightly infiltrated but sharply defined. This ulcer is extremely painful upon contact and is sometimes attended by salivation. The lesions may be single or multiple and are usually secondary to tuberculous disease of the lungs. The glands at the angle of the jaw are not usually enlarged. Syphilis is a common cause of ulceration of the tongue. In secondary syphilis superficial and linear ulcers are common at the border of the tongue as the result of the irritation of the teeth. A single ulcer with an indurated base and enlargement of the cervical glands may be the initial lesion of syphilis. A mucous patch may undergo ulceration, and in later syphilis a gumma may become necrotic, forming a deep foul ulcer. In some instances difficulty attends the differential diagnosis of a single ulcer, which may be due to tuberculosis, syphilis, or malignant disease. The resemblances upon inspection and palpation may be very close. In the first there are usually evidences of tuberculosis of the larynx or lungs and the presence of tubercle bacilli in the scrapings. In cases not otherwise to be determined inoculation experiments should be performed. In the initial lesion of syphilis the induration is dense and circumscribed. The age and habits of the patient are to be taken into consideration. Great enlargement and tenderness of the lymphatics of the neck constitute important symptoms. The evolution of the process and the development of mucous patches, cutaneous rashes, fever, and the like make the diagnosis clear. In gummatous ulceration the enlarged surface is greater and the infiltration less dense. The therapeutic test is important; the ulcer heals under antisyphilitic treatment. A carefully taken clinical history sheds light upon a doubtful case. In epithelioma of the tongue the diagnosis may be reached by exclusion. The process tends to spread, the sublingual lymphatics become involved, the ulcer is foul and indolent, and the patient is almost always past middle age.

The ulcer frequently observed on the frænum of the tongue in whooping-cough is traumatic. It results from the violent impact of the under surface of the tongue against the sharp lower incisors during the paroxysm.

(c) Mucous Patches and Plaques. — The multiple grayish-white superficial lesions of syphilis known as mucous patches occur upon the tongue as well as upon the soft palate, cheeks, and lips. A slightly raised, smooth, red, oval-shaped area sometimes seen in the middle of the dorsum of the tongue in pipe smokers is known as the smoker's patch. The surface is smooth and sometimes white or pearly white in appearance.

Xanthelasma occasionally appears upon the sides of the tongue in the form of yellowish, soft, slightly raised, oblong patches. It occurs in various conditions but is noticeably frequent in chronic jaundice and diabetes.

Leucoplakia is a condition characterized by the development of irregular white or pearly-white smooth patches upon the tongue which show no tendency to ulcerate. They are hard to the touch and gradually extend, sometimes becoming papillomatous. These patches may be the starting-point of epithelioma. The condition is described under various terms, as *buccal psoriasis, ichthyosis* and *keratosis mucosæ oris.* They present some points of similarity to the lesions of syphilitic glossitis, which is, however, more common at the edge and tip of the tongue than on the dorsum and yields to antisyphilitic medication.

Eczema of the Tongue—Geographical Tongue.—This condition is characterized by the formation of irregularly annular patches upon the tongue. There is desquamation of the epithelium. The process is attended with burning and itching. The patches extend at the margins with new formation of epithelium in the centre. The borders are slightly red and well defined but without induration. The condition is more common in infants and children than in adults. The process is recurrent and protracted.

(d) TUMORS OF THE TONGUE.—Solid tumors of the tongue are usually tuberculous or syphilitic. They invade the substance of the organ, usually presenting toward its dorsal surface. Tuberculous nodules break down, promptly giving rise to an indolent ulceration with caseation. Gummata rapidly undergo extensive necrosis but yield to treatment. Retention cysts occur in connection with the tongue. Ranula is the most common; it is due to an obstruction and dilatation of a duct of the sublingual or submaxillary glands. Mucous cysts also occur. Echinococcus cysts, which develop as a rule by preference in highly vascular structures, are rare in the tongue. Carcinoma is much more common in men than in women and extremely malignant. Sarcoma is comparatively rare.

(e) CICATRICES.—Scars upon the tongue tell the tale of former traumatism, as the accidental biting of the tongue, a fall or blow upon the chin when the tongue is between the teeth, or the grinding of the teeth during the clonic convulsions of epilepsy. They may be the indications of former active diseases, especially syphilis. Sclerosis of the tongue with local deformity is a common result of the healing of gummatous ulceration.

The buccal mucous membrane is commonly implicated in infections involving the other organs of the mouth, especially the various forms of stomatitis. It is very often the starting-point of the progressive gangrenous affection known as noma or cancrum oris.

VIII.

THE DIGESTIVE SYSTEM (CONTINUED): THE PALATE: TONSILS; PHARYNX.

The passage from the mouth to the œsophagus by way of the pharynx is called the fauces or *isthmus faucium.* It is bounded above by the soft palate, laterally by the palatine arches and tonsils, and below by the base of the tongue. These structures are covered with mucous membrane continuous with that of the mouth and are liable to the same morbid processes. An inspection of these parts yields information of importance in the diagnosis of local and constitutional disease. Infection may take place directly or by extension from the mouth and nasopharynx. Forms of angina —simplex, follicular, suppurative, and diphtheritic—result. When the tonsils are principally or alone involved the condition is spoken of as tonsillitis. The underlying muscular structures may be involved by extension. The tonsils and adjacent lymph structures are points of invasion for the infecting agents in rheumatism and other affections. There are forms of acute tonsillitis that are essentially rheumatic. In children the articular manifestations of rheumatic fever and chorea frequently show a definite relationship to tonsillitis and the latter affection is not rarely followed by endocarditis and chorea. The tonsils may be the port of invasion for tuberculosis or the seat of tuberculous lesions.

Subacute and chronic pharyngeal inflammation may be secondary to gastric disorders or to the gouty diathesis. The pharynx is sometimes involved in rheumatism. Paralysis of the soft palate and spasm and paralysis of the pharynx occur. Superficial ulceration of the pharynx is very common in advanced pulmonary tuberculosis.

General redness of the faucial mucous membrane occurs in simple inflammations and in many of the specific febrile affections, as rötheln, the variolous diseases, influenza, and erysipelas. In the exanthemata, especially measles, scarlatina, varicella, and variola, there are efflorescences corresponding to the cutaneous eruptions. In these situations the pocks of varicella and variola, owing to the action of warmth and moisture, lose their roof in the vesicular stage and are converted into small circular or oval superficial ulcerations with purulent or necrotic bases and a more or less marked areola. Redness of the mucous membrane in this region is a symptom of chronic gastritis or the action of certain drugs, as the iodine compounds and belladonna, and of corrosive poisons.

Hemorrhage occurs into the mucous membrane in the form of petechiæ, infarcts, and extravasations, and there is bleeding from these surfaces in general hemorrhagic states. These tissues are pallid in the anæmias, yellow in jaundice, and show a bluish tint in cyanosis. The mucous patches of syphilis may be seen.

Pain is a prominent symptom in angina, especially in the acute forms. It may be spontaneous, but is excited by the movements of deglutition and by contact of articles of food and drink with ulcerated surfaces. Pain d tickling referred to the pharynx may be symptomatic of acute rhini-. These symptoms are common in hay fever. Sensations of dryness d tickling accompanied by the inclination to hawk and clear the throat e constant symptoms of pharyngitis. Annoying hawking is especially cited by disease of the nasopharynx.

Dysphagia is common. It varies in degree and may be due to pain or mechanical obstruction. When dysphagia is marked both these causes e commonly operative. In suppurative tonsillitis and retropharyngeal scess dysphagia may be complete. It is a symptom of the various forms stomatitis and glossitis as well as of tonsillitis and pharyngitis. Painful sphagia referred to the pharynx is a common symptom in cases showing signs of inflammation of the mucous membrane—rheumatic pharyngitis. e angina which attends diphtheria, scarlet fever, measles, varicella, and riola is accompanied by dysphagia which is often distressing.

Dyspnœa may become an important symptom in suppurative tonsil-is, retropharyngeal abscess, and erysipelas extending to the pharynx.

Chronic interference with respiration accompanied by mouth-breath-g results from hyperplasia of the tonsils and especially from hyperplasia the pharyngeal tonsil—*adenoid vegetations.* In severe acute angina and certain chronic diseases involving the tonsils and pharynx, as cancer d forms of syphilis, the drainage of the fauces is interfered with and the cumulating secretions and exudates undergo decomposition. The odor the breath may be intense, fetid, and disgusting. Accumulations of ithelial cells, leucocytes, and bacteria in the tonsillar crypts are very mmon in chronic lacunar tonsillitis and in individuals presenting no her symptoms of disease of the throat. They appear as small white or llowish-white concretions which sometimes undergo calcareous changes. ey are sometimes expectorated and should be removed by the curette. ey impart a disagreeable odor to the breath.

THE PALATE.

Developmental deformities do not fall within the scope of this work. narrow, high, arched palate is regarded as among the stigmata of degen-ation. Circumscribed ulceration of the mucous membrane of the hard late is frequently met with in the new-born or may be caused in artifi-lly-fed children by the irritation of the rubber nipple. The ulceration us caused is sometimes described under the term *Bednar's aphtha.* In ung children patches of thrush are not uncommon upon the hard palate. scess formation attended with great pain occasionally involves the ucous membrane of the hard palate in connection with alveolar disease. rforations occur as the result of syphilis.

The soft palate in health is freely movable and symmetrical. The rm of the uvula varies in different persons. As a result of defective velopment it is sometimes bifid. It may be attached laterally to the ft palate or tonsil or to the posterior wall of the pharynx in consequence adhesive inflammation in diphtheria or syphilis. Perforation of the ft palate is almost always the result of syphilis. In very rare instances has followed scarlet fever. The uvula varies in length normally. It frequently becomes elongated in angina and bronchitis. Under these circumstances it causes irritation of the base of the tongue and excites cough, especially in the recumbent posture; the mechanical violence of intense paroxysmal cough elongates the uvula and thus a vicious circuit is established. It becomes elongated and œdematous in cases of debility, anæmia, and anasarca. When greatly œdematous the uvula becomes globular and may attain the size of a cherry, interfering with swallowing and breathing and producing a constant disposition to hawk. In constitutional hemorrhagic states submucous extravasation of blood may occur in the uvula. In very rare instances crops of vesicles resembling herpes show themselves upon the palate.

Anæsthesia of the hard and soft palate and of the anterior two-thirds of the tongue occurs in lesions of the sensory division of the fifth nerve. The tactile sense is usually lost before the pain. The palate is innervated by the accessory nerve to the vagus. Paralysis of the soft palate occurs in bulbar palsy, basal tumors, and meningitis of the base. By far the most common cause is postdiphtheritic neuritis. Upon inspection while the patient pronounces the long *a* the palate and uvula are thrown back and elevated. Under normal circumstances the extent of this movement is the same on both sides. In unilateral paralysis movement upon the affected side is greatly diminished. In bilateral paralysis the whole palate remains relaxed and motionless, the voice has a nasal character, the pronunciation of certain consonants—gutturals—is impaired, and upon attempts to swallow, liquids are returned through the nose. Lesions involving the nerve-supply of one side cause unilateral paralysis.

THE TONSILS.

The tonsils, also called *amygdalæ* from their almond shape, lie at the side of the pharynx between the anterior and posterior palatine folds. They are larger in childhood than in adult life and early undergo senile involution. The greater part of their surface is exposed to inspection by ordinary methods. Upon gagging they are rotated forward. In inflammation the mucosa is reddened and swollen and the surface covered with a mucoid or mucopurulent secretion which may be tinged with blood. In follicular or lacunar tonsillitis this secretion develops in the crypts, producing whitish-yellow spots. These may by extension and coalescence form patches upon the tonsils presenting a superficial resemblance to diphtheria. The pseudomembrane thus formed is not usually distinctly marginate and corresponds in appearance to the points of exudate seen to occupy adjacent crypts. It is not developed in the mucosa but lies upon it, as may be seen upon removing it by wiping or gentle scraping. A pseudomembranous exudate frequently forms in the course of various infections, as scarlet fever, measles, pertussis, enteric fever, and variola. In a great majority of these cases the Streptococcus pyogenes is the active organism. As a rule the development of this form of pseudomembrane does not constitute a serious complication of the primary disease. It may, however, give rise to an intense angina with local sloughing and grave constitutional disturbance. A general streptococcus infection is by no means infrequent. A pseudomembranous exudate occurs in its most typical form as a manifestation of diphtheria. It is caused by the Klebs-Löffler bacillus.

In suppurative tonsillitis or quinsy one or both tonsils may be involved. The earliest symptoms are those of an ordinary acute angina—pain, dryness, dysphagia, with fever and other symptoms of constitutional disturbance. The tonsils are enlarged, dusky red, and œdematous. They may even meet, or if one only is involved it may extend some distance beyond the median line. In many instances there is salivation. The breath is foul, the glands of the neck enlarged, and the patient opens his mouth only partially and with great difficulty. After suppuration occurs fluctuation may be felt.

Enlargement of the tonsils is common in children. It may be due to repeated attacks of acute tonsillitis or to a chronic inflammatory process leading to a hyperplasia of the lymphoid elements. The tonsillar crypts are enlarged. In some cases a probe may be introduced to the depth of a centimetre or more. Partial or complete adhesions of the anterior pillars to the tonsils are seen, and these structures are sometimes thin, red, and stretched by the enlargement of the tonsil. In some instances the tonsils are dense and firm, the connective-tissue stroma predominating. Enlargement of the tonsils is very often associated with adenoid vegetations in the pharyngeal vault. Mouth-breathing and its concomitant derangements accompany this condition. Ulceration of the tonsils is not very common. In syphilis the primary chancre has occurred upon the tonsils. In secondary syphilis mucous patches are very common in this region, and in the tertiary stage gumma may give rise to enlargement of the tonsil and, upon breaking down, result in deep circular ulceration with a necrotic base and little hyperæmia of the surrounding tissue. Tuberculous ulceration of the tonsils is not common.

THE PHARYNX.

This organ may be divided into an upper portion—the nasopharynx—and a lower portion—the oropharynx. The former may be examined by palpation with the finger or by the rhinoscopic mirror; the latter by direct inspection in a good light. Small foreign bodies, as fish-bones or a beard of wheat, may be recognized upon inspection; larger foreign bodies, as an artificial denture or fragment of meat or bone, by inspection or palpation. The presence of adenoid vegetations due to hyperplasia of the pharyngeal tonsil may be thus determined. Papillomatous masses sometimes fill the vault of the pharynx, extending into the posterior nares and greatly interfering with respiration. By occluding the orifices of the Eustachian tubes they cause deafness and middle-ear disease.

Cyanosis and Pulsation.—Cyanosis of the pharyngeal mucosa may result from general derangements of the circulation or respiration or from local causes, as obstruction to the return flow of the blood by way of the superior vena cava, from aneurism or from mediastinal tumor. In aortic regurgitation pulsation of the capillary vessels may be seen or unilateral pulsation may be the manifestation of a tortuous internal carotid artery or aneurism of that vessel. In the oozing that takes place from the pharynx in intense congestion or hemorrhagic states the blood may be swallowed and accumulate in the stomach. If vomited the hemorrhage may be attributed to a lesion of the stomach. This error of diagnosis may be avoided by careful inspection of the pharynx.

Pharyngitis.—In acute inflammation of the oropharynx the mucosa is congested and reddened. The patient complains of tickling and dryness with a constant desire to hawk. The secretions are diminished and altered. There is dryness with thin flakes or a whitish exudate, to be seen only upon close examination. The constitutional symptoms are slight.

Rheumatic angina is characterized by sore throat and dysphagia referred to the pharynx. In the majority of the cases the signs upon inspection are not distinctive.

Chronic pharyngitis may develop insidiously or as the result of repeated acute attacks. The mucosa is at first reddened and shows distended vesicles; later it is relaxed and presents a granular or warty appearance—granular pharyngitis, due to hyperplasia of the lymph elements. The secretion is mucoid or purulent and undergoes desiccation, forming dry crusts or scales which very often communicate an offensive odor to the breath. The process extends into the nasopharynx. There is very often a free mucoid or mucopurulent secretion which gives rise to the sensation of dropping or trickling into the throat and causes hawking. In other cases the secretion is slight and the mucous membrane reddish-brown, dry, atrophic, smooth and glistening—*pharyngitis sicca.* The pseudomembranous exudate of diphtheria frequently extends into the pharynx; the exudate of pseudodiphtheritic, diphtheroid, or diplococcus inflammation commonly appears upon the tonsils and does not as a rule involve the pharynx.

Ulceration of the pharyngeal wall is not uncommon. Limited areas of superficial ulceration occur in chronic pharyngitis. Small round or oval ulcers upon the posterior wall are sometimes seen in enteric fever. Irregular superficial patches of ulceration are frequently seen in the later stages of consumption. The bases are necrotic and grayish-yellow. The ulceration may involve the greater part of the posterior pharyngeal wall and cause intensely painful dysphagia. Ulceration of the pharyngeal wall occurs also in syphilis. In the secondary stage it is very often superficial and associated with mucous patches. In the tertiary stage it results from the breaking down of gummata which heal satisfactorily under treatment, leaving white cicatrices.

Ulceration of the pharynx may occur in connection with the various forms of pseudomembranous inflammation and attends cancer and lupus. The etiological diagnosis of ulceration of the pharynx is frequently attended with difficulty. As in the case of the tongue, tubercle, cancer, and syphilis are to be differentiated. A careful anamnesis is important. The associated clinical phenomena are very often characteristic. In tuberculosis the presence or absence of tubercle bacilli and the inoculation test are important; in syphilis the therapeutic test.

Acute phlegmonous inflammation of the pharynx may result from traumatism or foreign bodies in the pharynx.

Acute infectious phlegmon, a rare condition, characterized by anginal symptoms, dysphagia, rapid abscess formation, swelling of the neck, and vere constitutional symptoms, may result from direct traumatism, e injury caused by foreign bodies, or arise spontaneously.

Retropharyngeal abscess manifests itself upon inspection and palpa- on as a projecting fluctuating tumor upon the posterior wall of the pharynx the median line. Attendant phenomena are restlessness, dysphagia, and anges in the voice, which becomes nasal or metallic as the result of pres- re. Retropharyngeal abscess is a rare affection. It has been observed children previously in apparent good health as a sequel of the infectious seases, particularly scarlet fever and diphtheria, and in caries of the rvical vertebræ.

Angina Ludovici: Ludwig's Angina; Cellulitis of the Neck.—A rap- ly developing phlegmonous inflammation of the tissues about the floor of e mouth is described under these names. It apparently results from auma or some lesion about the roots of the teeth or from infection of the bmaxillary gland. It may occur as the result of secondary infection in e specific fevers, particularly diphtheria and scarlet fever. The inflamma- on is the result of streptococcus infection. Swelling usually appears first the submaxillary region of one side and rapidly spreads, with diffuse ll redness and brawny induration of the neck. The tendency is to speedy ppuration and extensive gangrene with general septicæmia. The disease rare and very fatal.

The innervation of the pharynx is derived from the pharyngeal plexus, rmed by the combination of the glossopharyngeal and branches of the vagus.

Spasm of the pharynx is a functional disorder. It is common in neuro- thic individuals. It is the cause of ordinary gagging and occurs in hydro- obia and as a convulsive manifestation of hysteria—*globus hystericus.*

Motor palsy of the pharynx occurs in postdiphtheritic neuritis, acute cending paralysis, and bulbar paralysis. It may result from lesions at e base of the brain. It is commonly bilateral. There is difficulty in allowing and food is not properly passed into the œsophagus. Particles food may pass into the larynx and, when there is associated paralysis the soft palate, into the posterior nares. Fluids are regurgitated rough the nose. In unilateral lesions the power of deglutition remains.

Anæsthesia of the pharynx is produced by bromidism and the local plication of cocaine.

IX.

HE DIGESTIVE SYSTEM (CONTINUED): THE ŒSOPHAGUS.

The upper limit of this organ is about at the level of the cricoid car- age and opposite the sixth cervical vertebra. It terminates in the car- c orifice of the stomach opposite the upper border of the body of the venth thoracic vertebra. It has a short infradiaphragmatic course of out one and a half centimetres. It begins about six inches—fifteen cm. rom the incisor teeth, is about nine and a half inches—twenty-four cm. n length, and varies from three-fourths to one and a fourth inch—two three cm.—in diameter, the narrowest parts being at the commencement, in the middle, where it is crossed by the left primary bronchus, and at its point of entrance into the stomach. The œsophagus is in relation with the trachea, the left bronchus, the thyroid body, the peribronchial lymph-glands, the pneumogastric and recurrent laryngeal nerves, the aorta, the azygos vein, the thoracic duct, and the pericardium and pleuræ. Nearly its whole course is in the posterior mediastinum.

The principal methods of examination are auscultation, direct inspection of the interior of the œsophagus, the use of the sound, and the Röntgen rays. Ordinary inspection, palpation, and percussion are of no practical value, although the first two of these methods may reveal a tumor upon the left side of the neck when there is a diverticulum or new growth in the cervical portion.

Auscultation. — Upon auscultation, the stethoscope being placed to the left of the ensiform cartilage or to the left of the spine opposite the tenth rib, a gurgling sound may be heard six seconds after the act of swallowing, as determined by the movement of the larynx. This murmur is due to the propulsion of the liquid or bolus of food into the stomach and is not to be confounded with the sound to be heard over the cervical part of the œsophagus during swallowing. The absence, delay, or prolongation of the first-named sound is evidence of obstruction at the lower end of the œsophagus.

Œsophagoscopy. — Direct inspection may be practised through a suitable tube or instrument with proper illumination. The mucosa in acute inflammation is reddened, swollen, and lax; in chronic inflammation, grayish-white, covered with a viscid mucus, and shows dilated veins. The instrument may be used as a sound to determine the presence or absence of dilatation or narrowing. Ulceration, new growths, and cicatrices may be recognized, and fragments of ulcerated tissue have been removed through the œsophagoscope for examination. Foreign bodies may be located and have been removed by instruments passed through the tube when their removal by ordinary methods has proved impracticable.

The Œsophageal Sound. — The ordinary rubber tube used in the examination and treatment of diseases of the stomach may be utilized or œsophageal bougies especially made for the purpose. The latter are of whalebone or narrow blades of metal with rounded edges and provided with adjustable olive-shaped tips made of hard rubber, ivory, or metal and of various sizes. The sound is introduced in the same manner as the stomach tube. It may pass directly into the stomach or be arrested by some obstruction. The location of the stenosis can be readily determined by measuring the distance from the teeth upon withdrawing the instrument. No force is to be used. Feeble and anæmic patients may faint during this examination and neurotic or hysterical individuals may have local spasm or even general convulsions. Under such circumstances the instrument should be immediately withdrawn. Sounding must be performed with due caution, since there is the danger of injury or perforation of the wall of the œsophagus, the rupture of an aneurism, or the laceration of the varicose veins of the œsophageal plexus in atrophic cirrhosis. By the use of the sound the location of strictures, dilatations, diverticula, ulceration or at least areas of sensitiveness, and the presence or absence of foreign bodies and their location may be learned. The careful use of this instrument yields information as to whether or not a stricture is dilatable or rigid and unyielding.

The X-rays.—The presence and position of foreign bodies in the œsophagus may be ascertained by this method of examination, and in appropriate cases information in regard to tumors of, or in relation with, the œsophagus. The possibility that a large atheromatous plate in the aorta may be mistaken for a foreign body in the gullet is to be borne in mind.

Symptoms of disease of the œsophagus are dysphagia, pain, and the regurgitation of food.

Dysphagia varies according to the disease and its site and is commonly greater with solids than with fluids; the pain may be sharply localized or diffuse; the regurgitation of food may be partial or complete and take place immediately or not for some time.

The œsophagus is subject to developmental defects, of which the most important is atresia. Liquids are immediately regurgitated and the sound cannot be passed. Death results from inhalation pneumonia or starvation.

Alterations in Calibre.—The œsophagus may be narrowed or dilated. Very often these two conditions are combined, the tube being narrowed at one point and dilated at another.

Narrowing may be intrinsic, due to lesions of the œsophagus itself, as congenital defect, stricture from inflammation, cicatrix or neoplasm, or muscular spasm; or extrinsic, due to pressure from without. The symptoms vary. In the first instance they are chiefly dysphagia, pain, and regurgitation; in the second there are superadded to these the symptoms of the disease causing the compression. Narrowing may be a congenital defect. Its position in this case is usually at the upper or lower extremity. The chief symptom is dysphagia.

In **inflammatory and cicatricial stenosis** there is a history of accidental or intentional swallowing of a caustic or corrosive fluid, or the history may point to ulceration as the result of traumatism produced by a foreign body, softened glands, syphilis, or peptic ulcer at the cardia. Ninety per cent. of the cases of stenosis are due to cancer, which acts by infiltrating the walls and causing the development of contracting connective tissue. In stricture arising from cicatrix the dysphagia comes on gradually and is progressive and permanent. It may begin abruptly and at first be caused by solids only; later by fluids. Associated spasm may cause variations in degree, but there are no intervals of complete relief as in spasmodic stricture. The bougie is always arrested at the same distance from the teeth. Food is regurgitated shortly after it is taken and, unless acid in itself, shows an alkaline reaction. Subjectively it seems to stop at or near the manubrium. There is actual progressive starvation and corresponding emaciation. Signs of pressure upon the recurrent pharyngeal nerves are rare in cicatricial stenosis.

Malignant stricture of the œsophagus is commonly carcinomatous. A limited number of cases of sarcoma have been reported. Carcinoma is more common in men than in women. It is rare before forty and most frequent between fifty and sixty. It occurs with about equal frequency in the upper and the lower half of the organ. The symptoms are not very different from those of cicatricial stricture. Pain is more prominent; it is usually referred to the gullet; sometimes to the back between the shoulder-blades. The food is commonly returned shortly after it is taken and is sometimes streaked with blood, or it may contain fragments of necrotic tissue. The obstruction may become complete by the impaction of food in the stricture. Cough is common, and hoarseness, aphonia or complete loss of voice may result from involvement of the recurrent laryngeal nerves. Hunger, at first urgent, gives place to indifference to food. Thirst is troublesome, the mouth dry, the breath foul, and hiccough frequent. The progress of the disease is rapid.

Spasmodic Stricture.—Œsophagismus occurs in neurotic persons and especially in hysterical women. It has some points of resemblance to the "globus hystericus." It may be due to mental shock or prolonged depressing emotions, but it is more frequently due to reflex irritation in disorders of the gastro-intestinal or reproductive tract. In rare instances it accompanies disease of the larynx, and it often recurs in diseases of the œsophagus. It occurs in human rabies and in the hysterical counterfeits of that disease, and has been observed in cerebrospinal fever, tetanus, and epilepsy. Dysphagia is paroxysmal and of varying degree. It comes on abruptly and often passes away as rapidly as it came. Food is regurgitated suddenly and with force. There are gulping sounds. The difficulty in swallowing is produced by liquids as well as by solids. The patients often complain of pain which is constricting and burning in character. Emaciation does not usually occur. The bougie is not always arrested at the same level and may usually be passed by firm pressure beyond the point of resistance and into the stomach.

Pressure.—Narrowing of the œsophagus by pressure from without may be caused by enlargement of the thyroid body, as in goitre, Graves's disease, cystic degeneration, or tumors involving that organ or enlarged lymph-glands. Within the thorax a mediastinal tumor, dislocation backward of the sternal end of the clavicle, prevertebral abscesses and tumors, aneurism of the aorta, a distended diverticulum or massive pericardial effusion may compress the œsophagus. The essential symptom is dysphagia. The sound may usually be passed with persistent gentle pressure. If there is reason to suspect the presence of an aneurism the sound must not be used. The œsophagus adjusts itself to external pressure to a remarkable degree and unless it is extreme the symptoms are slight.

Obstruction from plugging may result in infants from excessive proliferation of the thrush fungus; at any period of life from any foreign body swallowed by accident or design. Common among these are masses of meat, fragments of bone, artificial dentures, jack-stones and other small playthings. Pedunculate polypi and other tumors may obstruct the œsophagus without causing stricture.

Diverticula or circumscribed lateral dilatations are of two kinds, those caused by internal pressure—*pulsion diverticula*—and those brought about by the contraction of fibrous tissue outside the organ—*traction diverticula.*

Pulsion diverticula first show themselves by discomfort or a sense of obstruction after swallowing food, usually referred to the sternal region and often attended by cough. After a time liquids only can be taken and are sometimes regurgitated and swallowed again and again before reaching the stomach. Portions of food may be regurgitated several hours after having been swallowed. Pressure or upward stroking of the left side of the neck may aid in the regurgitation of food. A tumor is not often present. Upon auscultation the sound produced by the passage of food into the stomach is absent. A sound may be introduced into the diverticulum, the blind end of which may be eight inches—twenty cm.—or more from the teeth; it may pass into the stomach and be freely movable in that organ; or one sound may be passed into the diverticulum and while it is still in place another may be passed beyond it into the stomach. The symptoms increase in severity and in many cases there is progressive emaciation. As a rule the progress of the disease is tardy.

Traction diverticula are usually situated upon the anterior or lateral wall and near the bifurcation of the trachea. They are funnel-shaped and vary in depth from one-half to three-quarters of an inch and are usually single but may be multiple. They commonly give rise to no symptoms. Particles of food or foreign bodies may, however, be caught in them and cause ulceration and perforation, with bronchopneumonia, pulmonary gangrene, mediastinitis or pericarditis and pleurisy. A positive diagnosis cannot be made.

Ulceration may cause tenderness, dysphagia; perforation the secondary lesions just mentioned; and rupture, which usually results from the presence of a foreign body, may cause gangrenous mediastinitis and pleurisy.

Œsophageal hemorrhage may result from ulcer, cancer, the presence of foreign bodies, the rupture of an aneurism or of the dilated veins of the œsophageal plexus in thrombosis of the portal vein or in atrophic cirrhosis of the liver.

The bleeding may be occasional or constant and vary in quantity from a trifling amount to a copious loss which is quickly fatal. The associated symptoms may render the diagnosis easy, but in the case of varicose veins the differential diagnosis from gastric hemorrhage is often difficult. The presence of other symptoms of portal obstruction and the fact that the blood is regurgitated rather than vomited are to be considered, but the blood may be discharged into the stomach and subsequently vomited.

Inflammation of the Œsophagus.—Œsophagitis is attended by localized or diffuse pain upon swallowing, prostration, and in the severe acute forms by chills and fever. There may be tenderness upon pressure and upon bending the spine; for this reason the head is held rigid. Abscess formation may show itself by a circumscribed swelling upon one side of the neck with pressure upon the larynx and hoarseness and dyspnœa. In the phlegmonous form pus may be expectorated and in the chronic cases a glairy, viscid mucus.

Tuberculous and syphilitic ulcerations occur as local manifestations in these diseases, and their presence is to be suspected when there is dysphagia, persistent substernal pain, or the regurgitation of blood-stained mucus in connection with the general phenomena of these diseases respectively.

Paralysis.—The œsophageal muscles are sometimes paralyzed in central or peripheral diseases of the nervous system. Lesions in the neighborhood of the origin of the pneumogastric nerves, such as hemorrhage, softening, tumor, or sclerosis, are among the central causes; pressure neuritis of the pneumogastric from enlargement of the lymphatic glands, or disease of the vertebræ and toxic neuritis after diphtheria or in chronic alcoholism or lead poisoning are among the peripheral causes. Difficulty of swallowing, without pain, is a characteristic symptom. It develops gradually or abruptly, according to the cause. The food produces a sense of weight or pressure and a large bolus is more readily swallowed than small morsels. Fluids may be regurgitated. Gurgling sounds attend the act of swallowing, but the normal sound at the cardia is not heard. The bougie passes freely.

X.

THE DIGESTIVE SYSTEM (CONTINUED): APPETITE; THIRST; ERUCTATIONS; REGURGITATION; NAUSEA; VOMITING; THE VOMITUS; DEFECATION; CONSTIPATION; DIARRHŒA; TENESMUS; PAINFUL DEFECATION; FECAL INCONTINENCE; CHARACTER OF THE DISCHARGES.

Appetite, thirst, the frequency of defecation and the consistency and other characters of the stools vary within wide limits in health. They are to a considerable extent influenced by habit and the mode of life of the individual. Beyond these limits they, together with certain associated symptoms, acquire clinical significance of importance in disease of the digestive organs and other local and general affections.

APPETITE.

Appetite is dependent upon the state of the gustatory nerves, the condition of the stomach, and the requirements of the organism as a whole.

The appetite for food may be diminished, lost—*anorexia;* increased—*polyphagia* or *bulimia;* perverted—*pica* or *parorexia*; or insatiable—*acoria.*

Loss of appetite varies from mere indifference to food to complete anorexia. It is symptomatic of the most varied morbid states, the enumeration of which would comprise a nosological system.

The appetite is more or less impaired in:

(a) The acute infections. The absence of the normal desire for food is due chiefly to the toxæmia, and in part to the subacute gastritis which is usually present.

(b) The chronic infections, especially in the active stages of syphilis, tuberculosis, and malaria, and in the conditions of malnutrition and cachexia to which these diseases give rise.

(c) Septic conditions, both acute and chronic, and in all forms of local suppuration. Loss of appetite in the absence of gastric disease or other adequate obvious cause, especially when associated with persistent leucocytosis, may be symptomatic of local suppuration in some part of the body.

(d) Pyrexia. Fever is attended by loss of appetite as in any of the foregoing conditions, and this symptom is commonly present in the early convalescence from febrile diseases. A notable exception to the latter statement occurs in enteric fever, in which hunger is usually a prominent and urgent symptom after the defervescence.

(e) All forms of anæmia, chronic wasting diseases, and in many functional and organic diseases of the nervous system. The loss of appetite is not only an important symptom in these conditions but it is also an etiological factor. A vicious circuit is established. The inability to take food aggravates the condition that causes it.

A remarkable suppression of the desire for food is manifest in certain cases of hysteria. There are instances in which the suppression of appetite is maintained for long periods, as in "fasting girls." Deception is to be guarded against. To hysteria is to be referred the condition described by Gull as *anorexia nervosa,* in which there is not only complete loss of appetite but also absolute inability to take food, with the gravest symptoms of inanition, sometimes ending in death.

(f) Cachexias and terminal states. The patient is not only unable to take food save in minimum quantities, but life is also often maintained without it for considerable periods—a fact due to the extreme limitation of vital activities.

(g) Toxic conditions. Complete loss of appetite attends all acute toxic conditions, and there is great impairment in chronic intoxications, as that of lead, arsenic, or mercury. The anorexia is due in part to the general malnutrition, in part to local disorder of the organs of digestion. In chronic alcoholism appetite is irregular and enfeebled and at the close of a debauch is completely lost. Aversion to food is frequently the forerunner of an attack of delirium tremens.

(h) Psychic states. Depressing emotional conditions, such as result from worry, anxiety, suspense, and grief, are usually attended with anorexia. The impairment of appetite under these circumstances is largely dependent upon the temperament of the individual.

(i) Functional or organic disease of the stomach. Appetite may persist normally or in some abnormal form in the gastric neuroses, and is maintained in some cases of gastric ulcer. Patients suffering from disease of the stomach frequently have a craving for food which is immediately dispelled upon attempts to eat. Loss of appetite not infrequently results from a monotonous or inadequate dietary. Under these circumstances the appetite frequently returns when the patient is permitted to take ordinary food. There is a French proverb to the effect that *appetite comes with eating.*

Polyphagia is a term used to indicate excessive or voracious eating. It may be occasional, as in the convalescence from enteric fever or in children suffering from whooping-cough, the frequent vomiting caused by the paroxysms not permitting the absorption of sufficient food to meet the needs of the organism; or persistent, as in diabetes.

Bulimia and acoria are terms used to designate an insatiable appetite. This is symptomatic of certain insanities and some forms of idiocy and occurs in paroxysms in certain cases of hysteria, neurasthenia, epilepsy, and exophthalmic goitre. In polyphagia the patient eats large quantities of food and is for the time being satisfied. In bulimia the ordinary sense of satiety after eating does not occur. Acoria is the loss of the sensation of satiety.

Pica or parorexia is a craving for unnatural articles of food—a depraved appetite. It is seen in some cases of hysteria, chlorosis, and in pregnancy. These terms are also employed to designate a nervous craving for special articles of diet or for articles that are not fit for food.

THIRST.

Thirst is in some instances an individual peculiarity. There are persons who rarely experience the sensation of thirst and do not consume enough fluid to fully meet the requirements of the body; others who without impairment of health manifest an habitually abnormal desire for fluid.

Impaired Thirst.—The sensation of thirst is diminished in soporous states, even when the buccal and salivary secretions are diminished and the mouth and tongue are dry, as in enteric fever.

Increased thirst is symptomatic of many morbid states. It is a constant symptom in fevers and occurs in all conditions attended with abundant or profuse loss of fluids, and is proportionate to the dehydration of the tissues. It therefore attends profuse sweating both physiological and pathological, abundant diuresis from any cause, persistent vomiting, abundant watery discharges from the bowels, and sudden copious hemorrhage. It occurs at the time of crisis from acute diseases, as croupous pneumonia; in the polyuria of hysteria and persistently in diabetes insipidus and mellitus; in the copious vomiting of acute irritant poisoning and in some cases of uræmia; after the action of elaterium and other drugs producing large watery discharges from the bowels; in cholera nostras and Asiatica and after all kinds of abundant hemorrhages both pathological and traumatic. An unusual desire for water is observed in some cases of chronic gastritis. Persistent excessive thirst is very often the first symptom to attract attention in diabetes. The arrest of the buccal secretions in xerostomia or dry mouth gives rise to continued and distressing thirst. Polydipsia is a term used to describe the habitual taking of fluid in excessive amounts.

ERUCTATIONS, REGURGITATION, NAUSEA, AND VOMITING.

The œsophagus enters the stomach at an angle, forming a valve-like fold which serves to prevent the return of the contents of the stomach. The relation of the central tendon of the diaphragm to the œsophagus is such that it closes the œsophageal opening only at the time of inspiration.

Eructations or Belching.

The spasmodic forcible discharge of gas or air from the mouth is a mmon symptom. It may come from the œsophagus; much more comonly it comes from the stomach. It is sometimes odorless, frequently fensive. It may consist of air swallowed with the food or with the saliva, of the gaseous products of the chemical decomposition of the food in e stomach. The eructations may be occasional or occur in paroxysms sting for periods of some hours. Eructations are symptomatic of ute indigestion such as results from over-eating, various forms of stritis and other organic affections of the stomach, or they may be of rvous origin. Large quantities of odorless gas are sometimes forcibly pelled at intervals during a period of several hours in hysterical and urasthenic individuals, the stomach at the same time being tensely stended.

Regurgitation.

The liquid portions of the food, and in some instances the solids, are rerned to the mouth without the violence characteristic of vomiting. Regurtation from the œsophagus occurs as a symptom of stricture, dilatation or verticulum, the food being returned immediately or after an interval. Rergitation from the stomach may be due to over-distention with food, or ink and relaxation of the cardiac orifice. The regurgitation of considerable antities of an opalescent, slightly alkaline fluid is spoken of as *water-brash.*

Merycism or rumination is the regurgitation of solid food from the omach to the mouth, when it is again chewed and swallowed. The food returned in small portions without nausea. This phenomenon appears first to be the result of regurgitation, later a habit.

Pyrosis or heart-burn is a burning sensation behind the sternum, tending to the pharynx. It is often accompanied by eructation and metimes by the regurgitation of an acid fluid. It is due to the ejection the gastric contents into the œsophagus. It occurs in hyperchlorhydria it may appear as a neurosis when the gastric secretion is normal.

Nausea.

Nausea or sickness at the stomach is closely associated with vomiting its mechanism and clinical significance. It belongs to the group of normal sensations referred to the stomach, and occurs in functional d organic affections. Those causes which excite vomiting also excite usea, though the latter may occur in the absence of the former. The rm "nervous nausea" is applied to this symptom when it arises in conitutional disorders and diseases of the central nervous system. It is mmon in neurasthenia and hysteria and is very often the result of reflex ritation in distant organs, for example, the uterus and ovaries. Nausea very common in the early stages of pregnancy, and, associated with tching and vomiting, constitutes in pregnancy the syndrome known as *orning sickness*, which in exceptional cases is persistent and intractable d may even cause death.

Vomiting.

Vomiting is the forcible expulsion of the contents of the stomach through the mouth. In exceptional cases the contents of the intestines may also be expelled through the mouth—fecal or stercoraceous vomiting.

The Muscular Mechanism.—The act of vomiting is a complex reflex movement in which many muscles take part. There is usually a sensation of nausea and a reflex flow of saliva into the mouth, accompanied or followed by a series of more or less violent retching movements which consist of deep inspirations with closure of the glottis. As a result of these movements the stomach is compressed by the diaphragm and the negative pressure in the thorax and especially in the œsophagus is decidedly increased. In the course of these retching movements the act of vomiting is brought about by a sudden convulsive contraction of the abdominal muscles which exerts additional pressure upon the stomach. With this the cardiac orifice of the stomach is dilated and the stomach contents are forced through the œsophagus, the glottis being closed by the adductor muscles and the nasal chambers shut off from the pharynx by the contraction of the posterior pillars of the fauces upon the palate and uvula. In the vomiting of unconsciousness, as in anæsthesia, the laryngeal muscles may relax and vomited matters be insufflated into the trachea, and in violent vomiting the material may in part be forced past the palate and uvula and ejected through the nose.

It is not uncommon for the contents of the duodenum to be forced by the violence of the contraction of the abdominal muscles through the pylorus, so that the vomitus consists of bile-stained material and sometimes of pure bile.

The muscles concerned in vomiting are respiratory. The act consists essentially in the simultaneous spasmodic contraction of the diaphragm, an inspiratory muscle, and the abdominal or expiratory muscles, contraction of the muscular fibres of the stomach being altogether of subsidiary importance.

The Nervous Mechanism.—The reflex nature of vomiting is shown by the frequency with which it is produced by the stimulation of sensory nerves and by injuries to various parts of the central nervous system. Disagreeable emotions and derangements of the equilibrium of the body, irritation of the mucous membrane of various parts of the alimentary canal, pathological states of the genito-urinary tract, and lesions or injuries of the brain may all cause vomiting. Vomiting may also be caused by direct action upon the medullary centres, as in the case of drugs—apomorphine and various narcotics—and by or in the toxæmia of the infections and autointoxications, as uræmia and cholæmia.

The causes are many, but the most common is irritation of the sensory fibres of the gastric mucous membrane. In this case the afferent path is by way of the sensory fibres of the vagus; the efferent path by way of the motor fibres innervating the muscles concerned in the act of vomiting, namely, the vagi, the phrenics, and the spinal nerves distributed to the abdominal muscles. It is now generally conceded that there is a definite vomiting centre situated in the medulla in close proximity to the respiratory centre.

The readiness with which children vomit is due in part to the greater lex excitability of the nervous system in early life; in part to the posi- n of the stomach, which is more nearly vertical than in adults. The developed state of the fundus and the defective closure of the cardia rease the liability of infants to vomiting, which often occurs without ort as a mere regurgitation of a portion of the food upon change of sture or slight pressure upon the epigastrium.

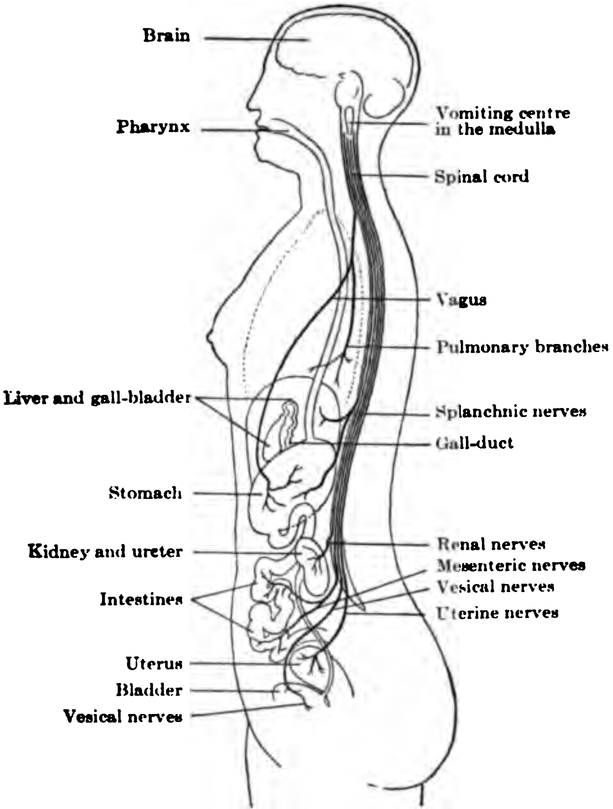


Fig. 200.—Diagram of afferent nerves which may excite the vomiting centre. Modified from Brunton.

1. **Vomiting from Direct Irritation of the Terminal Fibres of the Vagus the Stomach.**—Vomiting from this cause is very common. It may sult from anatomical lesions of the stomach itself and from quantitative d qualitative abnormalities of the contents of the organ. Vomiting a common phenomenon in various forms of gastritis. In acute gastric tarrh there is vomiting of the gastric contents followed by mucus en stained with bile; a sense of relief is then experienced. In chronic stric catarrh vomiting is common; it occurs at various intervals after e taking of food. Frequently, and especially in the gastric catarrh of oholic subjects, there is distressing vomiting of tough mucus on rising—*nitus matutinus potatorum.* Vomiting is common in peptic ulcer of the

stomach and is frequently provoked by the intake of food, which also causes pain. The pain very often precedes the vomiting and is relieved by it. The vomiting which attends carcinoma ventriculi is a common and distressing symptom. It is not often present until the disease has made considerable progress. It may occur when the stomach is empty, but usually follows the ingestion of food, after varying intervals. When the growth involves the cardia food may be immediately vomited; when the pylorus, after an interval of several hours. Vomiting may be absent in carcinoma of the fundus or lesser curvature. In stenosis of the pylorus from carcinoma or other cause food is retained in the stomach, which gradually undergoes dilatation, and is vomited after some hours or a day or two—*retention vomiting.* The vomiting of large quantities of fluid after considerable intervals of time is characteristic of gastric dilatation. Vomiting does not occur in gastrectasis of slight degree and in the extreme cases, by reason of the impaired contractility of the wall of the stomach, may wholly cease—an unfavorable symptom. Vomiting is a common symptom of cholera morbus and cholera Asiatica and may be regarded as the direct result of the inflammation of the gastric mucous membrane. It usually occurs after the diarrhœa, sometimes coincidently with it, scarcely ever before it. Vomiting in cholera is usually unattended with effort, is frequently repeated, and ceases or alternates with singultus in the algid stage. The vomitus is liquid and sometimes resembles the rice-water discharges. This symptom occurs in hyperacidity and hypersecretion and may be so persistent in cases of gastric hyperæsthesia that all food is promptly ejected. External pressure upon the stomach, as in pericarditis, ascites, or pericardial effusion, may cause vomiting.

2. **Vomiting from Central Irritation of the Vagus.**—To this cause must be referred the vomiting which is so common in diseases of the brain and its membranes—anæmia, hyperæmia, concussion, sea-sickness, Ménière's disease, tumor, abscess, and various forms of meningitis. So-called cerebral vomiting is characterized by the absence of nausea, its suddenness, projectile character, and the fact that it occurs independently of the taking of food. Vomiting of gastric origin is mostly followed by a sense of relief, while that dependent upon cerebral causes usually aggravates the symptoms, probably because of the mechanical disturbance produced by the act. Vomiting is an early and important symptom in tuberculous meningitis and cerebrospinal fever.

3. **Reflex Vomiting.**—The following forms are to be considered:

(a) Vomiting produced by irritation—tickling—of the base of the tongue or the fauces. Nausea, gagging, and vomiting are frequently caused by the unskilful use of the tongue-depressor or the laryngoscopic mirror. In the older medicine tickling the throat with a feather often played the part of an emetic. When the mucous membrane is abnormally sensitive, as in neurotic individuals or as the result of acute or chronic catarrh, very slight irritation of the fauces may cause vomiting. The vomiting of acute angina, that caused by efforts to dislodge tough masses of mucus, that attendant upon hypertrophy of the tonsils, and the vomiting which accompanies the paroxysm of pertussis must be referred to this group. The irritation caused by partially detached diphtheritic membrane sometimes produces efforts at vomiting which may have the favorable effect of wholly detaching the mass.

The vomiting of consumption is sometimes an early symptom; it is more common and troublesome in the later stages. It is frequently caused by severe paroxysms of coughing. The vomiting of phthisis may be cerebral, as from tuberculous meningitis, of which it is often an early and ominous symptom; due to pressure upon the vagi by caseous glands; the manifestation of irritation of the peripheral distribution of the vagi; pulmonary, pharyngeal, or gastric or mechanical, as from the succussion of urgent cough.

(b) The vomiting of peritonitis, which is frequently severe and intractable and always significant.

(c) That caused by irritation of the intestinal mucous membrane. In some instances the action of purgatives is preceded by vomiting. This symptom may attend intestinal parasites, colic, enterocolitis, appendicitis, strangulated hernia, intussusception, torsion, and ileus. In any form of obstruction of the bowel retroperistalsis may occur with vomiting, which gradually becomes stercoraceous.

(d) That attendant upon visceral diseases of various kinds, as biliary and renal colic, acute nephritis, pyelitis, cystitis, Addison's disease, and acute yellow atrophy of the liver.

(e) That which is symptomatic of disorders of the female sexual organs. Vomiting is common in anomalies of menstruation, uterine displacements, and pelvic exudates and new growths.

Of especial importance is the vomiting of pregnancy. A little mucus is thrown up with great nausea and effort when the patient rises in the morning. Commonly the vomiting does not recur until the next day; sometimes it is persistent and distressing. Usually it ceases after a few months. The pernicious form has been spoken of above.

(f) So-called nervous vomiting. The most typical form is that which occurs in hysteria. It depends upon the hyperæsthesia and abnormal motility of the stomach and upon quantitative and qualitative changes in the gastric secretions. The vomitus is often of large amount and consists of thin fluid. It is a notable fact that notwithstanding persistent vomiting hysterical patients lose little weight.

The persistent vomiting of Leyden is a form of nervous vomiting characterized by recurrent attacks coming on without obvious cause or as the result of slight indigestion, fatigue, or worry, and lasting from some hours to several days. The vomiting is copious and continuous; the abdomen retracted and the bowels constipated. There is epigastric pain together with intense headache and intolerance of light and sound. The pulse is frequent but there is no fever.

The gastric crises which occur in tabes, and less frequently in acute myelitis, disseminated sclerosis and paresis, are to be mentioned in this connection. Together with distressing pain there is vomiting, usually persistent and uncontrollable. Food is at first ejected, then a colorless stringy fluid and in some cases a blood-stained mucus. There is vertigo and a sense of sinking at the pit of the stomach. The attack lasts from some hours to two or three days. In the intervals there may be no signs of gastric disease.

The vomiting of migraine belongs to the category of nervous vomiting.

(g) Reflex vomiting may accompany diseases of the heart, especially myocarditis, fatty heart and angina pectoris. Vomiting due to cardiac disease is not infrequently associated with hiccough.

4. **Direct Irritation of the Centre for Vomiting.** — This form is less frequent. It arises under the following conditions:

(a) The action of certain emetics of which apomorphine is a type.

(b) The action of toxic substances in the blood, as for example those present in nephritis both acute and chronic. Vomiting is an early and ominous symptom in many cases of uræmia and not rarely the first indication of contracted kidneys. Uræmic vomiting occurs independently of the taking of food and is often severe and distressing.

(c) As an early manifestation of the toxæmia of the acute infections, especially in childhood. Vomiting may attend the stage of onset in scarlet fever, croupous pneumonia, diphtheria, and other acute febrile diseases.

The Gross Characteristics of the Vomitus.

The general appearance, quantity, odor, and reaction of the ejected material is of importance in diagnosis. These peculiarities depend largely upon the presence or absence of food in the stomach, its character and the time that has elapsed since its ingestion. When vomiting occurs directly after eating, the food shows little or no change. On the other hand, if some hours have elapsed there may be no trace of food. In retention vomiting, however, it is not uncommon to find particles of food taken at a previous meal or upon a preceding day. In sucklings the appearance of the vomited milk is of importance. The presence of curds indicates the presence of the milk-curdling ferment; an uncurdled milk some time after nursing shows the absence of normal gastric secretions and may be the sign of grave changes in the stomach.

Aside from the presence of food the following peculiarities are of diagnostic importance:

Watery Fluid and Mucus.—The vomitus may consist of a watery fluid containing little or no mucus. This is common in the morning in chronic gastric catarrh, especially that of alcoholic subjects. If the reaction is alkaline, the fluid usually consists of saliva that has been swallowed during the night and the vomitus consists largely of saliva in cases in which prolonged nausea has preceded the act of vomiting. If the reaction be acid the vomitus consists either of gastric fluid in excess—hypersecretion—or of food and mucus that have undergone acid fermentation. More commonly the vomited matter contains mucus and in some cases of acute and chronic gastric catarrh it is composed of masses of tenacious mucus. The vomiting of hyperacid gastric juice occurs in peptic ulcer of the stomach and in neurotic conditions, as migraine, hysteria, the gastric crises of tabes and exophthalmic goitre. In some cases of cholera the contents of the intestines are forced into the stomach and vomited, presenting the usual characters of the rice-water discharges and containing the comma bacillus of Koch.

Bilious Vomiting.—Bile is very commonly present, imparting a green or yellow color. It occurs after repeated or violent vomiting and is of no great diagnostic importance. The early vomiting of considerable amounts of bile occurs in some cases of peritonitis and intestinal obstruction.

Vomiting of Blood — Hæmatemesis — Gastrorrhagia. — This symptom occurs in a number of morbid conditions and is of great importance in diagnosis. The differential diagnosis between hæmatemesis and hæmoptysis has already been considered. The vomited blood may be bright red and fluid—a sign that it has remained in the stomach but a brief period; or it may consist of reddish or reddish-brown clots that have formed during a longer period; or finally it may present the appearance of coffee grounds, indicating that it has been subjected to the action of the gastric juice for a sufficient time to undergo partial digestion, with alteration of the hæmoglobin and destruction of the erythrocytes. In some instances a superficial resemblance to recent blood may be due to the presence of red wine or various reddish-colored fruits or the jellies or preserves made from them; in others altered blood—"coffee grounds"—may be suggested by the presence in the vomitus of coffee, cocoa, minute fragments of boiled or over-cooked meat, and certain drugs, as the preparations of bismuth and iron. As a rule these uncertainties may be settled by close inspection and an inquiry into the facts, but there are rare cases in which a chemical, microscopic, or spectroscopic examination may be necessary to determine the question. Bright red blood is usually vomited in considerable amounts and in association with small clots, while the altered blood which resembles coffee grounds is mixed with the vomitus in small quantities.

Not all blood ejected from the stomach is derived from the vessels of that organ. Blood is frequently swallowed and then vomited. In hæmoptysis a portion of the blood coughed up is often swallowed. Blood readily finds its way from the nasal chambers or pharynx into the stomach, especially when the patient is in the recumbent posture. The blood oozing from the bitten tongue in the epileptic paroxysm may be swallowed during the postepileptic stupor or the vomited blood may be derived from the vessels of the œsophagus. Blood may be swallowed by malingerers, who sometimes suck it from a wound made for the purpose in the mouth or upon the hand or forearm. In very rare instances infants vomit milk stained with blood derived from a fissured or ulcerated nipple.

Bleeding from the stomach occurs under various conditions, of which the following are important:

1. Circulatory Derangements.—Portal obstruction and the resulting passive hyperæmia of the gastric mucosa lead to hæmatemesis. This symptom therefore occurs in cirrhosis of the liver, in malignant and other tumors of the porta, and in adhesive pylophlebitis. Copious hæmatemesis, occurring in hepatic cirrhosis and terminating fatally, occasionally arises from rupture of the veins of an enlarged œsophageal plexus. The visceral congestions resulting from cardiac mural and valvular disease tend also to hemorrhage. Hæmatemesis is occasionally encountered in massive enlargement of the spleen.

2. HÆMIC DISORDERS. — Hæmatemesis is frequently symptomatic of the grave anæmias. It occurs in pernicious anæmia, leukæmia, hæmophilia, scurvy, and purpura hæmorrhagica, in profound jaundice, and after extensive burns. It has been observed in phosphorus poisoning and in acute yellow atrophy of the liver.

3. THE INFECTIONS.—Vomiting of blood is of occasional occurrence in epidemic influenza, typhus, relapsing fever, and dengue. It is a prominent event in some forms of pernicious malarial fever, malignant variola, and yellow fever. In the last the vomiting of altered blood—*black vomit*—is characteristic.

4. TRAUMATISM.—Contusions of the epigastric region, as from a blow or kick, crushing, and other injuries, are sometimes followed by the vomiting of blood. The vomitus is often blood-streaked after prolonged or violent vomiting. Under this caption must be placed the direct injury to the gastric mucosa caused by the corrosive poisons, caustic alkalies, the mineral acids, arsenic, and the like.

5. SPECIFIC ANATOMICAL LESIONS OF THE STOMACH. — Cancer is a common cause of gastric hemorrhage. The blood is usually dark and altered and rarely profuse, slight oozing, either continuous or frequently repeated, being the rule. Even more common is gastric ulcer. The blood is usually abundant, bright red, and fluid. Copious hæmatemesis is suggestive of ulcer. Free and even lethal bleeding may occur in superficial erosions, and profuse hemorrhage may come from the erosions of the gastric mucous membrane which sometimes occur after operations upon the abdomen and especially in cases in which the omentum has been wounded. In gastric and duodenal ulcer, especially the latter, the blood may not be vomited but is passed in the stools. Miliary aneurism is a rare cause of gastric hemorrhage. It is not common for death to result directly from the bleeding, which is often repeated from time to time. Anæmia, frequently of high grade, results. Syncope with or without general convulsions may immediately follow profuse hemorrhage. Hemiplegia and amaurosis, which may be followed by optic atrophy, are rare sequelæ.

6. CERTAIN NERVOUS AFFECTIONS.—Hæmatemesis is an occasional event in hysteria, and cases of gastric bleeding have occurred in apparently healthy individuals in the absence of any local or general condition to account for it, and without a second appearance. This symptom is comparatively infrequent in epilepsy and in general paresis, and Schiff and others have directed attention to it as a rare phenomenon in local cerebral disease. In the newborn it may occur as an isolated symptom or with hemorrhage from other mucous tracts.

7. FATAL GASTRIC HEMORRHAGE may result from the rupture of an aneurism of the aorta or its branches into the stomach. Under such circumstances death may occur from blood loss without vomiting, the stomach being distended with blood.

Fecal or Stercoraceous Vomiting.—This is a significant symptom in acute obstruction of the bowel. The anatomical condition may be strangulation, intussusception, volvulus, or abnormal contents. The last of these—fecal masses, biliary calculi, and enteroliths—may cause acute obstruction by the sudden shifting of their position. Vomiting comes on early and is persistent. The vomitus consists at first of the contents of the stomach, then of bile or bile-stained material, and finally of a brownish or blackish fluid of a distinctly fecal odor. In this fluid masses of fecal matter may be present. Retroperistalsis not rarely occurs in peritonitis and in some cases stercoraceous vomiting is the result of a gastro-intestinal fistula. Chronic intestinal obstruction is not usually attended by this form of vomiting even when of high grade. In the terminal paroxysms, however, it may occur.

Purulent vomiting is rare and not usually dependent upon primary disease of the stomach; it may, however, occur in phlegmonous gastritis. The more common cause is perforative ulceration of the wall of the stomach in hepatic abscess or empyema.

Parasites in the Vomit.—The *Ascaris lumbricoides* occupies the upper part of the small intestine. From this position it finds its way readily into the stomach and is often ejected with the vomit. In rare cases the segments of tænia are present in vomited matter and the hooklets and fragments of echinococcus cysts have been observed; so also trichinellæ and the larvæ of insects.

The quantity of the vomitus depends upon the volume of the stomach contents and the intensity of the act of vomiting. Very significant is the retention vomiting of pyloric obstruction and the large fluid vomiting in gastrectasis from other causes.

The Odor.—The vomit is commonly sour-smelling and often intensely acid. It is ammoniacal in uræmia and fecal in acute intestinal obstruction and in some cases of peritonitis. The odor of the vomitus in poisoning is sometimes of great diagnostic importance. Striking examples are carbolic acid, the garlicky smell in phosphorus poisoning, that of bitter almonds in poisoning by hydrocyanic acid and nitrobenzole, the vinegar-like odor in poisoning by acetic acid, and the smell of ammonia; less significant are the odors of alcohol or laudanum.

The reaction is commonly acid. Where there is an excess of saliva, bile, or blood the reaction is alkaline. In hypersecretion the reaction is intensely acid and patients speak of their teeth being set on edge by the taste. In uræmia the reaction may be alkaline.

The taste of the vomitus is, according to the patients, commonly sour and when bile is present, bitter. Blood imparts a salty or sweetish taste.

DEFECATION.

Significance of Abnormal Defecation.—The indigestible parts of the food, with débris, bacterial masses, and secretions from the intestinal tract, pass slowly through the large intestine and reach the sigmoid flexure, in which they accumulate. As the semisolid or solid material passes into the rectum it stimulates the sensory nerves of that part of the intestine, giving rise to a peculiar sensation and desire to defecate. This material is retained in the rectum by the two sphincter muscles, the internal of which is a band of the circular layer of involuntary muscles of the rectum. Upon the passage of fecal matter into the rectum the internal sphincter passes into a condition of tonic contraction, the relaxation of which marks the beginning of the act of defecation. The internal sphincter is composed of unstriped muscular fibre and receives its innervation from the sympathetic system and from the sacrospinal nerves. The external sphincter ani is made up of striated muscular fibres and is to a large extent under the control of the will. Upon intense rectal stimulus the voluntary control is overcome and this sphincter is also relaxed. The act of defecation is therefore in part voluntary and in part involuntary. The voluntary factor is made up of the inhibition of the external sphincter and the simultaneous action of the abdominal muscles, the diaphragm being contracted and the glottis closed. Pressure is thus exerted upon the abdominal and pelvic viscera, with the result that the contents of the descending colon and sigmoid flexure are forced into the rectum. This pressure is augmented by deep inspiration and fixation of the respiratory muscles. The involuntary factor consists in the contraction of the muscles of the rectum, in particular the circular layer, and the relaxation of the internal sphincter, in part the result of reflex stimulation from the lumbar cord and in part from automatic peristaltic movements. The action of defecation is essentially, however, an involuntary reflex, as is well seen in infants and in soporose states.

Under normal conditions the bowels move once a day, the act being, like sleep and the taking of food, of diurnal rhythmical recurrence. There are healthy individuals, however, in whom the rhythm is not diurnal, but at intervals of two or three days or exceptionally longer, and in whom efforts to bring about the diurnal movement by means of purgatives are followed by manifest derangements of health. The normal periodical movement of the bowels is maintained by the observation of a fixed hour for this function, and various derangements, especially constipation, result from the neglect of this rule.

It is important for the physician to inform himself as to the periodicity, frequency, and character of the bowel movements and in certain cases to inspect the stools. Departures from the normal in respect of this function relate to constipation, diarrhœa, tenesmus, painful defecation, fecal incontinence, and the character of the discharges.

CONSTIPATION.

Constipation—costiveness—infrequent or difficult evacuation of fæces; retention of fæces. This condition is of great and varied diagnostic importance. Its cause may be constitutional or intestinal. Very often several causes are associated.

The more important constitutional or general causes of constipation are:

1. Personal peculiarity: Sluggishness of the bowels is frequently an hereditary and family tendency. It is far more common in persons of dark than in those of fair complexion and is especially associated with the traits that constitute the bilious temperament.

2. Unhygienic habits, as want of proper exercise, the failure to observe regularity in the hour of defecation or to devote to the act sufficient time, irregularity or undue haste in meals and the eating of unwholesome food or of excessive quantities of food. From this point of view constipation is primarily not a condition of the body but a condition of the mind. On the

her hand too little food or a diet consisting largely of proteid substances which contains a minimum of undigested residuum tends to constipa-on. It is obvious that a sufficient bulk of residual material is required to orm the fecal mass and excite peristalsis. The insufficient ingestion of luid tends also to cause constipation.

3. Dehydration of the tissues by profuse and frequently repeated weating, diuresis from the action of drugs, the polyuria of diabetes insip-lus and mellitus, or repeated hemorrhages is attended by constipation.

4. The febrile infections, except those in which diarrhœa is an especial mptom, are characterized by a tendency to constipation. Even in ese affections constipation very often gives way in the later course of the tack to diarrhœa, and the latter may assume the guise of a critical dis-arge, as sometimes occurs in croupous pneumonia.

5. The habitual use of purgative drugs is a fruitful cause of constipation.

6. Constipation is a very common condition in the anæmias, especially chlorosis, and is often a troublesome symptom in neurasthenia and steria.

7. General asthenia and cachectic states are very often attended by nstipation; so also conditions in which the abdominal muscles are over-stended and their contraction hampered, as obesity, ascites, large ab-minal tumors, and pregnancy.

Among local causes of constipation the following are to be considered:

1. Alterations in the quantity and quality of the intestinal juices and deficiency of bile or pancreatic secretion. Under these circumstances nstipation may be an important symptom of fever, chronic diseases of the stro-intestinal tract, and diseases of the liver, biliary passages, and the ncreas. It is to be borne in mind that the normal presence of bile in the testine constitutes a powerful stimulus to peristalsis.

2. The motor mechanism of the intestine may be at fault. The defect ay be nervous, as in organic disease of the nervous system—myelitis, eningitis, and tetanus, or functional, as in hysteria and neurasthenia. r the defective intestinal innervation may be the manifestation of a general thenia. The arrest of peristalsis and tympanites in severe enteritis, me cases of appendicitis and in peritonitis and acute pancreatitis are imarily due to derangements of the nerve-supply to the bowel, second-ily to paresis of its muscular wall. Chronic intestinal catarrh and portal ngestion from hepatic or cardiac disease are often attended by constipation e to impaired nutrition of the muscular coat of the bowel. Atony of the lon and especially of the muscular wall of the sigmoid flexure is an im-rtant local cause of constipation. Dilatation of the colon is attended ith constipation. Large collections of scybala may accumulate in the gmoid flexure and be felt upon palpation of the abdomen. Constipation e to this cause is encountered in neurasthenia and hysterical persons d is common in the insane. It occurs also in bed-ridden and elderly dividuals.

3. Local disease of the rectum or anus or of adjacent organs is a com-on cause of constipation. When such conditions, as is usually the case, nder the act of defecation painful, the patient is apt to postpone it unduly d there is very often reflex spasm of the sphincters which renders it for

the time impossible. Under these circumstances fecal material accumulates in the rectum and sigmoid flexure of the colon and greatly adds to the discomfort of the patient. Such local disorders are inflamed hemorrhoids, anal fissure, irritable ulcer, prostatic inflammation or abscess, and a tender retroverted uterus or prolapsed ovary.

4. Constipation is observed in malignant disease of the œsophagus, pylorus and bowel and in other chronic conditions in which a minimum of food is ingested or that which is taken cannot pass onward or is persistently vomited.

5. This symptom may be due to a contracted condition of the bowel—so-called spasmodic constipation. The narrowing of the bowel may be the result of ulcerative colitis or dysentery; a manifestation of hysteria or of the atrophic processes of advanced life. The bowel may be in a condition of permanent contraction or spasm at one part and dilated elsewhere. The stools are small and sausage-shaped or they may be liquid with hard scybalous masses varying in size from a marble to a walnut. Spasmodic constipation occurs in the pelvic disorders of women and in chronic lead poisoning.

6. Strangulated hernia is attended with acute constipation. Laxatives are without effect. There are vomiting and abdominal distention. Pain is usually present. Similar symptoms attend volvulus and other forms of intra-abdominal strangulation. In intussusception, the sausage-like tumor, tenesmus, bloody mucus, and a relaxed anus are significant. Acute retention of fæces with the signs of obstruction demands very careful and systematic examination of the abdomen, a digital exploration of the rectum, and examination of the hernial rings.

Chronic intestinal obstruction may be due to foreign bodies, very large gall-stones, tumors within the gut or exerting pressure upon its wall, masses of scybala, and strictures of every kind. The constipation is gradually developed; occasionally interrupted by watery diarrhœa and sometimes by attacks with the symptoms of acute obstruction. Three facts are of great importance: First, that fluid fecal matter may work its way past the obstruction from time to time; second, that the dilated and congested bowel below the obstruction may discharge a thin fecal-stained mucus; and, finally, that both these conditions are occasionally mistaken for diarrhœa.

7. Constipation in infants. Constipation in the new-born may be due to imperforate anus or a congenital stricture. In some cases it results from dilatation of the colon, which may attain enormous dimensions, or it may be due to simple atony of the large bowel.

Constipation in sucklings and especially in bottle-fed infants is often due to deficiency of the intestinal secretions, the fæces being dry and hard. This condition has been attributed to insufficient water and a deficiency of fat in the food. In older children attention to the hour of defecation and regular habits are as important as in later life. Constipation often results from enterocolitis, from impairment of the contractility of the muscular wall and derangement of the normal secretions. Acute constipation is frequently symptomatic of mechanical obstruction by foreign bodies, hardened and impacted fæces, twists, and intussusception.

Associated Symptoms.—Sensations of pressure and distention in the abdomen, uneasiness and pain, especially in the course of the transverse colon, loss of appetite, a furred tongue, a disagreeable taste, and uneasy precordial sensations are common. Patients very often attribute these phenomena to derangements of the liver or stomach. An effectual purge is of diagnostic importance. The results very often show that these symptoms are due to constipation.

Of especial importance are the morbid phenomena in the distribution of the hemorrhoidal veins that result from constipation. Pain before and after defecation, protrusion of the dilated blood-vessels, bleeding and the discharge of stringy mucus are common. Paroxysmal neuralgic pain referred to the coccyx or the suprapubic region or to the inner aspect of the thigh are less frequent. Gastric catarrh with manifold symptoms and occasional implication of the duodenum and bile passages also occurs. In some instances catarrhal jaundice results and in chronic constipation a slight icteric discoloration of the conjunctiva is often seen.

Constitutional derangements are not less common. They consist of headache, vertigo, depression of spirits, disinclination for work, and debility. Actual neurasthenia with the most varied and depressing symptoms may result from obstinate and prolonged constipation. It is on the other hand important to bear in mind that nervous disease is a frequent cause of constipation and that the most troublesome constipation may occur, for example, in hysteria. Under such circumstances a vicious circuit is established, the constipation aggravating and intensifying the symptoms of the disease of which it is itself a symptom.

The duration of constipation is largely dependent upon its cause. Simple forms resulting from neglect of hygienic laws may last three or four days; more troublesome cases may resist usual treatment for weeks. Stubborn constipation with severe symptoms suggests mechanical obstruction of the bowel. The passage of flatus is a favorable sign. In transient constipation the indican in the urine is not increased; in chronic obstruction it is apt to be increased.

DIARRHŒA.

Abnormal frequency and diminished consistence of the stools. This symptom is of the most varied significance. It results from increased peristalsis, particularly when the large intestine is affected, from diminished absorption of the contents of the bowel, from an excess of fluid in the bowel either in consequence of hypersecretion of the substances entering into the formation of the *succus entericus* or of transudation of serum, and in rare instances from direct abnormal communication between the stomach or small intestine and the colon.

Diarrhœa may therefore be symptomatic of deranged innervation of the bowel, mechanical or chemical irritation by its contents, the action of toxic substances, either in the bowel or in the blood-current, as in poisoning, autointoxication or the infections, defective nutrition or circulatory derangements of the wall of the bowel, or local disease, as ulceration or new growths in the bowel itself or adjacent organs.

Diarrhœa may be primary or secondary or it may be acute or chronic. The number of stools varies from 3 or 4 to 30 or more in the course of twenty-four hours, their consistency from semisolid to watery, and their color, odor, and other physical characters vary within equally wide ranges (see pp. 518, 519).

The recognition of the following forms of diarrhœa is essential:

1. Nervous diarrhœa. This symptom may denote mere increase of peristalsis in the absence of any lesion of the intestine, in hysteria, neurasthenia, the intestinal crises of tabes, exophthalmic goitre, Addison's disease, movable kidney, in the first dentition, and in emotional disturbances in healthy individuals of neurotic temperament. The characteristic manifestations of the underlying nervous disorder are of diagnostic importance. The stools are of gruel-like consistence and contain nothing of pathological importance. The attack begins abruptly and is of short duration.

2. Irritation of the intestine secondary to constitutional conditions. Diarrhœa may occur in uræmia, hyperpyrexia, extensive burns, sudden chilling of the surface, certain infectious conditions, as malaria and septicæmia, and as the result of the subcutaneous injection of such purgatives as podophyllin or magnesium sulphate. The urine should be examined in every case.

3. Increased intestinal fluid. The stools are abnormally frequent and watery after the administration of the hydragogue cathartics and in cholera nostras and Asiatica.

4. Irritation of the intestine by various ingesta, or pathological bowel contents. Abnormal peristalsis and looseness of the bowels is caused by indigestion, intestinal parasites, local fecal accumulations, poisoning by the salts of mercury, antimony, arsenic, copper and so forth, the purgative drugs, organic acids derived from the food or from its decomposition in the stomach or intestines, mushroom poisoning, unaccustomed or improper articles of diet, bulky or indigestible food, large quantities of cold food or drink, or the administration of enemata. In all cases the anamnesis and physical examination are of diagnostic importance.

5. Abnormal irritability of the bowel. Diarrhœa may be the manifestation of an idiosyncrasy, and is symptomatic of catarrhal inflammation and of ulcerative processes of all kinds, from superficial erosions from mechanical irritation to the specific ulcerations of enteric fever, dysentery, or tuberculosis.

6. Impaired absorption. Diarrhœa is not rarely due to extensive ulceration or atrophy of the mucosa, amyloid disease, and portal congestion. The diarrhœa of tabes mesenterica is largely due to failure in fat absorption.

7. Mucous colitis — membranous enterocolitis. This syndrome is characterized by paroxysmal diarrhœa accompanied by severe hypogastric or left iliac pain and the discharge of masses of mucus or membranous casts of the bowel. The attack occurs at varying intervals, and the disease is observed in neurotic persons, usually women.

8. Under very unusual circumstances a fistulous communication between the stomach or upper part of the intestine and the colon—usually its transverse part—may be the cause of diarrhœa with stools

containing undigested food and conversely of the eructation of intestinal gas or the vomiting of fecal material.

9. Lienteric diarrhœa. Normal stools are usually more or less homogeneous. They frequently, however, contain such indigestible articles as seeds, husks, the capsules of berries, fruit pits, and the like. The diarrhœa caused by excessive quantities of food or the ingestion of food that cannot be digested, or which attends forms of enteritis that interfere with normal digestion is characterized by the presence in the stools of undigested or only partially digested particles of food and is known as lienteric. Fragments of food may be recognized in the stools shortly after it has been eaten. This form of diarrhœa may be acute, as after errors in diet or acute enteritis, or chronic, as in chronic intestinal catarrh.

Associated Symptoms.—Diarrhœa is often unattended by any symptom other than the frequent recurrence of the peculiar sensation which invites to the closet. Usually there is uneasiness in the abdomen, which may be associated with local or general pain, often colicky, and tenderness. Severe diarrhœa is attended with thirst, appetite is impaired, and there is debility proportionate to the urgency of the intestinal symptoms. Local or general tympanitic distention of the bowel also occurs. Vomiting is common, especially in the diarrhœas of infancy. The loss of fluid not only causes thirst, but may give rise to faintness, collapse, cramps of the muscles, subnormal temperature, diminution of urine even to suppression and albuminuria. The acidity of the urine is increased and it gives the reaction for indican.

TENESMUS.

Rectal tenesmus—painful, ineffectual, and usually long-continued straining at stool. This symptom occurs alone, but it is very often associated with vesical tenesmus, partly because of the anatomical relationship of the organs, partly because of the common action of some of the causes. It consists of spasm of the musculature concerned in defecation and micturition. The violent spasmodic contractions are repeated at short intervals and are attended with such distress that in extreme cases children fall into general convulsions and adults faint. The discharge consists of small amounts of stringy, sometimes bloody, mucus from the anus or a few drops of urine as the case may be. Rectal tenesmus occurs in the course of irritating lesions of the rectum and anus, whether these be primary or secondary. It is a symptom of intussusception, dysentery, polypus, adenoma and malignant tumors of the rectum and sigmoid flexure, proctitis and periproctitis, hydatid cysts of the pelvis, mechanical injuries to the rectum by foreign bodies, or in exceptional cases in highly neurotic persons it may follow digital or instrumental examination. Tenesmus is not a common symptom of hemorrhoids or fissure of the anus. It may be caused by impacted fæces, masses of round worms, the presence of foreign bodies, and, in connection with vesical tenesmus, by stone in the bladder. It is also a distressing symptom in acute inflammation and abscess of the prostate gland. Tenesmus is easily recognized. Its cause may be obscure. When it is violent or persistent a digital or proctoscopic examination should be made under local or general anæsthesia.

PAINFUL DEFECATION.

The pain may be such as to cause fecal impaction from voluntary postponement of the act. The passage of a large hard fecal mass is attended with pain under normal conditions. In proctitis, inflamed hemorrhoids, fissure of the anus, prolapsus, irritable ulcer, and malignant disease of the rectum pain upon defecation is a conspicuous symptom. It is usually present in inflammation of the prostate and is sometimes symptomatic of acute inflammatory affections of the pelvic organs in women.

FECAL INCONTINENCE.

This symptom may be due to local causes, as laceration of the perineum involving the anal sphincters, surgical over-stretching, and malignant or syphilitic disease of the rectum; more commonly it is due to general conditions which profoundly affect the nervous system, as coma from any cause, myelitis and other diseases of the spinal cord, grave chorea, convulsive diseases, as epilepsy, tetanus, and strychnine poisoning, and certain severe infections, as enteric fever, dysentery, cholera Asiatica and nostras and cholera infantum. Involuntary discharges very often precede dissolution. The unclean habits of some forms of insanity cannot be placed in this group of symptoms.

THE GROSS PHYSICAL CHARACTERS OF THE STOOLS.

The fecal discharges of the healthy adult are of brownish color, cylindrical form, soft solid or semisolid consistency, 150 to 200 grammes in daily quantity, usually neutral or faintly alkaline in reaction when passed, and emit the offensive characteristic odor.

Abnormal variations in these respects constitute diagnostic criteria of some importance. The macroscopic examination is too often neglected. Laboratory investigation is sometimes necessary (see p. 217).

1. The color, which is due to the presence of altered bile pigment, principally hydrobilirubin, may be modified by certain articles of diet or by drugs. It may be rendered black by blackberries or by iron, manganese, or bismuth; yellow by rhubarb, [illegible], senna, or santonin; green by spinach or calomel, or by certain chromogenous bacteria. In sucklings and others who subsist upon an exclusive diet of milk the fæces are golden yellow or whitish; in those who live largely on meat they are brownish-black in color, and this is also the case with fecal matter long retained in the bowel as in constipation. In jaundice due to obstruction they are grayish or putty-colored. When they are increased and thinned by intestinal hypersecretion or transudation their color is usually light brown or yellowish; when very watery, as in cholera, they are of a dirty-gray color—"rice-water" stools. The presence of blood colors the stools red or black: red when the blood comes in considerable quantity and not long after its escape, from the ileum as in enteric fever, or when it comes from the lower bowel as in dysentery, or from the rectum as in piles; black

when it is derived from the upper regions of the gastro-intestinal tract as in peptic ulcer, or when it is thoroughly mixed with the stool.

2. The form is lost in diarrhœa, the discharge being gruel-like or watery in consistence. The normal cylindrical or sausage-shaped stool may be modified in various conditions of the lower bowel. In prolapsus ani or stricture of the rectum, more rarely in intussusception, the diameter may be much narrowed—*pipe-stem stools;* in stricture or cancer of the rectum or the pressure of an enlarged prostate gland or abscess or in large pelvic tumors impinging upon the rectum the stools may be flattened or *ribbon-shaped;* in constipation from any cause, but especially that which results from atony and distention of the colon, they often assume the form of irregular, round, hard masses like the dung of sheep—*scybala.*

3. The consistence is increased in constipation. The fluid is resorbed and the mass tends to become hard and dry. The consistence is diminished in diarrhœa. Serous stools are observed in cholera Asiatica, cholera nostras and cholera infantum; in poisoning by antimony, arsenic, and mushrooms. Small, dribbling, serous stools occur in some cases of intestinal obstruction from cancer and other causes. Serous stools contain little or no fecal matter.

4. The quantity varies greatly. It is diminished when the diet is concentrated or consists principally of meat; increased when the diet is argely made up of starchy and vegetable foods. The amount voided at one effort depends of course upon the frequency of the act and may attain n cases of constipation as much as 1000 grammes. The quantity in diarrhœa is increased by the hypersecretion of the intestinal glands and the transudation of serum from the blood-vessels. In starvation the total quantity may not exceed 90 grammes a day.

5. The reaction and odor. The reaction is faintly acid in nursing nfants and alkaline in some forms of intestinal fermentation. The acidity s due to carbohydrate fermentation or the presence of fatty acids. The reaction is of no great diagnostic value. Depending upon the amount of proteid decomposition and the putrefactive bacteria present, the odor of the stools is more or less offensive. Diets that allow much proteid residue to reach the large bowel usually give foul-smelling movements. A milk diet in health gives an almost odorless stool. Indol and skatol, derivatives of proteid decomposition, are mainly responsible for the characteristic fecal odor.

The odor in healthy infants is sour and unlike the fecal odor of the stools of adults. The so-called "albuminous decomposition" in the fæces of infants and the resulting putrid odor are due to the decomposition of the undigested proteid of the milk in the large intestine. In cholera infantum it is sometimes faintly musty, sometimes suggestive of the washings of meat. In the absence of bile the stools have a peculiarly offensive odor.

The presence of milk curds in the stools of infants indicates an error in the quantity or quality of the food and is one of the earlier symptoms of enterocolitis; the presence of curds in the stools of adults who are taking a milk diet, as in enteric fever, constitutes an indication for the use of alkalies in connection with the milk or a reduction in its quantity.

Abnormal Substances in the Stools.—In lienteric diarrhœa the stools contain undigested particles of food. Other abnormal substances are by no means uncommon and may be of great diagnostic importance. Among these are mucus, blood, pus, fat in excess, gall-stones, intestinal sand, concretions, intestinal parasites, sloughs, and foreign bodies that have been swallowed.

Mucus.—Minute particles of mucus may be observed upon the surface of the formed stools in health. Large quantities covering the stools or expelled with them in masses indicate a deranged secretion of the mucous glands of the colon or rectum. Masses of mucus that may be shaken out in water into sheets or tubular casts of the intestine are diagnostic of membranous colitis. Mucus intimately admixed with the fecal matter may come from the small bowel. Mucus in the stools is symptomatic of mechanical or pathological irritation of the bowel and is seen in such conditions as impacted fæces, foreign bodies, intestinal parasitism, new growths, intussusception, and all forms of dysentery, enterocolitis, and proctitis.

Blood.—A distinction is made between "hemorrhage from the bowel" —the discharge of red blood unmixed with fecal matter—and "melæna"— blood intimately mixed with the fæces and occurring in the form of "tarry" or pitch-like masses, usually of semisolid consistence and glistening appearance. The difference consists chiefly in the time the blood remains in the intestine and therefore in general terms indicates the portion of the gut into which it has been discharged. If, as in the case of peptic ulcer of the stomach or duodenum, the hemorrhage has been high up in the intestinal tract, the blood remains a considerable time in the bowel, and is subjected to mechanical conditions by which it is incorporated with the fecal contents, undergoing at the same time a sort of digestion by which its physical characters are much changed. If, on the other hand, the blood is poured out lower down in the bowel and under the influence of an active peristalsis is speedily evacuated, it maintains the characteristic appearance of fresh blood, often bright red and sometimes commingled with recently formed clots. The appearance of the evacuations therefore is of diagnostic importance in this respect. On the other hand, a copious hemorrhage from the ileum, as in enteric fever with active peristaltic movement, usually shows itself in the discharge from the bowel of bright red blood, while a slow oozing from the colon with tardy onward propulsion in the bowel may appear in the stools as "coffee-grounds" or even as "tarry" material.

Blood is frequently present in the stools in quantities so minute that its presence can only be detected by chemical examination—occult blood (Part II, p. 220).

The more important causes of blood in the stools are portal congestion, ulceration of the intestinal mucosa, neoplasmata and in particular malignant disease of the gut, intestinal parasites, embolism of the mesenteric arteries, intussusception, and traumatism.

1. *Portal Congestion.*—This occurs in cirrhosis of the liver, portal thrombosis, and dilatation of the hemorrhoidal veins—piles. The diagnosis of hemorrhoids rests upon the habitual or occasional discharge of bright red blood with the stools, the appearance and habits of the patient, and the signs obtained upon inspection or a digital examination.

2. *Ulcerative Processes in the Bowel.*—Intestinal hemorrhage occurring in the course of an attack of enteric fever is of positive diagnostic importance. It means the erosion of an arterial twig in an ulcer. If the hemorrhage is profuse it may at once lead to collapse with the associated symptoms of internal hemorrhage; if slight, the stools may be tarry or contain slight amounts of red blood without symptoms. In either case the appearance of blood in the stools is of prognostic importance, since it denotes deep ulceration which may be followed in a day or two by a more abundant blood loss or by perforation. Other ulcerative processes that lead to the appearance of blood in the stools are dysentery, syphilis, and tuberculosis. Under these conditions the blood appears in the form of streaks or stripes upon the stools or admixed with mucus or pus. Dysenteric stools may present the appearance of meat washings or of masses of blood commingled with liquid fecal matter. The mere presence of blood in the stools does not under ordinary circumstances justify a diagnosis. The anamnesis and a systematic investigation of the present condition of the patient are necessary.

3. *Malignant Disease of the Bowel.*—Blood in the stools is in many cases the first symptom to attract the attention of the patient to carcinoma. The stools are not, however, characteristic, and a systematic examination, which may reveal the presence of an abdominal tumor, is necessary. General failure of health, secondary anæmia, signs of intestinal obstruction, and cachectic phenomena are confirmative.

4. *Intestinal Parasites.*—The *Ankylostomum duodenale* is a common cause of persistent melæna among workers in the soil and miners. Griesinger first drew attention to this parasite as the cause of Egyptian chlorosis. The worms infest the upper portion of the small intestine and are very abundant in the jejunum. The diagnosis rests upon the prevalence of the condition among workmen in tunnels, brick-yards, excavations, and the like, and the presence of the ova in the stools.

5. *Embolism of the Mesenteric Arteries—Infarction of the Bowel.*—In consequence of valvular lesions of the heart, but with no great frequency, embolism of this distribution may occur. It is probable that the occlusion of small vessels produces no symptoms of importance and that the circulation may be reëstablished. If the superior mesenteric artery is occluded, or a large branch, the symptoms are sudden collapse, violent colicky pains, signs of peritonitis, and thin, blood-tinged stools or hemorrhage from the bowel.

6. *Intussusception.*—This affection occurs in infancy and childhood. Bloody stools are of diagnostic importance since they occur in at least sixty per cent. of the cases either spontaneously or after the administration of an enema. The blood is commonly mixed with mucus. Associated symptoms are tenesmus and a sausage-shaped tumor in the line of the colon. Vomiting and tympany are less common.

7. *Traumatism.*—Injuries of the bowel as a cause of bloody stools commonly involve the rectum, and when they do not penetrate the peritoneum may be readily overlooked. The abundant venous supply favors free bleeding, and, since the blood is often retained in consequence of spasm of the sphincters, the signs for a time may be simply those of inter-

nal hemorrhage and collapse. The anamnesis is of importance and a digital examination reveals the actual condition. The presence of foreign bodies may be thus discovered in children, idiots, and insane persons.

8. *Constitutional Conditions.*—Intestinal hemorrhage is occasionally symptomatic of leukæmia, hæmophilia, purpura hæmorrhagica, and scurvy. This symptom is, however, so closely associated with the general phenomena of those diseases that it is of secondary importance in their diagnosis.

9. *Miscellaneous Causes of Intestinal Hemorrhage.*—Bloody stools are of infrequent occurrence in consequence of the rupture of an aneurism of the abdominal aorta into the bowel, jaundice, acute yellow atrophy of the liver, phosphorus poisoning, yellow fever, pernicious malarial fever, and very rarely septicæmia.

CONCEALED HEMORRHAGE. — Concealed intestinal hemorrhage may occur in the foregoing conditions. If small it may give rise to no symptoms, although prolonged and unsuspected bleeding may be the cause of profound secondary anæmia with its usual symptoms; if large the hemorrhage, while not for a time appearing at the anus, occasions the symptoms of internal hemorrhage,—namely, collapse, restlessness, air-hunger, pallor, a pinched face, cold extremities, a rapid, weak, even imperceptible pulse, urgent thirst, and a tendency to syncope.

Pus.—In small quantities pus may be present in the stools in dysentery, enteritis, colitis, proctitis, and in ulceration of the colon or rectum due to malignant growths or syphilis. Small amounts of pus may be present in the stools in profuse leucorrhœa or urethritis; but under these circumstances its appearance is without diagnostic importance, since the associated symptoms will fully explain it. In large quantities and usually in single discharges, or in large quantities at irregular intervals, pus may be present in the stools in consequence of the rupture of an abscess, or the establishment of a fistulous communication between a purulent collection and the bowel. Such abscesses are usually periproctic, pelvic, or perinephric; sometimes appendicular; and, less commonly, in the gall-bladder, hepatic or infradiaphragmatic.

Fatty Stools.—The appearance of the discharges is greasy and glistening. An excess of neutral fat is present in obstructive jaundice and in various forms of pancreatic disease. Fatty diarrhœa, with icterus and sugar in the urine, has been observed in acute suppurative pancreatitis. Over-feeding and indigestion in infants may be the cause of fatty stools, and Biedert has described a fat diarrhœa in which the percentage of fat is enormously increased. The condition is primary in which the ingestion of fat is excessive and which may be corrected by modification of the food, and secondary which is due to catarrhal inflammation of the intestine or disease of the pancreas.

Gall-stones—Biliary Calculi. — Gall-stones have been found to be present in Europeans in from 5 to 10 per cent. In the East gall-stones are said to be extremely rare. Gall-stones vary in size from a concretion barely perceptible to the naked eye to the size of a walnut or larger. They are spherical, oval, or angular, the surface being smooth, mammillated, or faceted. When large they are commonly single; when small they may number hundreds. In a case of mine the small stones numbered by actual count 300. When extremely small they are described as biliary or intestinal sand. Their color varies from a whitish-gray to dark yellow or brown, sometimes black. Their consistence is usually firm, but they are often friable, being crushed by pressure between the thumb and forefinger, with crystalline fracture. In some cases, however, they are extremely hard.

Intestinal Sand.—Small brown or green calculi, spherical or irregular in shape and of rough surface, and varying in size from grains of sand to small shot, are sometimes present in the stools in considerable quantity. This material may or may not be preceded by attacks of colic. These calculi are of variable composition. They consist in some instances of inorganic salts, as calcium carbonates and phosphates, magnesia and iron, together with organic matter, bacteria and urobilin. Cholesterin is not present. A nucleus may sometimes be demonstrated. It is formed of a grain of quartz sand or a minute particle of the case of a fruit seed. In other very rare cases calcium sulphate has been the chief constituent. This form of intestinal sand occurs in intestinal neuroses of the secretory type.

Pancreatic Calculi.—Kinnicutt has recently studied the subject of the discharge of pancreatic calculi during life. The decisive evidence of pancreatic lithiasis consists in the presence of the characteristic concretions in the stools. They are composed chiefly of calcium carbonate. They are extremely rare—a fact due in part to the small size of the calculi and their friability, so that they may be voided in fragments or particles not easily recognized.

Intestinal Concretions — Enteroliths. — Concretions of various kinds occur in the stools. They are comparatively rare. The following forms are encountered:

1. Hard round fecal masses—scybala. They occur in chronic constipation, especially in elderly people, and in cases in which after abdominal operation partial obstruction of the bowel occurs as the result of adhesions.

2. Enteroliths. Earthy concretions are sometimes observed in the stools. They are largely composed of magnesium phosphate, the alkaline carbonates, and organic matter. They are hard, dense, and made up of concentric layers about a chalky nucleus that very often surrounds a foreign body. They are usually oval and are very rarely, when several are present, faceted, and occur in early and middle life.

3. Concretions composed of vegetable fibres or of hairs that have been swallowed are light, porous, usually of irregular shape, and frequently show upon section open spaces or cavities in their substance. They are sometimes found in the cæcum and may attain the size of an orange. They are sometimes made up of the insufficiently ground husks of oats or the capsules of small fruits. They occur more commonly in early life and in females.

4. Certain drugs and similar substances, as chalk, magnesia, bismuth, and shellac, when taken in undue quantities, form intestinal concretions, which appear in the stools and reveal their true nature only upon chemical examination.

Intestinal concretions when of small size occasion no characteristic symptoms. When of larger size they may be arrested at a point of stenosis of the bowel, or then the occurrence of irritation and inflammatory swelling, and they may then give rise to the symptoms of intestinal obstruction. Large concretions are usually arrested in the rectum, in the colon, or in the [illegible] of the rectum, less frequently above the ileo-cecal valve. Obstruction in the upper part of the small intestine may be caused by concretions formed in the stomach or of gall-stones.

Intestinal Parasites.—The Ascaris lumbricoides—*round worm*—and its ova are frequently found in the stools of children and young adults. Oxyuris vermicularis—*thread-worm, pin-worm*—a very common parasite, infests the rectum; and the intestinal cestodes—*tape-worms*—of which the common forms are the *Taenia saginata* or *mediocanellata*, the *Taenia solium*, and the *Bothriocephalus latus*, show themselves in the stools in the form of segments or proglottides, and their ova are usually present in great numbers (see p. [illegible]).

Sloughs.—The invaginated portion of the bowel in intussusception may slough off en masse and be discharged from the bowel. Polypi of the intestine or rectum may also become detached by sloughing and be discharged with the feces. Masses of necrotic tissue may become separated from malignant or other ulcerating growths in the intestine and be discharged with the feces. They are to be distinguished from fragments of undigested meat. The intestinal sloughs in enteric fever may sometimes be recognized in the stools and are often mistaken for milk curds.

Foreign Bodies.—The most diverse articles may be found in the stools, having been swallowed by accident or design. Small articles of all kinds may be swallowed by children, lunatics, and dements; bird-seed and the like by hysterical persons; coins, rings, and gems by professional thieves; nails, glass, fragments of china, etc., by fakirs, and such articles as artificial teeth, or even a clinical thermometer by unconscious persons, and all of these things have been voided with the stools.

XI.

THE SKIN: PHYSIOLOGICAL AND PATHOLOGICAL CHANGES AND THEIR SIGNIFICANCE: ŒDEMA: SUPERFICIAL VASCULAR CHANGES: NAILS: HAIR.

THE SKIN.

Changes in the skin not only occur as manifestations of cutaneous affections but they also constitute important diagnostic signs of diseases of the internal organs. The methods of examination are inspection and palpation. The clothing should be so arranged as to facilitate the necessary investigation.

The condition of the skin varies within physiological limits at different periods of life and in the sexes. In infancy and childhood the skin is distensible, elastic, full, of fine texture, and faint rosy color. The capillary irculation is active, pressure causes local pallor which quickly disappears. n middle life the skin is finer, softer, and shows more physiological turgescence in women than in men. With advancing age the skin loses its lasticity. Partly for this reason, partly on account of the diminished mount of subcutaneous fat, and partly because of the larger development of connective tissue, wrinkles develop. The skin in elderly persons is paler and more abundantly pigmented than in the young. The skin of very fat persons frequently has a disagreeable unctuous feel; it may be firm and tense or loose and flabby. The skin is sometimes flabby and relaxed in fat babies who are not properly fed. In the cachexias of infancy, such as that of congenital syphilis or marasmus, the skin is muddy, loose, inelastic, and sometimes wrinkled like that of old men.

Color.—The normal tint of the skin, the so-called flesh color, depends upon the blood showing through the upper layers of the integument and the epidermis. The changes in color are quantitative and qualitative, physiological and pathological. Quantitative changes consist in varying degrees of color, from blushing to blanching. They are best observed upon he face. On the other hand qualitative changes in the color of the skin re studied best upon other parts of the body where the flesh color is paler nd less variable. The mucous membrane of the conjunctiva, lips, and mouth must always be examined.

Variations in the flesh color depend upon the amount of blood in the utaneous vessels, the amount of the blood-coloring matter, that is, the ercentage of hæmoglobin, and the thickness of the tissues covering the essels. It is obvious that since any of these factors may vary in degree ne quantitative changes in the color of the skin do not always have the ame diagnostic significance.

Pallor.—The skin may be pale by reason of general or local deficiency f blood, that is to say, in consequence of anæmia or of contraction of the apillaries. The various forms of anæmia have in common a diminution the coloring matter of the blood—*oligochromæmia*. Pallor, even hen persistent, does not in all instances justify a diagnosis of anæmia, nce there are many habitually pale persons whose blood shows upon xamination a practically normal constitution both as regards the erythroytes and the hæmoglobin. Many such individuals present no symptoms f constitutional or local disease and regard themselves as in perfect health. he pallor in these cases is due to an abnormality of the skin, either an nusual opaqueness of the superficial layers or a deficiency in the blood upply or a combination of these two conditions. If the conjunctival ucous membrane and that of the lips and mouth present a normal ppearance, the pallor is due to the first of these anomalies. In the majory of instances, however, marked and persistent pallor is associated with ther evidences of more or less decided derangement of health. Even nder these circumstances in a certain proportion of the cases the blood ows no abnormal change. Two explanations of the pallor may be adanced: first, a reduction in the total quantity of the blood, which nevereless retains its constituent elements in normal proportion; second, that e skin, particularly of the face, as the result of abnormal conditions of e circulation receives a diminished amount of blood. Since we have no clinical method of determining the total volume of blood in the body, the first of these explanations is purely theoretical and without practical application. The second explanation finds support in the constant presence of other symptoms indicative of circulatory derangements, among which are a small and feeble pulse, general asthenia, over-filling of the superficial veins, slight cyanosis, faintness, and dizziness. The part played by enfeeblement of the heart's action on the one hand and by vasomotor derangements on the other cannot in all cases be satisfactorily determined. Lowered blood-pressure does not necessarily induce pallor, since in this condition the lumen of the peripheral vessels is widened and their contents increased; but diminished blood-pressure gives rise to pallor when the chief factor in its production is cardiac weakness and the vasomotor tonus is maintained. Increased vasomotor tonus may be the cause of pallor of high intensity. Among the more important diseases in which pallor occurs as the result of a diminution in the blood supply to the vessels of the face, without marked changes in the composition of the blood, are gastro-intestinal affections, both acute and chronic, diseases of the heart, pulmonary consumption and other chronic infections—conditions ultimately leading to anæmia which in many cases is profound. To this group the transient pallor of intense emotion, nausea, vertigo, syncope and collapse, in which vasomotor derangements and cardiac failure are associated in the production of lowered blood-pressure, bears a close etiological relation. Indoor occupations, dependence upon artificial light, mining and the like cause permanent pallor.

Clinically the following points are important: (a) Transient pallor is caused by cardiac failure, as in nausea, rigors, syncope, and shock, or by vasomotor spasm, as in the intense emotions of fright, fear, anger, in pain, epilepsy, and other paroxysmal neuroses. Transient pallor is frequently but not always followed by more or less intense flushing.

(b) Sudden and more persistent pallor accompanies hemorrhage, acute poisoning, and overwhelming infection—the malignant forms. Associated with other symptoms of collapse it is a striking and suggestive sign of internal hemorrhage, such as may occur in a large pulmonary cavity; as the result of the rupture of an aortic aneurism into the pericardial, pleural, or peritoneal sac; in consequence of a perforating lesion in peptic ulcer or enteric fever; in rupture of the sac in ectopic gestation, or in concealed uterine hemorrhage before or after parturition. Small hemorrhages do not necessarily cause pallor except when frequently repeated or persistent.

(c) Gradually developing pallor is a symptom of almost all serious acute and chronic diseases. In the acute infections it usually passes off with convalescence; in the chronic diseases its intensity is very often a measure of the gravity of the case. It is sometimes seen in altered conditions of living, as in the case of young immigrant girls who during the process of acclimatization not rarely permanently lose their color without changes in the blood or other signs of ill health. The pallor in persistent slight hemorrhage, such as occurs in neglected hemorrhoids, is very often intense, as is the pallor of chlorosis, pernicious anæmia, and the secondary anæmias which occur in chronic poisoning, chronic infections, chronic suppurative processes, nephritis, and valvular and mural disease of the heart.

Redness. — The change in the color of the face is quantitative. is due to two causes: first, thinness and transparency of the super- al layers of the integument; second, increased fulness of the capillaries *hyperæmia.* An abnormally high hæmoglobin percentage cannot of elf be regarded as a cause of the increased redness of the complexion. hether or not a true plethora occurs is undecided. Physiologically we d the redness of the skin of the face greater in persons who live in the en air and are especially exposed to sunlight and the wind, which increase e cutaneous circulation. An abnormally transparent skin is the evident use of the blooming redness of the cheeks occasionally seen in chlorotic le— *chlorosis florida.* Very characteristic in these cases is the contrast tween the color of the skin and the blue-white conjunctivæ. Among the ysiological causes of intensification of the color of the skin are powerful uscular effort and the action of external heat, as in hot baths, friction of e surface, exposure to fire or heat, radiation from other sources, sunburn d the like. Extreme cold also produces cutaneous hyperæmia of the face. bitual exposure to heat or cold, especially when associated with moist- e, causes the chronic purplish hyperæmia of the hands frequently seen washerwomen and bartenders who are otherwise in good health.

Transient reddening of the skin dependent upon vasomotor influences curs under certain psychic influences, especially embarrassment and me. The reddening of the skin in such cases is not restricted to the face t may spread over the throat and even the upper part of the chest. these latter situations it may be irregularly distributed in such a way to give rise to errors in diagnosis as regards actual disease of the skin, as thema, especially in sensitive persons, and particularly in women when s necessary to remove the clothing from the upper part of the body for purposes of examination. One-sided flushing of the face occurs in tain forms of migraine and in affections of the cervical sympathetic.

In addition to the foregoing facts the flushing incident to pyrexia, tain infections, and the action of drugs deserves attention.

FEVER.—The flushing of the skin in acute febrile conditions is very racteristic. It is often attended with slight turgescence and sometimes h a tendency to sweat. The flush of fever is usually widely distrib- d over the surface. It is more marked in young persons of fair com- xion than in older persons and in brunettes. It has a tendency to alize itself in the cheeks where it is sometimes circumscribed or unilateral, n croupous pneumonia. Circumscribed flushing of the cheeks in persons erwise pallid is a very striking phenomenon in the hectic fever of ad- ced phthisis. In children the fever flush is sometimes so intense as to gest the existence of erythema or scarlatina. In rare instances pyrexial hing occurs during the first week of enteric fever, especially in young sons of fair skin, and may be so marked as to give rise for a time to ertainty in diagnosis.

Tache cérébrale is a cutaneous vasomotor phenomenon which occurs ecially in young persons in acute febrile affections, as cerebrospinal ningitis, enteric fever, and influenza, in certain functional nervous ections, as hysteria, neurasthenia, and sometimes in organic diseases of brain and spinal cord. It is called forth by slight irritation of the skin, such as is produced by tapping with the finger-tip or drawing the finger or a pencil smartly over the surface. A white spot or line appears and is shortly followed by a bright red discoloration which persists for several minutes.

Dermatographism.—This condition, closely allied to the above, is not uncommon in neurotic persons, particularly in those who suffer from urticaria. Wheals may be produced by drawing the finger or a pencil somewhat firmly over the surface. Letters and other symbols may be brought out in a conspicuous manner and often last for several hours. The itching characteristic of urticaria does not occur.

DRUGS.—The reddening of the face caused by alcohol is of diagnostic importance. The expression "flushed with wine" is significant. The slightly turgid, purplish-red face of chronic alcoholism, with its distended venules, is unfortunately too familiar. The flush produced by the nitrites and especially by the inhalation of amyl nitrite resembles the blushing due to psychic causes. Flushing of the face follows the administration of certain poisons, as belladonna, opium, and hyoscyamus.

Cyanosis.—This term is used to designate the dusky blue or purplish color of the skin dependent upon the circulation in the capillaries of blood abnormally rich in carbon dioxide and poor in oxygen. Cyanosis may be general or local.

GENERAL CYANOSIS is dependent upon two factors, first, deficient oxygenation of the blood in the lungs, as the result of which the arterial blood reaches the capillaries containing less oxygen and darker in color than normal; second, stasis in the venous radicals, resulting in an accumulation of venous blood in the capillaries of the skin, which by the retardation in its flow becomes richer in carbon dioxide and darker in color. . Since the conditions are universal it may be assumed that the bluish discoloration exists not only in the skin but in all the tissues of the body. Only in its intense forms does cyanosis show itself in all parts of the surface. When slight it appears in certain parts only and here it is in all instances more intense than elsewhere. These regions are the face and especially the cheeks, the tip of the nose, the ears, lips and mucous surface of the mouth, which have an especially abundant capillary circulation and translucent integument. Other points in which cyanosis is especially manifest are the hands and feet, particularly the terminal phalanges and the nails, in which blood stasis is favored by their remoteness from the heart.

The primary derangement may be respiratory or circulatory. The interdependence of the respiration and circulation is such, however, that when cyanosis is marked there is general derangement of both in varying proportion.

Respiratory.—All conditions which interfere with the respiratory function and thus reduce the aëration of the blood may give rise to cyanosis. They are comprised in four groups:

(a) All affections which interfere with the access of air to the vesicular structure of the lungs, such as retropharyngeal abscess, stenosis of the larynx caused by pseudomembranous exudate, as in diphtheria, œdema of the glottis, pseudocroup, laryngismus stridulus, pertussis, paralysis of the abductor muscles, tumors of the larynx, foreign bodies in the pharynx, larynx, trachea, or bronchi, all forms of stenosis of the trachea, including thyroid enlargement and other deep-seated tumors of the neck, as well as mediastinal and other intrathoracic tumors, strangulation, bronchitis, and bronchial asthma.

(b) Affections which interfere with the action of the respiratory muscles, including paralysis and atrophy such as occur in bulbar paralysis and peripheral neuritis; spasmodic contraction of these muscles, as that of tetanus or epilepsy; painful affections, such as myalgia, pleurisy, and peritonitis, in which the respiratory movements are instinctively restrained; finally, the action of drugs, such as opium and its preparations, which depress the respiratory centres.

(c) Affections which diminish the respiratory surface. This group includes all forms of consolidation of the lung, croupous pneumonia, bronchopneumonia, including tuberculous infiltration and acute miliary tuberculosis, atelectasis, pressure atelectasis from pleural and pericardial effusion and pneumothorax. In emphysema the respiratory surface is not only greatly restricted but its functional integrity is also impaired.

(d) Conditions in which respiratory movements are restricted and the respiratory surface is circumscribed by subdiaphragmatic pressure, as in hydramnion, enormous ascites, enlargement of the liver or spleen, or massive abdominal or pelvic tumors.

Under all these circumstances the aëration of the blood in the lungs is diminished and venous stasis is favored by the reduction in the normal aspiratory function of the lungs which constitutes an important factor in the circulation. The absence of cyanosis, often observed in advanced phthisis with extensive destruction of the lungs and very limited respiratory movement, is probably due to the great wasting of the body and corresponding reduction in the mass of the blood, to the aëration of which the remaining limited vesicular structure is still adequate. Cyanosis is marked in proportion as the interference with respiration is rapid and urgent. In chronic cases the interference may reach a high grade without causing cyanosis during repose, though this symptom may appear upon slight exertion.

Circulatory.—Primary derangements of circulation which cause cyanosis may be referred to the following groups:

(a) Affections of the heart and arteries, including valvular disease with impaired or ruptured compensation, myocarditis, acute dilatation of the heart, the cardiovascular changes which occur in chronic nephritis, other forms of arteriosclerosis, and pericarditis.

In persistent foramen ovale and other forms of cardiac malformation, such as stenosis of the pulmonary artery, there is very often marked and continuous cyanosis. To this condition of congenital cyanosis the term *morbus cæruleus* has been given. In acquired conditions permitting an admixture of venous blood with arterial within the vessels, as in the very rare cases of aneurism of the aorta communicating with the vena cava, cyanosis is a suggestive symptom.

(b) Conditions affecting the pulmonary circulation. In disease of the mitral valve, both stenosis and insufficiency, even when compensation is good there may very often be seen, especially upon exertion, a slight degree of cyanosis. This is a manifestation of the changes caused by the habitual increase of tension in the pulmonary circuit and the bronchial catarrh which to some degree is almost constantly present. Though having its primary cause in the circulatory apparatus this form of cyanosis must be looked upon as respiratory.

Pressure upon the pulmonary artery or veins by massive pericardial effusion, mediastinal tumor or aneurism is a very common cause of cyanosis. The circulation of the pulmonary capillaries is obstructed in many of the conditions involving the respiratory apparatus which give rise to cyanosis.

Blueness of the general surface, very often intense, is produced by overdoses of certain of the coal-tar derivatives, especially acetanilid, by nitrobenzole, and by poisoning with illuminating gas.

Local cyanosis results from venous stasis, from compression of the part or from venous thrombosis. Cyanosis of the head and neck or an upper extremity may result from the pressure of a tumor or aneurism upon the jugular, subclavian, innominate, or descending cava, the distribution of the cyanosis corresponding with the point of pressure. Similar cyanosis of one or both lower extremities may result from pressure involving iliac veins or the ascending vena cava or from venous thrombosis. Local venous thrombosis giving rise to cyanosis of an arm is sometimes seen in cancer of the breast with secondary implication of the axillary glands.

Cyanosis, often of high grade, results from vasomotor derangements. To this cause must be referred the cyanotic discoloration of the extremities and ears which follows exposure to intense cold, the cyanosis of paralyzed members, and the bluish discoloration of the hands which occurs in hysterical and neurasthenic persons. In the latter group of cases the local cyanosis is sometimes associated with œdema—the *blue œdema* of French authors.

Local cyanosis is seen in intense inflammation involving the skin.

The conditions which give rise to cyanosis, namely, retarded circulation and reduced oxygenation, interfere with the local production of animal heat. In cyanosis the skin and extremities show reduction of surface temperature.

Jaundice—Icterus.

These terms are used to designate the peculiar pathological yellow discoloration of the skin, mucous membranes, and fluids of the body caused by the circulation in the blood of bile pigment. The change is qualitative. There are two forms, obstructive and toxæmic.

Obstructive Jaundice.—This is the more common form. The discharge of bile into the intestine is interfered with wholly or in part by stenosis or closure of the bile passages. As a result there is resorption of the bile, the pigments of which discolor the tissues in shades varying from light yellow to a dark brownish-yellow or olive-green. The darker shades of jaundice result either from change of the original bile pigments to darker pigmentary bodies or from their excessive accumulation in the skin. The more intense and darker forms of jaundice occur in protracted cases. In permanent obstruction the color may be greenish-black or bronze—the so-called *black jaundice*.

Among the more important causes of obstructive jaundice are catarrhal inflammation of the mucous membrane of the duodenum or the common uct; gall-stones and parasites, as the round worm, in the ducts; stricture or bliteration of the duct; tumors developing in the duct or exerting pressure pon its orifice; external pressure upon the duct by tumors of the liver, tomach, pancreas, kidney, or omentum, or by enlarged glands in the porta, r in rare instances by aneurism or fecal accumulation.

The yellow discoloration is observed first and, when slight, only in he conjunctivæ and the mucous membrane of the mouth. Its presence ay be detected by pressure upon the mucous membrane of the everted p with a glass slide, thus expressing the blood and permitting the yellow ain of the tissues to become apparent. It is sometimes distinct at certain ale areas of the hard palate. The slighter grades of icterus cannot be ecognized in artificial light. Superficial resemblances to jaundice are seen the dirty yellow or muddy discoloration of the malarial and malignant achexias. In these conditions the absence of yellowness in the conjuncval and oral mucous membranes is conclusive. The collections of yellow bconjunctival fat occasionally seen in elderly persons are only in the ost remote way suggestive of jaundice. The yellow discoloration which ccurs in picric acid poisoning presents superficial resemblances to jaunice. The absence of bile pigment in the urine is important.

Pruritus is a troublesome symptom. It is usually more marked in the hronic cases. Lesions of the skin, the result of scratching, are not uncomnon. Sweating is common and may be localized. Urticaria, furuncles, ichen, xanthelasma, and other diseases of the skin occur. In some of the hronic cases circumscribed patches of dilatation of the capillary vessels nd minute arteries—telangiectasis—develop in the skin of the face and ody and occasionally upon the mucous membranes. In protracted and evere cases there may be hemorrhages into the skin, usually in the form f purpuric spots upon the lower extremities, but sometimes as large cchymoses, and in some instances spontaneous bleeding from the mucous nembranes occurs. The blood in chronic jaundice coagulates very slowly—en to twelve minutes, instead of about four in the case of normal blood—nd troublesome and even fatal hemorrhage, usually in the form of unconrollable capillary oozing, may follow operation or injury. The sweat is ile-stained and discolors the clothing. The urine contains bile pigment nd may show the color reaction to Gmelin's test before the yellow tint ppears in the mucous membranes or the skin. The color varies from light ellow with a greenish tinge to a deeply opaque black-green. In intense or ng-standing jaundice the urine commonly contains albumin and tube asts which are bile-stained. Upon agitation the dark urine of jaundice is rothy and is often popularly compared to porter. The sputa are not often ile-stained, except when pneumonia is present. On the other hand the aliva very rarely shows the yellow discoloration, which is likewise absent the tears and milk.

As no bile is discharged into the intestine the stools are of a pale drab r clay color. They are usually pasty and very fetid. The absence of bile the fæces is of importance in the differential diagnosis between obstrucive and toxæmic jaundice. Commonly there is constipation; occasionally iarrhœa. The pulse, in obstructive jaundice, [illegible] recent cases is sually slow and may fall to 30 or lower. [illegible] ation is also diminished, in some instances to 10 or 8 per minute. The temperature may be subnormal. These symptoms are attributed to the action of the biliary salts, which undergo resorption together with the bile pigment. They are not constant and when present not necessarily unfavorable.

The patient is usually depressed and irritable. In severe cases melancholia may develop. The liability to the occurrence of the condition called cholæmia constitutes a serious danger in persistent jaundice. The patient falls into the so-called typhoid state, with fever, rapid pulse, dry tongue, and muttering delirium. Convulsions and coma develop and rapidly prove fatal. This group of symptoms resembles uræmia. They have been attributed to poisoning by cholesterin—cholesteræmia. The toxic substances have not been determined.

Toxæmic Jaundice.—The jaundice is associated with the presence of various poisons in the blood which act directly upon the red blood-corpuscles and in some cases upon the liver-cells. Among these poisons are (a) snake venom, phosphorus, arsenic, chloral hydrate, chloroform, and ether; (b) toxins elaborated within the organism in the course of the specific infectious diseases, as yellow fever, relapsing fever, malaria, pneumonia, enteric fever, typhus, and scarlatina; (c) the toxins of septic conditions, pyæmia, malignant endocarditis, acute yellow atrophy of the liver, Weil's disease, and epidemic jaundice. The symptoms are generally less intense than in obstructive jaundice. The discoloration of the skin is usually slight; exceptionally, as in the case of acute yellow atrophy and malignant jaundice, it is intense. The stools are colored with bile, sometimes deeply. The urine may be dark from increase in the normal urinary pigments but gives little or no reaction for bile pigment. Toxic jaundice of slight degree frequently appears during the course of febrile affections and under other circumstances and may be without unfavorable prognostic significance. On the other hand in many cases the conditions in which this form of jaundice occurs are attended with profound constitutional disturbance, manifest in intense fever, delirium, suppression of urine, hemorrhages into the skin and from mucous surfaces, convulsions and coma, and very often terminate in death.

The jaundice due to obstructive changes in the bile passages was formerly spoken of as *hepatogenous;* toxæmic jaundice as *hæmatogenous.* Concerning the mode of origin of toxæmic jaundice there is much diversity of opinion and the cases differ among themselves. In groups of cases there is probable resorption of bile pigments from the liver as the result of pathological processes involving the finer ducts or the liver parenchyma itself. Some pathologists attribute the icterus, so common in pneumonia, to a catarrh of the finer bile passages dependent upon venous stasis, while others attribute it in part at least to the interference with the respiratory movement of the diaphragm caused by the consolidation of the lung, and resulting in an accumulation in the smaller ducts of bile which undergoes resorption. The rapid course and profound disorganization of the liver in acute atrophy and in phosphorus poisoning suggest the possibility that other forms of grave toxæmic jaundice may be due to as yet unknown parenchymatous changes in the liver. On the other hand most of the poisons which cause icterus exert a destructive influence upon the erythrocytes. It has been shown experimentally, however, that the yellow pigment in poisoning by certain substances, as toluylendiamine, is not formed in the blood but in the liver, the hæmoglobin being transformed into biliary pigment in that organ. As a result of this transformation the bile pigments accumulate in the liver in such quantity that they cannot be wholly excreted, a certain portion undergoing resorption. In consequence of these facts the term *hæmatohepatogenous* has been suggested for this form of jaundice. In the present state of knowledge the etiological designation toxæmic jaundice is to be preferred. The term toxæmic-obstructive jaundice has been suggested by Hunter.

Normal and Abnormal Pigmentation—Melanoderma.—The physiological pigmentation of the skin shows wide variations not only in different races but in different individuals of the same race. Among the fair-skinned a blonde and à brunette type are recognized. The latter is characterized by a darker color of the hair, skin, and iris. Normally the skin is more deeply pigmented in the exposed portions of the body to which the light and air have free access than elsewhere; upon extensor than upon flexor surfaces in the region of the joints; and about the nipples, linea alba, and genital organs. During pregnancy the pigmentation in these latter situations is greatly increased, especially in brunettes, and upon the face and in other portions of the body there are occasionally seen irregular, abnormally pigmented areas known as *chloasma gravidarum—masque des femmes enceintes.* Patchy pigmentation of the skin is a common symptom of uterine disease. In sedentary persons of constipated habit irregular patchy pigmentation of the skin is common, especially about the face and eyes.

FRECKLES OR EPHELIDES are another physiological pigmentation of the skin without diagnostic importance. The pigmentation appears in circumscribed spots varying from one to several millimetres in diameter, chiefly upon the face, but also in other parts of the body, especially the backs of the hands and arms. They are more common in fair than dark persons and are almost always present in individuals with red hair. The spots are more abundant and the pigmentation deeper in summer than in winter, when they sometimes wholly disappear.

THE PIGMENTATION following measles and showing the characteristic form and arrangement of the eruption is not wholly without interest to the clinician, and the localized pigmentation which follows the application of sinapisms and blisters deserves passing mention.

THE VAGABOND'S SKIN is a term applied to the diffuse pigmentation resulting from lousiness and dirt and the scratching caused by these conditions. The pigmentation sometimes reaches a very high grade. It may be arranged in a very characteristic manner in stripes corresponding to the lines of scratching. This condition has been confounded with the pigmentation of Addison's disease.

MELANOSARCOMA, especially when generalized, very often produces a deep and widespread cutaneous pigmentation. Under these circumstances in exceptional cases the urine also contains abnormal pigment.

In ADVANCED PULMONARY TUBERCULOSIS a striking brownish discoloration of the face or the whole body is sometimes observed.

In ABDOMINAL NEW GROWTHS, especially cancer or lymphoma, diffuse cutaneous pigmentation occasionally occurs. It is not uncommon in tuberculosis of the peritoneum.

In HÆMACHROMATOSIS, such as occurs in hypertrophic cirrhosis, diabetes, and other conditions, pigmentation of the skin may be present.

EXOPHTHALMIC GOITRE may be associated with abnormal pigmentation.

GASTRIC.—In rare instances diffuse pigmentation attends gastric ulcer and dilatation.

In SCLERODERMA cutaneous pigmentation may be general and of high grade.

CARDIAC.—In chronic disease of the heart and arteriosclerosis diffuse pigmentation may occur.

ADDISON'S DISEASE.—The bronze discoloration of this affection is clinically the most important form of abnormal pigmentation of the skin. It usually shows itself first upon exposed surfaces, as the hands and face, and is more intense in those regions in which the skin is normally more deeply colored than elsewhere. It begins as a faint smoke-gray discoloration and progressively deepens to an intense bronze or mulatto hue. In the diffuse smoky coloration isolated intense dark brown points may be distinguished. The grayish pigment patches seen upon the mucous membrane of the mouth are characteristic of Addison's disease. The palms and soles as well as the nails commonly remain pigment free. The discoloration of Addison's disease may suggest intense jaundice, but the general condition, the yellow staining of the conjunctivæ and the mucous membrane of the mouth, and the presence of bile pigment in the urine are of positive diagnostic importance.

HEPATIC DISEASE.—The peculiar discoloration of the skin occasionally seen in cirrhosis and other diseases of the liver demands consideration. The color is a dirty brownish-gray. It is to be differentiated from icterus by the color itself, the absence of staining of the mucous membranes, and the condition of the urine. This pigmentation is of especial interest in connection with the bronzing of the skin that occurs in certain cases of diabetes—*diabète bronzé*—developing late in hæmachromatosis and associated with pigmentary cirrhosis of the liver and pancreas. The color suggests Addison's disease, but the presence of grape sugar, the physical signs of hepatic cirrhosis without jaundice, and the absence of the characteristic symptoms of Addison's disease are of diagnostic importance.

ARSENOMELANOSIS.—The pigmentation of the skin produced by the prolonged administration of arsenic in full doses sometimes presents a very close resemblance to Addison's disease. In a majority of the cases it entirely disappears when the drug is withheld; exceptionally it is persistent. It is important to know that in some instances the pigmentation of the skin has followed the use of arsenic in moderate doses.

ARGYRIA.—The prolonged administration of silver nitrate results in the deposition of particles of metallic silver or its albuminate in the internal organs and in the skin. The resulting discoloration is a peculiar bluish-gray which is more intense upon the face and hands and is not changed by pressure. The discoloration may be observed in the mucous membrane of the mouth. It is persistent and not amenable to treatment.

ALBINISM is a term used to designate developmental deficiency of pigment. In albinos the skin, hair, and eyes are conspicuous by the absence of pigment. The affection may be partial or universal. It is frequently associated with other developmental defects, especially coloboma. Nystagmus is common.

VITILIGO is a condition of the skin characterized by deficiency of pigment. The patches are usually circumscribed, very often distinctly marginate, and sometimes surrounded by a zone of pigmentation slightly deeper than normal. It may occur on any part of the body, but is common on the back of the neck and shoulders, the abdomen, and scrotum. There are no subjective symptoms. It occurs in adolescents and young adults.

LEUCODERMA or pigment atrophy, usually circumscribed or irregularly distributed, is encountered in exophthalmic goitre, myxœdema, scleroderma, and other constitutional disturbances.

Moisture.—There are wide variations in the activity of the sweat-glands within physiological limits. Perspiration is excited by those causes which determine an active blood supply to the skin. It is therefore more abundant in warm weather, after exercise, hot baths, and hot drinks. An outburst of sweating may occur in connection with sudden intense emotion. A pathological increase of perspiration is termed hyperidrosis; its absence anidrosis. These terms are commonly used to designate conditions in which the increase or absence are persistent or habitual.

Hyperidrosis.—Free perspiration attends certain febrile diseases, especially rheumatic fever, some cases of enteric fever, acute polyneuritis, miliary fever, and septic conditions. A critical decline of fever, whether spontaneous or the result of the administration of antipyretics, is almost always attended by more or less abundant sweating. Perspiration is one of the processes by which, both physiologically and pathologically, the temperature of the body is lowered. Profuse sweating attends the crisis in pneumonia, relapsing fever, and typhus. Sweating is often abundant toward the close of enteric fever when the temperature curve assumes a distinctly remittent or intermittent type. The fall of temperature in the ague paroxysm is almost always attended with copious sweating. That of the hectic fever of phthisis and other wasting diseases usually occurs during the night or toward morning. It is attended with abundant sweating—*night-sweats*—and is of unfavorable prognostic significance. Profuse sweating occurs in some cases of phthisis in the absence of fever. Sudden abundant sweats are accompanied by sensations of great weakness and prostration which are in part due to the relaxation of the vessels following the sudden withdrawal of fluid. Excessive sweating occurs in the convalescence from some diseases. It occurs in collapse, urgent dyspnœa, and sometimes accompanies severe paroxysms of pain. In rare instances of diabetes abundant perspirations have alternated with polyuria. Increased sweating sometimes attends the suppression of urine that occurs in certain forms of nephritis. Under these circumstances crystals of urea may accumulate upon the skin and especially upon the face.

Localized sweating is not uncommon in pathological conditions. Hyperidrosis of the hands and feet occasionally occurs in neurotic individuals and sometimes in persons otherwise healthy. The condition is very annoying. The sweat is usually copious and foul-smelling. Axillary sweating is an annoying constitutional peculiarity. Sweating of the head, especially during sleep, is an important symptom in rickets. Unilateral sweating of the head or face occurs in certain nervous diseases, as migraine and neuralgia, and may result from pressure upon the sympathetic by a thoracic aneurism or mediastinal tumor. Localized sweating depends upon vasomotor derangements. Diaphoresis follows the administration of certain drugs, especially ammonium acetate, pilocarpine, and many of the coal-tar derivatives.

Anidrosis.—Abnormal dryness of the skin occurs under conditions in which an excess of fluid is withdrawn from the body by way of its internal surfaces, or very little water reaches the blood by way of the gastro-intestinal tract—for example, profuse diarrhœa, continuous vomiting, diabetes mellitus and insipidus, chronic nephritis with polyuria, and the deprivation of fluid. The dry skin of myxœdema and general anasarca is largely attributable to the interference with the cutaneous circulation resulting from tension.

Modifications in the Perspiration.—Perspiration when abundant usually has a peculiar acid odor. That in rheumatic fever is acid and ill-smelling; the sweat of the hands, feet, and axilla is almost always foul; that in certain forms of nephritis has a urinous odor. The sweat may be discolored—*chromidrosis*—yellow from biliary pigments in jaundice; blue from the action of the Bacillus pyocyaneus. There are instances recorded of the sweating of a blood-stained fluid or blood in hysterical females—*hæmatidrosis*—and there exists a term—*menidrosis*—to describe vicarious menstruation by way of the skin. These conditions are of no importance in diagnosis. Various colored perspiration-stains upon the linen are not to be mistaken for instances of chromidrosis. It may prevent error to call attention to the fact that some of the aniline dyes undergo more or less marked changes in color under the action of perspiration.

Fulness of the Skin—Turgor.—The normal appearance of fulness of the skin is due to the blood and lymph in its vascular and lymph spaces. It varies in different individuals and in different parts of the body, and is more pronounced in females. In connection with an abundant panniculus it has much to do in causing the condition described by the French as *embonpoint*. Increased fulness of the skin is seen in fever and other conditions attended by active cutaneous circulation; decreased fulness in all conditions in which the cutaneous circulation is diminished without stasis, particularly in emaciation, the cachexias, and under the deprivation of fluid. Increased fulness is manifested by rounding of the contours, especially those of the face, and usually by a deeper color of the skin, while diminished fulness produces accentuation of the angles and is usually associated with more or less pallor. In the former condition the skin is smooth, soft, and elastic; when pinched up into folds it rapidly reassumes its normal surface. In the latter such folds only slowly disappear. Normal fulness or turgor is to be distinguished from œdema and anasarca by the pathological amount of fluid in the skin in the latter, the loss of the normal cutaneous elasticity, and by the persistence of the pitting made by pressure of the finger. The difference between "looking well" and "looking bad" ften depends upon slight transient variations in the normal fulness face, which is diminished in conditions of exhaustion and depression creased after repose and in pleasur- xcitement. The turgor of the skin ally increased in exophthalmic goitre. ly diminished fulness of the skin such urs in ileus, peritonitis, cholera, and cases of shock, and which precedes , gives rise to the *facies Hippocratica* n these conditions.

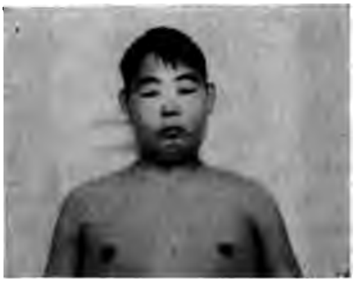
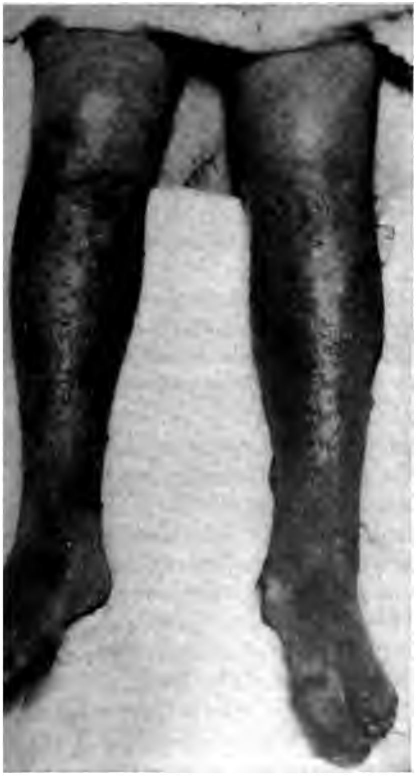


Fig. 201a.—Œdema in chronic parenchymatous nephritis.—Jefferson Hospital.


. 201b.—Œdema of the legs with cutaneous a case of subacute parenchymatous nephritis.— Hospital.

Edema—Dropsy.—An abnormal accu- ion of serous fluid collects in the lymph s of the skin and the subcutaneous ctive tissue as the result of a disturb- f the balance between the fluid which transudes from the capillaries and hich is taken up by the lymphatics. This disturbance of balance may be due to (a) venous obstruction, (b) altered condition of the blood —*hydræmia*, (c) inflammation, and (d) œdema of nervous origin. The diagnostic significance of œdema depends upon its location, extent, and mode of development and its causal relations to local or constitutional diseases. General œdema is described under the term *anasarca.* The skin is distended and the normal surface landmarks obliterated. When œdema is marked the surface is tense, pallid, and glistening. In rapidly developing recent œdema it has a translucent appearance. In some surfaces, especially upon the abdomen and thighs, transparent parallel stripes appear, similar to those seen on the abdomen in pregnancy. These are due to the collection of the fluid in the lines of separation of the distended tissues or in the enlarged lymphatic spaces. They usually disappear upon the subsidence of the œdema without leaving traces. Occa- y they leave permanent irregular linear scars. In œdema of grade, especially under the influence of irritation or slight trau- n of the skin, blebs may form upon the epidermis which rupture and are followed by the discharge of serous fluid. Occasionally, especially upon the legs and ankles, transudation of the fluid takes place through minute openings of the skin without bleb formation. Under these circumstances infection may occur, giving rise to erysipelatous or other inflammation. The pale color of the skin in œdema is caused by diminished capillary circulation from compression. The œdematous parts are sometimes cyanosed and in inflammatory œdema the skin is reddened.

The normal elasticity of the skin is impaired by tension and the inhibition of fluid. Pressure upon the œdematous part gives rise to pitting which only slowly disappears. Where the skin is normally distensible and elastic the pitting is more transient. This is especially the case in children. In moderate œdema of long standing a gradual increase in the subcutaneous connective tissue develops and pitting is less marked and more transient.

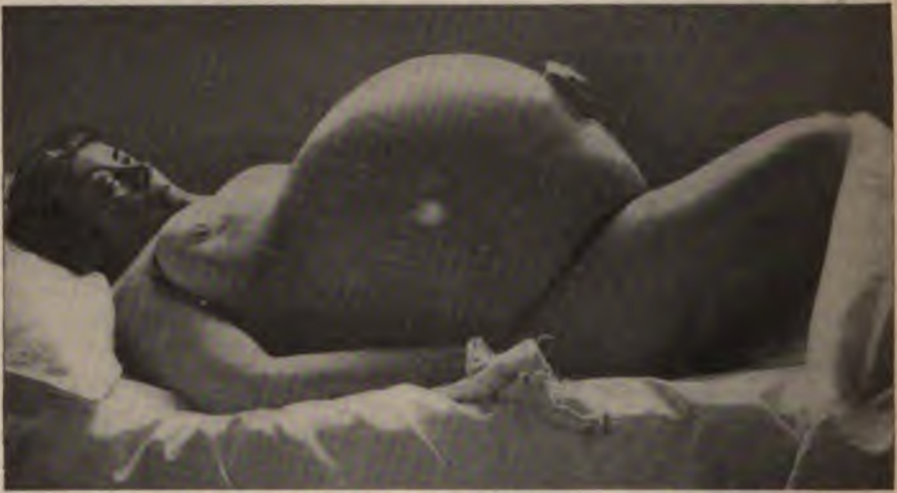


FIG. 202.—Œdema of abdominal wall and thighs in ascites due to atrophic cirrhosis of the liver.—Jefferson Hospital.

(a) **Venous Obstruction.**—Factors in the production of this form of œdema are diminished general muscular activity, impaired pumping action of the organs of respiration, diminution of the aspiratory force of the heart in diastole and positive pressure on the veins. Coincidently the return flow of the lymph which is brought about by the same forces that maintain the venous circulation is impeded. This form of dropsy is frequently associated with effusion into the great serous sacs. The fluid which collects is clear, usually colorless, of low specific gravity, fibrin free, and contains a slightly smaller amount of proteids than the blood-serum. It is to be distinguished from an inflammatory exudate which is often turbid, sometimes bloody, of high specific gravity, and usually contains masses of fibrin. Changes in the tissues and particularly in the endothelium of the lymph-spaces also play an important part in œdema-formation—so-called "vital secretory" processes.

The collection of serous fluid in the pericardium is known as *hydroperi-*

cardium, in the pleural cavity as *hydrothorax*, in the peritoneal cavity as *hydroperitoneum* or *ascites*, in the brain as *hydrocephalus*, in the joints as *hydrarthrosis*. Any of the affections of the heart and lungs which, by interfering with the return of the venous blood, cause cyanosis may also cause œdema. Cyanosis and œdema are therefore frequently associated. This form of œdema appears earliest and reaches its fullest development in those regions in which the circulation, by reason of remoteness from the heart and the influence of gravity, is less active, as in the extremities and the lumbar regions and other dependent portions in bedridden patients. The face at first is free and becomes œdematous only when the anasarca reaches a high grade. Gravity plays an important part in the localization of the œdema. Œdema of the legs and feet while the patient is in the upright position may alternate with œdema of the back and thighs when he is in the recumbent posture. The patient who is apparently free from œdema while in bed may show œdema of the feet and ankles when he first rises. In prolonged maintenance of the lateral decubitus the œdema is more marked upon the dependent side. In anasarca of high grade, partly on account of their dependent position and partly on account of the distensibility of the skin, the penis and scrotum and the labia majora become enormously swollen.

Local œdema may be due to the obstruction of a venous trunk by thrombosis or pressure. Œdema of the arm from the pressure of enlarged axillary lymphatics upon the veins, and the œdema of the leg in thrombosis of the femoral vein are familiar examples. Obstructive œdema of the lower extremities is frequently secondary to peritoneal effusion, such as results from cirrhosis or portal thrombosis or from chronic peritonitis. The accumulation of the fluid presses upon the inferior vena cava or the common iliac veins. In other cases the œdema of the lower extremities and the peritoneal effusion are due to the same cause. When, upon investigation, the signs of peritoneal effusion are found to have preceded the œdema of the limbs, the latter condition is usually secondary.

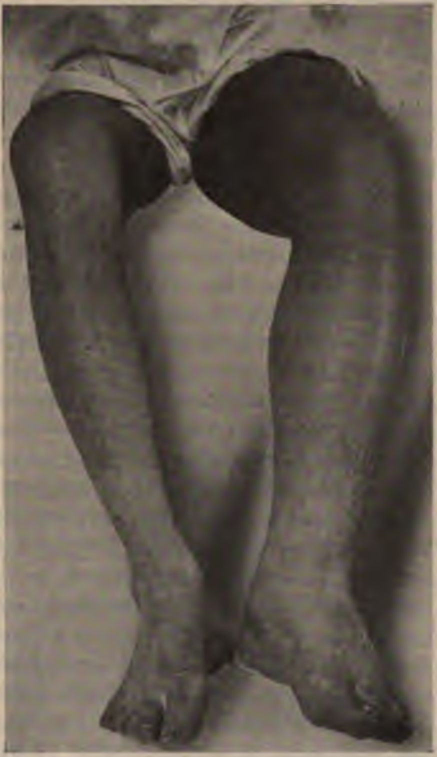


FIG. 203.—Œdema of left leg due to a thrombus in the external iliac vein.—German Hospital.

(b) **Altered Condition of the Blood—Hydræmia.**—A watery condition of the blood is a common cause of œdema and other forms of dropsy. To this condition may be referred those forms of œdema which occur in nephritis, chronic wasting diseases, the anæmias, and cachexias. Not infrequently associated cardiovascular disorders are present which interfere with the venous circulation, and in such cases the œdema from venous obstruction and the œdema of hydræmia are combined. This form of œdema differs markedly from the œdema of venous obstruction in its early localization, which is dependent much less upon remoteness from the heart and the action of gravity and much more upon the peculiarities of the lymph structures. It is characteristic of the œdema of certain forms of nephritis that it first appears in the face and especially about the eyelids. With this early œdema of the face pretibial œdema is often associated and is sometimes present in cases of nephritis, especially the chronic interstitial forms in which facial œdema is slight or absent altogether. The œdema of acute nephritis often develops rapidly and reaches a very high grade. Not infrequently it is associated with effusion into the serous sacs. In that form of nephritis characterized by contraction of the kidney œdema is very often slight in amount and a late manifestation, first showing itself when the hypertrophied heart begins to fail. In the subacute and chronic forms of parenchymatous nephritis the œdema is usually moderate, showing, however, temporary increases which accompany exacerbations of the disease. In the hydræmia resulting from large or frequently repeated hemorrhage, œdema is often pronounced. Œdema of the feet and ankles is a very unfavorable symptom in pulmonary consumption. Occurring in the absence of renal disease or especially in the absence of conditions giving rise to venous obstruction it is commonly an indication of approaching death.

(c) **Inflammatory Œdema.**—The local œdema in the region of inflammatory and suppurative processes is of diagnostic importance. It is sometimes known as collateral œdema. The color of the surface varies from a faint blush to a deep, mottled, cyanotic, purplish red. It is due to obstruction of the lymph circulation by the inflammatory exudate. In some instances it appears to be caused by an accumulation of the fluid part of the exudate in the tissues surrounding the inflammatory focus. It occurs in the region behind the ear in mastoid disease; about the angle of the jaw in mumps and parotid bubo; at the base of the thorax in empyema. It is an important sign of hepatic abscess, acute suppurative gall-bladder disease, and is sometimes seen in the right lower quadrant of the abdomen in appendicular abscess. It constitutes the so-called collar of brawn in severe anginose scarlatina.

(d) **Œdema of Nervous Origin**.—The rare cases of sudden transitory œdema of the face and neck, sometimes associated with symptoms of œdema of the respiratory or gastro-intestinal mucous membranes, must be ascribed to angioneurotic derangements. The mechanism which causes it remains unknown. The condition known as angioneurotic œdema is characterized by the sudden occurrence of local œdematous swellings of transient duration upon the face, hands, and elsewhere. Forms of localized œdema, described under the term giant urticaria, are of angioneurotic origin. The acute œdema associated with urticaria and gastro-intestinal crises which occurs in severe purpura, and the cases of œdematous swelling and tumefaction of the whole arm upon exertion, are to be referred to this group. The local œdema occurring as a symptom in peripheral multiple neuritis and the œdema of beriberi are probably of nervous origin, as is hysterical œdema.

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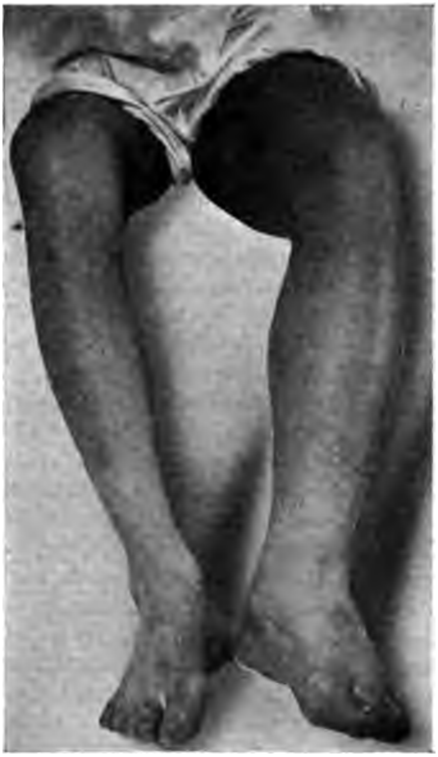

Fig. 203.—Œdema of left leg due to a thrombus in the external iliac vein.—German Hospital.

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(e) **Œdema due to Other Causes.**—Œdema neonatorum is a rare condition sometimes confused with sclerema, from which, however, it is pathologically distinct. It is encountered in feeble infants, especially those born prematurely or exposed to cold after birth. Cases of hereditary œdema have been described. The œdema is congenital and persistent; it involves one or both legs and is dense and inelastic. It shows no disposition to increase and is unattended by special inconvenience.

The œdema which occurs in trichiniasis is of diagnostic importance. It appears in the face and over the affected muscles, and undergoes remarkable fluctuations in degree during the course of the disease.

General œdema in the absence of nephritis is not infrequently observed in certain of the infectious diseases, as scarlet fever and diphtheria; it may follow the therapeutic injection of the different sera and in some instances the administration of potassium iodide. Slight œdema of the feet and ankles—a mere puffiness—is not uncommon in individuals otherwise healthy, after prolonged standing or walking or after forced marches.

Lymphœdema.—The transudation of lymph through the walls of the lymphatic vessels, or distention of the lymph spaces from mechanical obstruction, may cause great swelling, which is usually local or confined to a single limb. It results from pressure upon, or internal occlusion of, a lymph-vessel and is seen in the lymph scrotum and certain forms of elephantiasis caused by the Filaria sanguinis hominis and accompanied by chyluria. Lymphœdema involving a member—*macromelia*—sometimes occurs in lymphadenoma. This form differs from ordinary œdema by its greater firmness and brawniness—a very important point in differential diagnosis.

It is of diagnostic importance to recognize the distinction between the various forms of œdema and myxœdema—an affection of the thyroid gland characterized by swelling of the skin, eyelids, and other parts of the body, due to the deposition in the skin and subcutaneous tissues of a mucinous material. The skin is dry, rough, and swollen, but firm and inelastic, and does not pit on pressure.

Certain connective-tissue dystrophies present a superficial resemblance to localized œdema. The swellings usually involve the outer or posterior aspect of the extremities, but may appear at various parts of the trunk. They are to be differentiated from œdema by their localization, the absence of pitting upon pressure, and by other appearances of the skin characteristic of œdema.

Scleroderma, a brawny induration of the skin, in some instances suggests chronic œdema. Two forms are recognized, the circumscribed and the diffuse in which large areas are involved. The skin is brawny, hard, and inelastic. When circumscribed the patches are irregularly oval and vary in diameter; they may be as large as the hand. They are preceded by hyperæmia of the skin. The disease is more common in women than in men and frequently shows itself about the neck and breasts. The diffuse form involves the extremities and face. The skin is hard and firm with stiffness and tension. It is adherent to the underlying tissues and cannot be pinched up into folds. There is impairment of movement. Very often there are vasomotor disturbances with cyanosis. Pigment alterations are frequent—both melanoderma and leucoderma.

Sclerema neonatorum is a rare disease of the new-born in which the skin rapidly assumes the clinical appearance of scleroderma. It is usually fatal. It presents superficial points of resemblance to œdema neonatorum, from which it is to be distinguished by the complete absence of the ordinary signs of anasarca.

Scurvy sclerosis—a deep brawny infiltration of the subcutaneous tissues and muscles, with hemorrhagic discoloration of the overlying skin—frequently seen on the calves of the legs, is not to be confounded with œdema, although it is very often associated with it.

Subcutaneous Emphysema.—The presence of gas, usually air, in the meshes of the subcutaneous tissue gives rise to swelling and puffiness of the surface which may be either general or local. The appearance is not unlike that of œdema, but upon palpation a peculiar crackling is to be felt and heard, due to the displacement of bubbles of air in the tissues. The surface resistance is lower than normal and pitting from pressure does not occur. Upon percussion the sound is tympanitic. The skin is pale and has a distended appearance. In very rare cases subcutaneous emphysema is due to the presence of aërogenous bacteria—Bacillus aërogenes capsulatus—and allied organisms. This gaseous and necrotic œdema occurs in serious wound infection and may extensively involve the subcutaneous tissues of the body. The infection may proceed from the uterus, gastro-intestinal canal, or respiratory tract. Analogous to this condition is the subcutaneous emphysema of malignant œdema. It is sometimes associated with tetanus.

In the greater number of cases the air finds its way under the skin through an external wound or through the ulceration or laceration of some air-containing organ. Subcutaneous emphysema is, therefore, an accident of carcinomatous or other ulceration of the œsophagus, of diseases attended by violent paroxysmal cough by which the alveolar tissue is mechanically torn, or occasionally of the after-treatment of tracheotomy, the air being forced into the subcutaneous tissues by efforts of cough. The air usually accumulates about the root of the neck and over the manubrium. It may invade the tissues underlying the skin very extensively and sometimes involves the greater part of the body. As a rule it undergoes rapid resorption.

Cutaneous hemorrhages appear as spots or streaks of varying size, at first red, but quickly becoming darker. Small hemorrhages—*petechiæ*—frequently have their origin in the hair follicles. Larger hemorrhages—*ecchymoses*—are diffuse. Hemorrhages arranged in the skin in the form of lines and streaks are called *vibices*. The term *suggillation* is sometimes used to describe the ecchymosis following a bruise. *Hæmatoma* is a tumor containing effused blood. Cutaneous hemorrhages may occur upon any part of the body, but when due to constitutional disease they are more abundant upon the lower extremities. In consequence of transformations in the hæmoglobin the color during resorption undergoes progressive changes to blue, green, and yellow, and gradually fades. The appearance and distribution of petechiæ is characteristic of cutaneous hemorrhage. They are not usually elevated above the skin. Occasionally in purpura there are vesicular points distended with blood. In doubtful cases cutaneous hemorrhages may be distinguished from local hyperæmia or erythema by the fact that they do not disappear when the skin is made tense by traction upon it of the thumb and finger or by pressure with a glass slide. In local hyperæmia the spot of redness disappears; in hemorrhage, owing to the expression of the blood from the surrounding capillaries, it becomes more distinct. Affections characterized by the extravasation of blood into the skin are collectively described under the term *purpura*.

Cutaneous hemorrhage is in all cases of diagnostic importance. The more important conditions with which it is associated are traumatism, intense venous stasis, the severe and especially the malignant infections, sepsis of various kinds, deep jaundice, and cachectic and anæmic states. It is a characteristic phenomenon of the action of certain snake venoms and under exceptional circumstances follows the administration of copaiba, quinine, ergot, iodine, and other drugs.

Hemorrhage into the skin occasionally occurs in acute myelitis, severe neuralgia, and in tabes. In the last it is very often transient. The bleeding points or stigmata that have attracted so much attention in rare cases of hysteria are of nervous origin.

It is frequently associated with arthritis. The relationship of these conditions has been regarded without adequate reason as rheumatic.

Other changes in the skin of diagnostic importance are: striations, desquamation, furunculosis, cicatrices, and glossy skin.

STRIATIONS.—The striæ of the skin of the abdomen and those occurring in œdema and peritoneal effusion, which resemble the striations of pregnancy, have already been described. They frequently disappear after resorption of the fluid, but may persist for a long time. Similar striations may attend the rapid development and equally rapid resorption of a thick panniculus adiposus. They are encountered in cases of great abdominal distention from rapidly developing tumors or other cause.

DESQUAMATION.—Shedding of the epidermis is of diagnostic importance. A diffuse desquamation of the trunk and extremities, usually in the form of fine scales, occurs in the cachexia associated with emaciation. A similar fine-scaled desquamation follows measles. A coarser desquamation, sometimes lamellar, is almost constant after scarlet fever. A coarse desquamation follows erysipelas. The decrustation of the variolous diseases may be mentioned in this connection.

FURUNCULOSIS.—Boils or furuncles are the expression of an acute inflammation of a hair follicle and its sebaceous gland and the connective tissue immediately surrounding them. It is a local process due to an infection through the follicle by pus-producing organisms, usually the Staphylococcus aureus. Furunculosis occurs in conditions of lowered vitality, as during the convalescence from infectious diseases, especially enteric fever. The occurrence of furuncles in crops, or their persistent recurrence, is a common event in diabetes mellitus and should always lead to an examination of the urine for the presence of sugar.

CICATRICES OR SCARS.—These, whether recent or old, constitute important diagnostic signs. In doubtful cases special significance attaches to the presence or absence of the scars of vaccination and their characters and to the scars of smallpox. The scars of furuncles and carbuncles, of lupus, of inguinal buboes, and those left by tuberculous glands which have healed spontaneously or been removed are very suggestive in doubtful walls on the one hand or to thrombosis or pressure on the other. Enlargement of the veins of both legs is caused by the obstruction of the vena cava or both iliacs. Great enlargement of the veins of the legs, with the formation of varices, sometimes occurs after repeated pregnancies, and enormous varicosity of one leg, with great dilatation, frequently results from venous thrombosis following pregnancy or the infectious diseases, especially enteric fever. The varicosities which occur in the absence of pressure or thrombosis are largely due to changes in the walls of the veins themselves.

THE NAILS.

The appearance of the nails is to some extent indicative of the state of nutrition and habits. The deformity arising from biting the nails is characteristic and consists in shortening of the nail with projection of the tip of the finger, into which the edge of the nail tends to bury itself. Coarse longitudinal striæ associated with brittleness are said to indicate gouty tendencies. Small white flecks—*leucopathia unguis*—are the result of trifling knocks; the color is due to the presence of air among the cells. Transverse arched bands, dull and opaque, contrasting with the normal glistening surface, are seen after severe acute illness and indicate a period of malnutrition. They appear at the root of the nail and gradually advance. They are often seen after enteric fever and sometimes, in the case of relapse, there is a corresponding secondary band. Pressure upon the nails drives blood from the capillaries of the bed. The blanching is marked and somewhat prolonged in anæmic states. The nail is normally of a pink tint. Cyanosis shows itself early in the nails and their blueness is a measure of its intensity. When the capillary pulse is present it may be seen in the nail-beds, especially after slight pressure. The nutrition of the nails is affected in various skin diseases. They become dry, fragile, and malformed in neuritis, syringomyelia, Raynaud's disease, and scleroderma. Destruction of the nails occurs in the neuritis of Morvan's disease and leprosy. In hemiplegia and infantile palsy the growth of the nails upon the paralyzed side is retarded. *Onychia* is ulceration of the nail matrix. It may be due to syphilis or tuberculosis. In chronic disease of the chest the nails become hypertrophied and incurvated and the terminal phalanges clubbed—the *Hippocratic fingers.* These changes are seen most frequently in bronchiectasis and empyema, less often in phthisis. The deformity may develop very rapidly. Trifling lesions at the root of the nail—a mere splitting of the fold of epidermis at the side of the finger-nail, may be the point of serious infection. Malignant endocarditis and tetanus have arisen from this cause, and such sores upon the finger of the surgeon are frequently the seat of the initial lesion of syphilis. Congenital absence and deformities of the nails are not common. They may be hereditary and are usually associated with defects in development of the hair and teeth.

Shedding of the nails sometimes occurs in syphilis, alopecia areata, saccharine diabetes, hysteria, and other neurotic conditions. Extravasation of the blood beneath the nails may occur from injury or very rarely in purpuric affections. The blood-clot brings about a separation of the nail from its bed and its ultimate detachment.

THE HAIR.

Wide variations in color, texture, and abundance occur in different individuals. Certain changes are of diagnostic importance.

Color.—Grayness or canities may begin early in life. It is a sign of old age but there are people who grow old without growing gray. Gray hair in young people is sometimes hereditary. It is often associated with early arteriocapillary sclerosis. In a family in which nearly every member for three generations was the victim of chronic nephritis it was characteristic for the hair to turn gray before thirty. Early grayness, however, is not incompatible with excellent health. In rare instances rapid whitening of the hair has been attributed to extreme terror or anxiety. Circumscribed patches of gray hair are occasionally seen in healthy individuals. Their development sometimes appears to depend upon severe neuralgia involving the distribution of the supra-orbital branch of the fifth nerve. It is easy for the close observer to detect bleached or dyed hair. To the physician the former is suggestive of an undisciplined life, the latter of chronic lead poisoning as the cause of nervous symptoms otherwise obscure. Discoloration of the hair occurs in workers in copper, cobalt, indigo, and from local contact with dyes. Change in color may occur after severe illness with temporary loss of hair or after frequently repeated excessive sweating such as follows the hypodermic use of pilocarpine.

Hypertrichosis.—A growth of hair that is abnormal in quantity or in location may be congenital or acquired. It is a deformity rather than a disease. Very rare instances have been reported in which a growth of hair has covered the entire body except the palms, soles, terminal phalanges, upper eyelids, borders of the lips, prepuce, and glans penis. Hirsuties is more commonly localized. The causes of this condition are obscure. Among them heredity and irregularities or arrest of the sexual functions are prominent. A luxuriant growth of deeply pigmented hair has been observed in Addison's disease.

Atrophy of the hair occurs as the result of systemic conditions interfering with nutrition. The hair becomes dry and lustreless and splits at the end. It may undergo atrophy in local diseases of the scalp and in general conditions, as extreme emaciation and cachexia. Imperfect nutrition of the hair is conspicuous in myxœdema and occurs in advanced cases of pulmonary consumption.

Alopecia may involve the scalp or other hairy parts of the body. It may be congenital and is usually accompanied by defects in the teeth and nails. This form is very often hereditary. The hair does not usually grow in scars upon the scalp. Alopecia senilis accompanies other senile changes. Premature falling of the hair sometimes appears to be an idiopathic condition. It occurs in various local and systemic diseases. Among the latter are acute febrile infections, syphilis, and erysipelas. As a rule the hair grows again.

ALOPECIA AREATA or circumscribed patches of baldness appears in some instances to be a trophoneurosis occurring after shock or injury to the nervous system. In others it appears to be a local parasitic disease.

Diseases of the skin as such do not fall within the scope of this work. The cutaneous manifestations of the individual constitutional and organic diseases are considered elsewhere (see Part IV).

XII.

GENITO-URINARY SYSTEM; MICTURITION; THE REPRODUCTIVE ORGANS.

The diagnostic significance of the results of examination of the urine by laboratory methods is set forth in a previous chapter. The clinical facts may properly be considered separately.

MICTURITION—URINATION.

The urine is secreted continuously and conveyed by the ureters to the bladder, from which it is ejected at intervals through the urethra by the act of micturition. The urine accumulating in the bladder is prevented from escaping by the elasticity of the parts surrounding the internal urethral orifice and the contraction of the internal sphincter. When the accumulation reaches a certain point the desire to pass water is aroused. The external sphincter may be controlled by voluntary effort. The act of micturition consists in strong contraction of the bladder with the simultaneous relaxation of the sphincters and the contraction of the abdominal muscles, especially toward the close of the act. The contraction of these muscles with closure of the glottis and fixation of the diaphragm increases the pressure upon the contents of the abdominal and pelvic cavities and favors the complete emptying of the bladder. The peculiar sensation caused by the accumulation of urine in the bladder is followed by the reflex muscular contractions which constitute the act of micturition. Not only is this act largely under the control of the will, but the ability to void small quantities is also to some extent voluntary.

The average total daily quantity of urine in healthy men is 1200 to 1700 c.c.; in women the amount is less by 200–300 c.c. This represents the water excreted by the kidneys, but there is in health as well as in disease a vicarious relationship between the function of those organs and the skin and lungs, so that during increased cutaneous and respiratory activity, as in prolonged exercise or in warm weather, the quantity of urine may be reduced to 400–500 c.c. in twenty-four hours.

The quantity is diminished in disease in a corresponding manner. Thus, the loss of fluid by pathological sweating, profuse vomiting, colliquative diarrhœa, and hemorrhage is attended by more or less marked reduction in the urine. The quantity is also reduced in acute nephritis, in lowering of the blood-pressure from any cause, in many febrile conditions, and in dropsies and effusions into the serous sacs. Suppression of urine more or less complete is designated *anuria*—to a less extent *oliguria*.

An abnormal and continued increase of the daily quantity of urine, not accounted for by increased ingestion of fluid, constitutes the pathological condition designated *polyuria*. This condition occurs in diabetes insipidus and mellitus, in emotional states, in hysteria, during the epileptic paroxysm, in irritable lesions of the floor of the fourth ventricle, under the influence of diuretics, in contracted kidneys, in chronic parenchymatous nephritis, in lardaceous renal disease, from increased blood-pressure, and as a result of the resorption of transudates and exudates.

The daily quantity is voided in several acts of micturition, usually about five, but the number within normal limits is largely determined by the habits and circumstances of the individual.

The following abnormal conditions are of diagnostic importance:

(a) **Dysuria.**—This term is comprehensively employed to designate difficult, slow, and frequent micturition, and, since these symptoms are mostly though not always attended with distress which is often urgent, it includes painful micturition.

1. **Vesical tenesmus** constitutes the most severe form of dysuria. It consists of painful spasm of the bladder and is often associated with rectal tenesmus. The spasm is often so urgent that the patient is unable to remain at rest, but returns at short intervals to the ineffectual and agonizing attempt to pass water, with the result that a few drops at most are voided with violent bearing down and burning pain in the urethra.

2. **Strangury.**—Not rarely a few drops of blood or bloody mucus are discharged in the spasmodic efforts at urination, and the condition is described as strangury. This term is, however, frequently employed interchangeably with tenesmus.

Very concentrated and acid urine is a cause of dysuria and the ingestion of certain condiments in excess, as mustard, pepper, and horseradish, may produce similar inconvenience. The absorption of cantharides and turpentine applied to the surface, or overdoses of these substances, may be followed by strangury. A sudden attack of vesical tenesmus for which no obvious cause is discoverable may be found upon investigation of the facts of the case to be a tabetic crisis.

Dysuria, especially these more intense forms, is liable to occur in almost all acute inflammatory diseases of the urinary tract. They are encountered therefore in posterior gonorrhœa and in gonorrhœal inflammation of the neck of the bladder, and in acute cystitis, prostatitis, and pyelitis. Dysuria also accompanies the chronic forms of these affections but is much less urgent and distressing. Tenesmus is also symptomatic of direct irritation of the bladder, as by stone, gravel, foreign bodies, parasites, and local ulceration. Reflex dysuria with tenesmus is sometimes present in renal colic.

Dysuria is a symptom of incomplete retention. When after the act of micturition there is residual urine, it is evident that the capacity of the organ will be more speedily reached than when it is emptied normally. Urination becomes more frequent and more difficult. This form of dysuria occurs in paresis of the bladder, as in tabes, hypertrophied prostate, tumor involving the neck of the bladder, stricture and phimosis, prostatic abscess, arteriosclerosis of the vesical arteries, spasm of the neck of the bladder, and œdematous swelling of the urethral mucosa in acute gonorrhœa. A chancre of the urethra may act in the same way, and in the variolous diseases pocks in the meatus may occasion similar symptoms.

Dysuria frequently attends general peritonitis, acute inflammatory diseases of the pelvic organs, and may occur in dysmenorrhœa.

(b) **Frequent Micturition.**—This occurs in polyuria. It is obvious that in the absence of dilatation of the bladder an increase in the quantity of urine must be followed by an increase in the frequency with which it is voided. Hence in diabetes insipidus and diabetes mellitus, in contracted kidneys and in some forms of pyelitis the frequency of urination is greatly increased. A diabetic who voids 6 litres of urine in twenty-four hours, with an average vesical capacity of about 300 c.c., would be obliged to pass water at least twenty times .in the course of the day—a requirement which is slightly diminished by a gradual increase in the size of the bladder. When, on the other hand, the bladder has undergone concentric hypertrophy in consequence of chronic cystitis and its capacity is greatly diminished, the necessity to void urine at short intervals becomes imperative. If the call be not obeyed, as in deep sleep, the urine may be involuntarily discharged.

Frequent micturition is often due to psychical causes, among them fright and excitement. Soldiers in battle and students awaiting examination constitute oft-quoted examples. The urine is voided at short intervals and in small amounts and often involuntarily. It is also a common symptom in hysteria and neurasthenia.

(c) **Slow Micturition.**—The act is slow, prolonged, and difficult in all conditions characterized by mechanical obstruction to the outflow and in nervous affections attended by paresis of the vesical wall. Hence the form of dysuria encountered in incomplete urinary retention from any cause is characterized by slow or prolonged micturition. *Stillicidium urinæ* or the slow discharge of urine drop by drop has been described under the term "incontinence of retention." It occurs in the low fevers and in soporose and comatose conditions, when, because of the neglect of a routine physical examination and of the use of the catheter, the bladder has been allowed to become overdistended.

(d) **Incontinence of Urine.**—This condition is due to mechanical and nervous causes.

1. MECHANICAL CAUSES are chiefly operative in women. Laceration of the perineum or injuries to the urethra sustained in parturition, relaxation of the floor of the pelvis, and cystocele are common causes of urinary incontinence. The urine may dribble constantly or be discharged in gushes upon any muscular effort which increases the pelvic pressure, as lifting, stooping, or coughing. Violent sudden cough, as in pertussis, may cause incontinence in depressed or asthenic conditions with relaxation of the sphincter muscles.

2. THE NERVOUS CAUSES of incontinence are much more common. They may be cerebral, as in coma and shock, idiocy and dementia, or the stuporous states of the profound infections; spinal, as in traumatism, hemorrhage, and tumors of the cord, transverse myelitis, meningitis, and tabes; or reflex in consequence of the local irritation of ascarides, phimosis, contracted urinary meatus, stone in the bladder, cystitis, or highly concentrated or acid urine. To the last of these causes may be referred enuresis nocturna, which occurs in neurotic children and acquires the force of a morbid habit, the urine being voided involuntarily, as a rule during sleep, but frequently during the waking hours under excitement or preoccupation. If the vesical centre in the lumbar cord is destroyed, complete paralysis will ensue, with retention or the dribbling incontinence of retention.

(e) **Retention of Urine.**—Retention and incontinence are very constantly associated, and are due in many instances to the same causes. Thus, retention may occur in coma from any cause, in the soporose states incident to profound toxæmia, as in the graver forms of the infectious diseases and especially in the terminal infections, in peritonitis, in acute pelvic inflammations, and in injuries and diseases of the spinal cord.

Temporary loss of vesical power sometimes results from overdistention in consequence of prolonged voluntary retention. Mechanical causes of retention are stricture, urethritis, the arrest of a calculus in the urethra, prostatic enlargement, and the pressure of the head in parturition. Remarkable retention of urine is sometimes observed in hysterical persons.

In infants this condition may occur from phimosis, inflammation of the prepuce, or highly concentrated or acid urine. These causes may act reflexly, by producing spasm of the sphincters, or mechanically. The passage of a renal calculus through the ureter may, by reflex irritation, give rise to frequent micturition on the one hand or to spasm of the sphincters and retention on the other.

(f) **Suppression of Urine—Anuria.**—This condition may be mechanical, renal or general, partial or complete.

1. Mechanical causes of complete anuria are renal calculi blocking both ureters simultaneously or the ureter when only one exists. The symptoms are those of uræmia. The condition is extremely rare. Life may be prolonged several days with complete anuria; in Polk's case in which a solitary kidney was removed, the patient lived eleven days. *Partial anuria—oliguria*—may be caused by the presence of an abdominal aneurism or tumor upon one or both ureters, or by a kink in the ureter in the case of an ectopic kidney, or by malignant disease of the wall of the bladder involving one or both urethral orifices. In any of these conditions hydronephrosis may occur.

2. Renal lesions leading to suppression of urine are acute congestion, acute nephritis; the acute exacerbations of chronic nephritis, pyelitis, abscess of the kidney, perinephric abscess, and hydro- and pyonephrosis. Among the rare causes of suppression is thrombosis of the inferior vena cava or of the renal vein.

3. General conditions accompanied by suppression of urine are extreme lowering of the blood-pressure such as occurs in profuse hemorrhage from any cause; collapse or shock from injuries, surgical operations; the perforation of hollow viscera, as in peptic ulcer, empyema of the gall-bladder, enteric fever, or rupture of the uterus; the stage of collapse in cholera Asiatica, cholera nostras, or yellow fever, the pernicious malarial fevers, and acute peritonitis. Operations upon the urinary tract—even so trifling a procedure as catheterization—may in elderly men be followed by urinary suppression.

This symptom also occurs in acute poisoning by phosphorus, lead, and turpentine, in acute yellow atrophy of the liver, and in sunstroke.

Anuria, more or less complete, and prolonged for days, is occasionally observed in hysterical girls. In rare cases there are symptoms of uræmia, but as a rule there are no associated symptoms other than those due to the hysteria. In such cases, in order to avoid deception, the patient must be isolated and carefully and continuously watched, and the catheter used at unexpected and irregular periods. Anuria may result from reflex irritation and functional arrest in a normal kidney, the ureter of the opposite side being blocked by a calculus, or the opposite kidney having been removed by operation.

Hæmaturia.—When small amounts of blood are present the color of the urine is smoky. With larger quantities it is bright red or even dark brown and opaque like porter. Erythrocytes are present, usually crenated or as rounded shadowy disks. The hæmoglobin is soon dissolved, especially in ammoniacal urines and those of low specific gravity. Blood from the kidneys is intimately mixed with the urine, which is discolored both at the beginning and at the end of the act of micturition. Clots are often present and they may be in the form of casts of the pelvis or ureters. Blood from the bladder may not appear until toward the end of micturition or at its close. Upon washing out the bladder the water returns tinged if the source of the hemorrhage be in the bladder but clear if it be in the kidneys. The differential diagnosis of the source of the bleeding, however, is often attended with difficulty and can be made only by means of the cystoscope or a differentiator by which the urine from each ureter may be obtained separately.

Hæmaturia may be symptomatic of the following conditions:

1. THE HEMORRHAGIC VARIETIES of the acute febrile infections, forms of purpura, hæmophilia, very severe cases of scurvy, and leukæmia. A special form of hæmaturia or hæmoglobinuria—black water fever—prevails in certain malarious districts.

2. DISEASES OF THE URINARY PASSAGES.—Sarcoma or tuberculosis of the kidney, calculus in the ureter, tumor, ulceration or calculus in the bladder, parasites of the bladder—Bilharzia hæmatobia, psorospermiasis—or rupture of veins in its wall may be the cause of hæmaturia. In rare instances this condition is due to disease of the prostate. The arrest of a calculus in the urethra or acute gonorrhœal urethritis is sometimes attended by the passing of blood. This symptom occurs in strangury and there are cases of persistent hæmaturia in which no adequate lesion has been found.

3. TRAUMATISM.—Hæmaturia follows operations upon the kidney. Gun-shot wounds or stabs involving the kidney, laceration of the organ from blows upon the back, falls or crushing accidents cause profuse bleeding. Similar injuries involving the bladder or prostate, falls or kicks resulting in severe contusion of the perineum and laceration of the urethra are also followed by hemorrhage, and this symptom frequently follows the use of the catheter.

(g) **Hæmoglobinuria.**—The urine is discolored by hæmoglobin, chiefly methæmoglobin. Red corpuscles are absent or few in number. The urine is smoky or brownish-red, even black, and upon standing deposits a dense, dirty brown sediment made up of granular pigment, the detritus of blood-corpuscles, epithelium, and pigmented urates.

Three forms are recognized: the toxic, the paroxysmal, and hæmoglobinuria of the new-born.

1. TOXIC HÆMOGLOBINURIA.—This variety is encountered in poisoning by those agents which produce rapid destruction of the erythrocytes.

Important among these are potassium chlorate, pyrogallic acid, carbolic acid, arseniureted hydrogen, carbon monoxide, naphthol, and muscarine. It is also produced by the transfusion of blood from one mammal into another, by exposure to intense cold and violent exertion, and occurs after extensive burns. In malarial subjects it may follow the administration of quinine—*black water fever.*

2. Paroxysmal Hæmoglobinuria. — An affection characterized by the occasional passage of urine colored by hæmoglobin. It occurs in adults and is more common in males than in females. The paroxysms are excited by cold and exertion and last from a few hours to a day or two. It is thought by some observers to have an essential relationship to Raynaud's disease; by others to malaria. Pain in the lumbar region is common. The attacks may be ushered in by chills followed by fever; more commonly the temperature is normal or slightly subnormal. They recur at irregular intervals for an indefinite time.

3. Epidemic Hæmoglobinuria of the New-born. — The disease develops about the fourth day and attacks a large proportion of the infants in the maternity institution where it appears. There is bloody urine with vomiting and purging, jaundice, hurried breathing, and cyanosis. It is rapidly fatal. Post-mortem examination reveals enlargement of the spleen with punctiform hemorrhages upon the surface and in the parenchyma of the viscera. This disease is to be differentiated from icterus neonatorum to which it bears only a superficial resemblance.

THE REPRODUCTIVE ORGANS.

In both men and women sexual neurasthenia, hypochondriasis, and perversion frequently occur. Ungratified desire, excessive venery, and unnatural sexual acts are more commonly the alleged than the actual causes of various nervous and mental diseases. The two latter are probably manifestations more often than causes of such forms of disease. Irregular manifestations may be on the one hand psychical, on the other physical; frequently they are both. The field is a large one and the extent to which it is to be investigated in individual cases may be left to the judgment of the clinician.

The history or the actual manifestations of venereal disease in a patient, or in an individual with whom the patient has had sexual relations, are often of great importance in the diagnosis of an otherwise obscure case. A gonorrhœal discharge may solve the problem of an obscure and intractable arthritis or indicate the nature of serious tubal or other pelvic disease, and explain an unlooked for ophthalmia in the new-born. Syphilitic lesions or the scar of a chancre in the husband may be the key to the solution of obscure nervous symptoms in the wife, or nutritional disorders and lesions of the organs of special sense in the child.

In the male, priapism, impotence, and spermatorrhœa occur as important manifestations of disease.

(a) **Priapism.**—This term is used to designate abnormally frequent and prolonged erection. The condition is not associated with *libido sexualis* but with distress and pain and constitutes a morbid symptom.

It is often manifest in a mild degree in young boys. Even at the age of one or two years it may be painful and distressing and often leads to *enuresis nocturna.* It may be due to phimosis and disappear after circumcision. In the adult it may result from inflammatory irritation of the urethral mucosa. It may follow the passing of a bougie and is very common in gonorrhœa and in the chronic inflammation of the prostatic portion of the urethra in those who have practised masturbation or indulged in sexual excesses or irregularities. The condition may be due to excessive stimulation of the centre in the lumbar cord. The latter form comes on during sleep. The patient awakes with intensely painful priapism unattended by the slightest libido sexualis. This presently subsides only to return when, under the influence of deep sleep, the inhibition of the special spinal centre is withdrawn. In severe cases sleep is seriously interrupted and the annoyance of the patient is increased by the discharge of a thin mucus from Cowper's glands and painful neuralgia in various parts of the body. This form of priapism is often accompanied by impotence.

Priapism may be the result of stone in the bladder, inflammation of the prostate, a perineal abscess, proctitis or periproctitis, inflamed hemorrhoids, or poisoning by cantharides. It is said to be symptomatic of certain forms of neurasthenia and hysteria. It is a common symptom in fractures of the spine, especially when the cervical portion is involved. It may occur in myelitis, spinal meningitis, and in lesions of the pons and cerebellum. It occurs in hydrophobia and tetanus and has frequently been observed in leukæmia.

(b) **Impotence—Impotentia Coeundi.**—This symptom may be mechanical, psychical, irritative, or paralytic.

1. MECHANICAL IMPOTENCE arises from congenital or acquired deformities; loss of substance from ulceration, gangrene, or operation; the presence of tumors, as hydrocele, enormous hernia, elephantiasis of the scrotum, and the like. To this list must be added hypertrophy of the organ, tumor of the glands, preputial or urethral calculi and defect, atrophy or destruction of the testicles. To this group of causes is to be added deviation of the erect penis from abnormally short frænum and various infiltrations and indurations in its tissues. A rare cause of impotence is deformity due to ossification of the fibrous tissue in the organ.

2. PSYCHICAL.—This form of impotence arises from apprehension, shame, or self-distrust. It may occur alike in those who have made too great experience and in those who have made none, and the fear of it frequently leads men about to marry to take medical advice. It is sometimes due to indifference, aversion, or dislike towards a particular person and in rare instances to constitutional lack of sexual feeling.

3. IRRITATIVE.—There is premature ejaculation or even ejaculation in the absence of sexual approach. This may occur in healthy individuals after long abstinence. It is very often due to local irritation, to lesions resulting from urethritis, or to excesses. The subjects are usually neurasthenic, the nervous condition being the cause in some cases, in others the effect of the sexual irregularity.

4. PARALYTIC.—Under this heading are to be grouped those forms of impotence caused by the loss of power to react to physiological stimuli on

the part of the sexual nerves or their centres. In the atonic cases anæmia and relaxation of the parts are present and the patients are neurasthenic. Sexual irregularities and excesses, immoderate indulgence in alcohol and tobacco are causes. Certain drugs, as opium and its derivatives, nitre, the salicylates and the bromides, taken in large doses or for long periods of time, lead to this form of impotence.

Diseases of the brain and spinal cord may be the cause of paralytic impotence. Tabes dorsalis and other affections, characterized by impaired function of the bladder or rectum or by local anæsthesia, are especially to be considered. This condition is also symptomatic of diabetes mellitus, obesity, and cachectic states.

(c) **Spermatorrhœa.** — This term is used to designate the pathological discharge of seminal fluid which takes place without erection or sexual sensation during the act of micturition or defecation. The emissions which occur at intervals of two or more weeks in continent young men during sleep, and which are accompanied by lascivious dreams, are physiological rather than pathological and are not to be considered under this heading. When, however, these emissions recur at short intervals, or every night, they become symptomatic of disease and the border-line between such nocturnal pollution and spermatorrhœa is no longer clearly defined. Gonorrhœa, onanism and sexual excesses are liable to be followed by spermatorrhœa. Constipation, nervous diarrhœa, fissure of the anus, seat-worms, and proctitis may act as accidental causes. The patients are neurasthenic and depressed, complain of headache, backache, and loss of energy, are much given to the reading of advertisements upon loss of manhood and are the easy prey of quacks. A large proportion of those who think they are victims of this disease do not have it, but suffer from chronic gonorrhœa, prostatorrhœa, urethrorrhœa, and forms of phosphaturia. The microscope is essential to the diagnosis, and it is necessary when spermatozoids are present to ascertain whether or not a sexual act has preceded the emission of the fluid in question. If not, and especially if spermatozoids are present upon repeated examination, the diagnosis becomes positive. These bodies are present in the urine, which may be acid, of high specific gravity, and contain oxalates, or alkaline with phosphates.

In the female pruritus vulvæ, leucorrhœa, and disorders of menstruation may be symptomatic of various local and general conditions.

(a) **Pruritus Vulvæ.**—This condition is a common result of inflammatory affections and displacements of the womb, ovarian disease, and affections of the urethra, bladder, and kidneys. It is, especially in children, a common manifestation of seat-worms and is very often the first symptom of the diabetic woman to attract her attention to her condition. This condition on the one hand frequently leads to masturbation; on the other is not rarely the result of it.

(b) **Leucorrhœa.**—Vaginal discharge is an important sign of many pelvic diseases. It is associated, very often in connection with pelvic inflammations of mild grade, with the anæmias, especially when intense, with conditions of debility and the later stages of chronic diseases when they occur in early life and in particular with pulmonary tuberculosis. In young children a purulent discharge indicates vulvitis or vaginitis, which may be due to trauma, filth, ascarides, or gonorrhœa. In middle life an offensive sanguinolent discharge may be the earliest sign of carcinoma uteri.

(c) **Menstrual Derangements.**—The normal menstrual function may be deranged in various ways. It may be absent for a time or cease altogether—amenorrhœa; abnormally profuse—menorrhagia; or attended with much distress and pain—dysmenorrhœa. These derangements are due to local and to constitutional conditions.

1. AMENORRHŒA.—Failure in the function may be a manifestation of arrested development of the ovaries and uterus. The interruption of menstruation may be physiological or pathological.

Physiological amenorrhœa is a characteristic sign of pregnancy and usually persists during lactation. There are important exceptions to both these rules. In very rare instances there is a slight menstrual discharge during the first two or three months of gestation and many women menstruate regularly during the period of nursing. Amenorrhœa occurs in extra-uterine fœtation.

Pathological amenorrhœa is observed in conditions of malnutrition, as in overworked school-girls, in those suffering from chlorosis, and in wasting diseases, as enteric fever, tuberculosis, diabetes, and exophthalmic goitre. It may be symptomatic of powerful depressing psychical states, as anxiety, worry, or grief, and of nervous affections, as hysteria, or of melancholia or other forms of insanity, and not infrequently occurs in young immigrants. It is common in morphinism and other drug habits and in cachectic states, whether due to chronic intoxication, as by mercury or lead, or to malaria, cancer, nephritis, leukæmia, or profound anæmia from any cause. The retention of the flow which takes place in cases of imperforate hymen, atresia vaginæ, and analogous conditions cannot be regarded as a form of amenorrhœa.

Delay in the establishment of menstruation is in some girls constitutional and often hereditary; its early cessation may in some instances be accounted for upon similar grounds. There are healthy women who cease to menstruate at thirty or thirty-five. Premature menopause may be due to atrophy of the ovaries following disease or their operative removal.

So-called vicarious menstruation, namely, the monthly discharge of blood from the nose, lungs, stomach, from hemorrhoids, ulcers or wounds, in the absence of the normal flow, is described. There is no physiological basis for such a phenomenon and it is probable that in the cases described the conditions causing amenorrhœa have also caused hemorrhages, the regular periodicity and duration of which have corresponded to the menstrual period less in fact than in fancy.

2. MENORRHAGIA.—Abnormally profuse menstruation may be symptomatic of disorders of the pelvic organs or of constitutional disease. It occurs in a great variety of local diseases but especially in chronic endometritis, submucous myomata, polypi, and uterine displacements. Menorrhagia is an occasional symptom in hæmophilia, scurvy, purpura hæmorrhagica, and leukæmia. When menstruation takes place in the course of the acute infectious diseases, for example influenza, enteric fever, or variola, it frequently amounts to menorrhagia. Other conditions in which this symptom is occasionally observed are intense jaundice, phosphorus poison-

ing, alcoholism, cirrhosis of the liver, and valvular disease of the heart. The administration of certain drugs, as ergot, gossypium, aloes, and the oil of savine, is sometimes followed by menorrhagia. Irregular menstruation, sometimes profuse, not infrequently precedes the menopause.

3. DYSMENORRHŒA.—This term is used to designate collectively the symptom-complex in difficult menstruation of which pain is the chief element. The morbid conditions in which it occurs may be arranged under two headings, affections of the sexual system and general diseases.

Under the first heading are to be included those diseases in which there is an obstruction to the outflow of the menstrual fluid, as in contraction of the internal or external os uteri, congenital narrowing of the cervical canal or a narrowing acquired as the result of flexions of the uterus, the presence of tumors or cicatricial contractions following unwise treatment. This form is spoken of as mechanical dysmenorrhœa. Here also are to be considered the dysmenorrhœas caused by irritable or inflamed conditions of the mucosa secondary to chronic metritis, displacements, tumors and disease of the ovaries.

Under the second heading we include the dysmenorrhœa of neurotic persons—neuralgic or nervous dysmenorrhœa. This form is common alike in badly-nourished, anæmic, unmarried women and in women who have borne children. Very frequently no adequate lesions of the pelvic viscera can be discovered; more commonly trifling abnormalities such as cause insignificant symptoms in otherwise well-nourished and healthy women. The patients are neurasthenic and frequently hysterical. The symptoms vary greatly. In many cases they amount merely to an intensification of the ordinary discomforts which attend the periodical sickness; in others the patient may writhe with anguish or manifest the most intense reflex phenomena as nausea, vomiting, headache, or convulsions. Usually these symptoms subside upon the establishment of the flow; sometimes they continue with remissions and exacerbations throughout the whole period, and in some cases they cease entirely only to recur toward the close of the process.

Membranous dysmenorrhœa—decidua menstrualis—a form of dysmenorrhœa in which, with recurring menstruation, hollow membranous casts of the uterus are expelled with great pain. These casts consist of a thickened menstrual decidua. They vary from membranous fragments to complete triangular casts of the interior of the womb, showing the openings of the tubes and the internal os. They are usually expelled upon the second or third day, sometimes later. The pains are paroxysmal and very intense and cease immediately upon the expulsion of the membranes from the womb. This form of dysmenorrhœa is sometimes encountered in women suffering from chronic metritis or endometritis. It is very chronic, sometimes continuing throughout the entire menstrual life of the individual. There is complete relief during the intermenstrual periods. The condition is to be differentiated from early abortion and extra-uterine pregnancy.

4. METRORRHAGIA.—An abnormal uterine hemorrhage is to be distinguished from an excessive menstrual discharge or menorrhagia, with which it is, however, very commonly associated. It may occur in diseases of the reproductive organs or in certain general affections. Metrorrhagia due to

local disease usually indicates disease of the uterus and mostly the presence of new growths, namely, carcinoma, sarcoma, or fibroid tumors. The bleeding in carcinoma at first takes the form of an increased menstrual flow usually more and more prolonged and frequently accompanied by a more or less abundant watery discharge. The bloody discharge after a time persists during the intermenstrual periods and becomes wholly atypical. The occurrence of bleeding in women who have passed the menopause is very suggestive and renders an examination per vaginam at once imperative. The metrorrhagia of sarcoma and in particular of sarcoma involving the uterine mucosa presents similar characters. Subserous fibromata do not bleed. Those situated in the substance of the uterus, if near the serous surface, bleed little or not at all. Submucous fibromata bleed more or less freely. Necrotic changes in uterine neoplasmata are attended by a foul-smelling discharge in which shreds of broken-down tissue are present. The atypical bleedings which attend inflammatory affections are less frequent and less profuse. Those which are caused by mucous polypi are often profuse and continuous.

Exceptionally metrorrhagia occurs in valvular disease of the heart, especially mitral stenosis, and is said to have been observed in cirrhosis of the liver. This symptom occurs infrequently in the acute infectious febrile diseases, as enteric fever, measles, scarlet fever, variola, cholera, and malaria, and in phosphorus poisoning and scurvy. In the last the blood loss is sometimes copious. Difficulties arise in the differential diagnosis of the cause of the bleeding when the patient suffering from the foregoing diseases has also local conditions in themselves capable of causing metrorrhagia or when, during the acute illness or shortly before its onset, an abortion or miscarriage has taken place.

XIII.

GENERAL SYMPTOMATIC DISORDERS OF THE NERVOUS SYSTEM.

PAIN.

Pain is a symptomatic sensory neurosis. The pain sense is to be distinguished from the tactile sense, the pressure sense, and the thermal sense. It is, however, so closely associated with the last two that a considerable degree of pressure, unusual heat, or intense cold is accompanied by pain. Pain is in the strictest sense a symptom. It is purely subjective, hence its value in diagnosis is to a large degree dependent upon the individual peculiarities of the sufferer, the nature of the primary lesion or disease, and concomitant phenomena, many of which are objective. Judged by these standards pain is a symptom of the most varied intensity, from a trifling discomfort without direct diagnostic significance to agony so extreme as to cause death. The pain sense is universally distributed throughout the body, the only structures in which it is wholly lacking being the hair and nails. Variations in the pain sense in different localities, probably due to modifications in the sensory nerve supply, must be invoked in explanation of the different kinds of pain in the various viscera and other anatomical structures. Etiological factors of the most diverse kind have to do with pain in its relation to time, as shown in its onset, course, and decline.

Pain is dependent upon consciousness. In profound coma, as that of surgical anæsthesia, consciousness and pain are alike wholly abolished. When consciousness is less completely impaired there are objective manifestations of painful impressions, though the patient, upon recovering, may have no recollection of pain. Pain may be absent in shock. Individuals usually make no complaint of pain during the period of shock following gun-shot wounds or other severe traumatism. Under these circumstances pain comes on as shock subsides.

Etiology.—Pain is functional or organic. The temporary pain in overworked muscles is functional. The pain in pleurisy and gastric ulcer is organic. Pain occurs as a more or less prominent symptom under the following conditions:

1. Excessive or unduly prolonged physiological activity, either physical, as in muscular strain or fatigue, or psychical, as in the headache which follows undue intellectual effort. The pains of parturition are physiological.

2. Traumatism of all kinds.

3. Circulatory disturbances. (a) Passive congestion. An example of pain thus caused is to be found in thrombosis of the crural vein, formerly known as *phlegmasia alba dolens*. (b) Active hyperæmia, for example, the cutaneous pain of local irritants, as heat, cold, mustard and the like. Pain in the region of the spleen after running is an example of visceral pain due to this cause. (c) Anæmia. Examples of this form of pain are headache upon exertion and the neuralgias.

4. Inflammation. Pain is a prominent symptom in all forms of inflammation.

5. Toxæmia. The offending substance or substances in the blood may be the result of (a) infection, as in the acute specific fevers and malaria; (b) incomplete or perverted physiologicochemical processes or the defective elimination of waste, as in the headache of uræmia and diabetes and the pains of gout, rheumatism, and lithæmia; (c) the action of drugs or poisons. Pain due to this cause may be hyperæmic, as in the head pain produced by amyl nitrite and quinine; inflammatory, as in the later stages of narcotic poisoning; purely nervous, as an abstinence symptom in morphinism and the pains of the chloral habit and lead colic.

6. Changes in the arteries. Examples of pain due to this cause are found in syphilis, chronic alcoholism, chronic lead poisoning, migraine, and aneurism. To this general topic must also be referred the pain in intermittent claudication and angina pectoris.

7. All organic painful diseases, abscess, tumor, both benign and malignant, and various diseases of the viscera, whether the pain be due to changes in the organ itself or disturbance of adjacent structures by pressure or displacement.

8. Caries and other diseases of the bones.

9. Neuropathic conditions, for example, neurasthenia, hysteria, tabes, dysmenorrhœa, and tetanus.

10. Reflex irritation, as the supra-orbital pain in indigestion and the various localized head pains of eye-strain, pain in the external auditory meatus in dental irritation, and coccygodynia in uterine disease. Analogous are the pains in the knee which occur in hip-disease and painful sensations due to the irritation of the nerve stump referred to the hand or foot, as the case may be, in an amputated limb.

The cause of pain is very often simple. In many cases, however, it is complex, two or more of the foregoing factors being operative.

Mode of Expression of Pain.—Pain must be studied subjectively, as we experience it in our own person, and objectively, as manifested by the movements, attitudes, and verbal descriptions of the sufferer.

Subjectively we know that certain external impressions give rise to the sensation of pain and that this sensation is accompanied by movements of withdrawal from the object causing the pain, by particular attitudes of the body and contortions of the facial muscles. Under certain circumstances there are inarticulate sounds, cries or groans expressive of pain; these phenomena are varied according to the suddenness and the intensity of the pain and its character.

Objectively we recognize in these phenomena a manifestation of pain in others. The gestures that are characteristic of different varieties of pain have been described by W. H. Thomson. In pains due to inflammation the patient avoids touching the painful part, or approaches it very cautiously. Thus the hand passes over an inflamed joint with a hovering gesture. If the pain be deeper seated the gestures are indicative of its distribution and the character of the inflamed tissue. Thus the substernal pain of bronchitis as indicated by the whole hand laid upon the sternum and passed over the chest. In pleurisy the location of the pain is indicated by the tips of the straightened fingers, the natural gesture expressive of the stabbing or lancinating character of the pain. Precordial pain, if severe, is indicated by the tips of the bent fingers. The gestures by which abdominal pain is indicated are equally significant. In pains associated with lesions of the intestines the open hand is passed over the abdomen with a rotary movement. In the localized pain of appendicitis the open hand is held over the affected area with the fingers lightly flexed. In peritonitis the tips of the fingers are used but they touch the surface very gently and cautiously. Local pains resulting from visceral disease or colic are indicated by less guarded gestures; radiating pains by a repeated sweep of the hand in the same direction; distention pains and colic by a firm pressure upon the abdomen; neuralgic pains by repeated firm pressing movements of the hand in the direction of the involved nerve. The lightning pains of tabes are often indicated by a quick sweep of the tips of the fingers along the limb.

The shrinking of the whole body or of a member from an object capable of causing or increasing pain is a characteristic gesture; so also is the limping gait in painful conditions of a lower extremity. For diagnostic purposes it is important to bear in mind the fact that limping is frequently due to restricted movement not necessarily accompanied by pain. Very characteristic are the attitudes in certain painful affections: retraction of the head in meningitis, the shallow breathing and flexion of the trunk toward the affected side in plastic pleurisy, the strong bending forward in colic, the rigid trunk and flexed thighs in peritonitis, the semiflexion and immobilization of inflamed joints.

Sudden immobility of the whole body is diagnostic of angina pectoris.

The facies of pain constitutes a most important objective manifestation, whether it be the contorted, dusky pale face of sudden agony or the drawn and pallid countenance of prolonged and repeated suffering. Severe pain, especially when paroxysmal, is frequently accompanied by dilatation of the pupils, rapid respiration, flushing or pallor, free sweating, increased arterial tension, and sensations of faintness. Inarticulate sounds and involuntary exclamations are familiar objective manifestations of sudden and intense pain.

Some of the objective manifestations of pain are involuntary and cannot be simulated; others may, with or without the conscious intention to deceive, be feigned or exaggerated. By the verbal description we gain information as to the location, character, intensity, and duration of pain, and the patient's opinion as to its cause. The accounts are much modified by the temperament, power of expression, and general experience of the sufferer.

Not only the ability to express the subjective sensation of pain varies greatly but also the susceptibility. There are on the one hand individuals in whom the pain sense is but slightly developed; on the other those in whom it is present to an abnormal and excessive degree.

There are racial differences in the susceptibility to pain and the mode of expressing painful sensations. The Latin races manifest a greater susceptibility to pain than the Anglo-Saxons. Oriental apathy is proverbial. On the other hand Hebrews appear to have a peculiar susceptibility to pain.

The individual susceptibility is much modified by temperament. Phlegmatic persons suffer less and show such sufferings as they experience much less forcibly than those of sanguine or nervous temperament. The neurotic individual suffers in proportion to the instability of his nervous organization. The pains of hypochondria and hysteria are probably of central origin. They are of irregular distribution, inconstant, and occur independently of the recognized causes of pain. They are probably none the less real. They diminish in intensity or disappear when the patient's attention is diverted from them and are aggravated by suggestion. The painful aura of epilepsy is also of central origin. Fright, expectation, and dread intensify painful impressions.

Somewhat analogous to the influence of temperament is that of the power of expression. The manifestations of pain are sometimes much less marked in the rude and uneducated than those in the higher walks of life. Apathy is a striking mental condition in hospital patients.

Experience is not less important. Habitual exposure to hardship benumbs the pain sense. On the other hand a life of refinement and luxury exalts it. Prolonged suffering or frequent recurrence of painful sensations augments the sensibility and each recurrence becomes less endurable. There is a popular phrase to the effect that the patient is *worn out with pain*

The manifestations of painful sensations are much influenced by circumstance and motive. Consciousness of pain is greatly diminished during intense religious or other excitement and upon the field of battle. When the excitement subsides pain asserts itself. The repression of the manifestations of pain by religious fanatics, the stoicism of captives under torture, and the fortitude with which the brave endure suffering set common experience at naught and emphasize the purely subjective nature of pain as a symptom. Not uncommonly patients understate their sufferings either from motives of pride or reserve or in order to avoid operation or treatment. On the other hand patients frequently appear to overstate their sufferings in order to secure sympathy or for other obvious motives. Women are more susceptible to pain than men and according to circumstances manifest it with greater intensity or endure it with greater fortitude.

The patient's description of his sufferings, the character of the concomitant phenomena, and the presence of an obvious cause will enable the physician to form an estimate of the significance of pain. In young children, in certain forms of insanity, and under other circumstances in which patients are unable to describe their sensations the objective manifestations of pain are of diagnostic value in determining its seat and intensity. The physician must be on his guard in any particular case against under-estimating the importance of pain or being deceived by its unintentional or purposeful exaggeration.

Varieties of Pain.—Pain in the broadest sense may be considered as parenchymatous or neuralgic. In the former the terminal sensory filaments are irritated; in the latter the nerve-trunks, the sensory roots, or the sensory centres. Parenchymatous pain is as a rule less intense than neuralgic pain and the spontaneous remissions are less marked. In the former the pain in the whole affected region is increased by pressure, while in the latter, though in some cases the entire region is tender under pressure, the general rule is that the tenderness is localized to the course of the nerve-trunk, especially when it is superficial or overlies a bone or makes its exit through dense fasciæ—so-called *tender points*. An example of parenchymatous pain is that which occurs in visceral diseases and the diffuse headaches; examples of neuralgic pains are the various actual neuralgias which occur as primary affections in persons otherwise in fair health, in the cachectic and broken-down, and as secondary affections in gout, syphilis, and diabetes, and the lightning pains of spinal disease, especially tabes. The pains originating from suggestion and autosuggestion and many of the forms of hysterical pain are of central origin and may be regarded as parenchymatous.

Pain has been described as acute, sharp, lancinating, dull, throbbing, grinding, shooting, burning, chilling, shivering, boring, creepy, griping or colicky, itching and formicating. These descriptive adjectives indicate not so much distinct variations in the quality of pain as the simultaneous recognition of other associated sensations; hence, the descriptions of pain are often complex or picturesque in proportion to the vividness of the patient's imagination and his powers of expression.

(a) ACUTE PAIN—SHARP, LANCINATING, OR STABBING.—These adjectives are employed to describe the pain which attends acute inflammations of serous membranes, as pleurisy, pericarditis, and peritonitis; the pains of acute arthritis; acute neuralgias; the painful forms of neuritis; acute phlegmonous inflammation, and the pains of thoracic aneurism. The lightning pains of tabes belong to this group and are characterized by their suddenness, brief duration, and intensity. They are sometimes spoken of as shooting pains.

(b) DULL PAIN is symptomatic of inflammation of the mucous membranes and the viscera. It occurs also in chronic inflammations.

(c) THROBBING OR PULSATING PAIN is encountered in acute superficial phlegmonous inflammations. This is the pain of whitlow—paronychia.

(d) GRINDING, BURNING, OR GNAWING are adjectives used to describe the pain which occurs in diseases of the bones and periosteum, in aneurism of the thoracic and abdominal aorta, in carcinoma of the viscera and of the breast. Pain of this kind sometimes occurs in lithæmic conditions and in the later stages of acute gout. The localized neuralgic pain in the head, known as clavus, and the persistent local pains which occur in some forms of tabes are described as boring.

(e) ACHING PAINS are not unlike the preceding. They are usually persistent and intense and, when severe, throbbing. Aching is a term used to describe pains in the head, those resulting from dental caries and forms of neuritis and myalgia, especially lumbago—hence, *cephalalgia, odontalgia, rhachialgia.* The pains which occur in the initial period of acute infectious diseases, as, for example, variola, influenza, and dengue, and are referred to the bones and muscles, are of this character. They are frequently associated with painful sensations of chilling or shivering and, since they spread from one part to another, are often described as creeping.

(f) BURNING PAIN occurs in the superficial cutaneous lesions caused by intense heat or the action of the sun's rays, and caustic applications. It is characteristic of certain forms of neuritis. Circumscribed neuralgias are frequently associated with the sensation of burning pain—*causalgia.*

(g) ITCHING PAIN occurs in irritable states of the mucous membranes, such as attend certain forms of conjunctivitis, some acute diseases of the upper air-passages, and hay fever and some forms of inflamed hemorrhoids. Formication is a term used to describe a sensation like that of ants or other insects crawling over the skin. It is occasionally painful.

(h) GRIPING OR COLICKY PAINS are those which attend the overaction of the muscular walls of tubal structures. Flatulent or other distention of the stomach or intestines induces pain of this kind—popularly gripes or belly-ache. The pains upon overaction of the muscular wall of the intestines caused by indigestible food, cathartic drugs, irritant poisons, and certain infections, as those of cholera morbus and cholera Asiatica, are colicky. To this group belong also the intense paroxysmal pains which attend the passage of hepatic and renal calculi—*biliary colic; renal colic.* These pains are frequently spoken of as cramp, a term also applied to painful contraction of the skeletal muscles, as those of the calf, toes, fingers, the pains of tetanus and strychnine poisoning and those which occur in habitually over-used muscles in certain occupations—writer's cramp, piano-player's cramp.

(i) TENESMUS is the term used to describe the painful bearing-down or straining sensations which accompany expulsive efforts from the outlets of the pelvic organs under certain abnormal conditions, as urination when there is acute inflammation of the bladder, urethra, or prostate gland, or stricture; defecation in proctitis or inflamed piles or hydatid or other tumors compressing the rectum. The bearing-down pains of labor are tenesmic.

Pain is modified by physical and psychical influences. Among the former are pressure, mechanical irritation, movement, and rest.

Modifications by Physical Causes.—The pain which is caused by pressure and the increase of pain upon pressure are described as tenderness. This will be discussed later under a separate heading.

Mechanical irritation causes pain or aggravates it in inflammation and ulceration of mucous membranes, as in aphthous and other forms of stomatitis, angina tonsillaris, peptic ulcer and fissure of the anus, inflamed hemorrhoids, and in various lesions of the tegumentary structures. Even slight mechanical irritation of the normal mucous membrane of the orifices of the body causes pain, as the presence of a minute foreign body under the eyelid, the introduction of a probe into the nasal chambers, or the passing of an urethral bougie.

Movement aggravates the pain of wounds, fractures, and inflammations. The pain which attends acute inflammation of serous membranes is especially increased upon movement, as is to be observed upon full inspiration in pleurisy and upon flexion and extension of the thigh in peritonitis. Movement intensifies the pain of arthritis, hence the involuntary immobilization of the joints and the relief afforded by splints. Movement also greatly increases the pains of vertebral disease and neuritis. The pains of myalgia and of all acute inflammations involving the muscles are augmented by movement of the affected part. In many instances the pains of inflammatory conditions and of visceral disease are increased by the motion of the body in transportation.

Rest, upon the contrary, is commonly attended with remission of pain; functional rest, by its temporary disappearance, as in myalgia, the headache of eye-strain, headache from prolonged study, and the pain of gastric ulcer. The foregoing facts indicate the value of attitude, posture, and movement in determining the diagnostic significance of pain.

Cold and heat modify pain. Hot applications are usually soothing; cold applications only occasionally afford relief. The application of heat or cold to the spine may indicate the level of disease by local intensification of pain. Applications of heat or cold frequently enable the dentist to locate the offending tooth in diffuse pain involving the distribution of the dental branches of the fifth nerve.

Seasonal influences modify habitual tendencies to pain. The pains of chronic arthritis, gout, and neuralgia are worse in cold and damp weather, better when it is warm and dry. The influence of climate upon such chronic painful affections is similar; dry, equable, warm, inland climates being more favorable than those of the opposite characteristics.

Modifications by Psychical Causes.—Among the psychical influences which modify pain and its manifestations, intense emotion, excitement, pride, and fortitude have already been mentioned. Other influences of more importance in diagnosis are diversion, preoccupation, expectant attention, suggestion, and autosuggestion. They may be active under certain circumstances and to some extent in almost any kind of pain; but they are agencies of especial importance in neurotic persons and in those suffering from hysteria, neurasthenia, and hypochondriasis. Not only are the pains for which there are no obvious physical causes augmented or diminished, or made to disappear or shift to other parts, by purely psychical influences, but even those which attend actual injury and manifest disease may be greatly modified for a brief period of time. In the hypnotic state pre-existing pain may be made to disappear and definite pain aroused with readiness. It is evident that persons of great determination may inhibit the manifestation of pain under the stress of powerful motives. There are also rare individuals who appear to be able to inhibit the sensation of pain.

Time.—Pain in relation to time may be occasional, constant, persistent, intermittent, recurrent, or paroxysmal. Pain that continues for any length of time shows marked remissions and exacerbations. The remissions are due to functional exhaustion of the pain sense.

Distribution.—Pain may be (a) diffuse or general, or (b) circumscribed or local.

Diffuse pain is symptomatic of the stage of onset in the majority of the acute febrile infections. It varies in intensity from a mere sense of malaise or general soreness, as in enteric fever, to the severe aching of influenza, dengue, or variola. It occurs also in angina tonsillaris, particularly the lacunar form, and in trichiniasis. Diffuse pains attend certain stages of some chronic diseases, as syphilis, lithæmia, and saturnine and mercurial intoxication. They are sometimes described as vague and are often shifting. They are probably peripheral in origin and due to the action upon the nervous system of toxic substances in the blood.

Circumscribed or local pain occurs as a symptom in the greatest variety of morbid conditions. It is in fact the most common and most important of the subjective manifestations of disease. Its value in diagnosis depends largely upon the ability of the physician to estimate the accuracy of the verbal description, the spontaneity of the accompanying objective phenomena, the anatomical relationships of the pain itself, the underlying pathological process, and the importance of alleged or manifest causes. Pain, and in particular local pain, may be a danger signal, a sign post, a gauge of the progress or extension of disease, a counter check to objective phenomena, or it may be to the unwary or ill-informed physician a delusion and a snare.

Feigned Pain.—The simulation of pain is common enough in malingering, neurasthenia, and hysteria. The motives of malingering are innumerable. In neurasthenia and hysteria they usually consist of a morbid craving for sympathy. The detection of simulated pain is in some cases attended with difficulties that are insurmountable. In malingering the simulation of pain is usually overdone. The distribution of the pain does not conform to known anatomical rules. Suggestion is of importance. The objective phenomena commonly associated with [illegible] or incongruous.

To properly estimate the value of pain in an obscure case it is sometimes desirable to have the patient under the close observation of an experienced nurse or attendant or in a hospital for some days.

Significance of Pain.—In general terms local pain is symptomatic of disease of the part to which it is referred. Organic headache, angina tonsillaris, the pain in the side in pleurisy, in the abdomen in peritonitis, in the joints in arthritis, and various forms of pain due to traumatism, are examples of the relationship of local pain to local disease. As regards the anatomical structure involved pain may be tegumentary, muscular, osseous, visceral, or neural. Very commonly the pain is also limited to the region or organ affected. But there are numerous exceptions to these statements, and we find local pain frequently symptomatic of a pathological process in a distant part, or local disease causing pain in an extended area. The recognition of these facts is of cardinal importance in estimating the value of local pain in diagnosis.

Referred Pain.—A familiar example is the intense pain over the supraorbital notch sometimes felt upon eating an ice. The organ affected is probably the stomach, the location of the pain being determined by the association of sensory nerves from that organ with the trifacial. Very curious instances of referred pain have been reported—a case in which rubbing the forearm caused pain in the chest; another in which rubbing or pinching a mole on the leg was attended by sharp pain in the chin.

Referred pains manifest themselves in:

1. Symmetrical Areas.—A case is reported by Mitchell in which a shell-wound of the right foot at once gave rise to burning pain in both feet. A shell-wound of the left thigh caused an immediate reference of pain to the same area on both sides, so that the patient supposed he was shot through both thighs. Again, an injury to the median and ulnar nerves was attended by pain in the opposite hand.

Allocheiria is the name given to the phenomenon of pain or other sensation referred to a symmetrical area. It has been observed in tabes and in postdiphtheritic neuritis.

2. Functionally Associated Organs.—Pain in the mammae is common in congestion of the pelvic organs and dysmenorrhea; pain in the glans penis or testicle in renal colic; diffuse pain in the abdomen in the early stage of appendicitis.

3. Segmental Areas.—Visceral disease is frequently attended by pain and tenderness referred to areas corresponding to the nerve supply of a given spinal segment. The affected organs receive their sensory nerve-fibres from the same segment of the spinal cord from which arise the fibres of the sensory areas to which the pain is referred. In the words of Head: "As the sensory and localizing power of the surface of the body is enormously in excess of that of the surface of the viscera, an error of judgment occurs, the diffuse area being accepted by consciousness and the pain referred to the surface of the body instead of to the organ actually affected." Hence the pain in intestinal colic is referred to the whole abdomen; that of hepatic colic to the epigastric zone; and that of renal colic to the lumbar region. So also pain in the heart, lungs, liver, and stomach may be referred to areas innervated by the cranial nerves and nerves given off from the

cervical plexus, and the pain in disease of the pelvic organs is very commonly referred to the back. A striking example of this kind of pain-reference is seen in the pain and exquisite tenderness of the right hypochondrium sometimes encountered in diaphragmatic pleurisy.

4. LONGITUDINALLY RELATED AREAS.—Pain arising in the course of a nerve may be referred to its terminal distribution. The pain in the stump, which appears to be in the amputated foot, is a familiar example. The lightning pains of tabes, the thigh pains in malignant disease of the rectum and in psoas abscess, and the pain around the umbilicus in vertebral caries are further illustrations. Sometimes the areas are not so directly related, as in the knee pain in hip-joint disease, the shoulder pain in disease of the liver, and the pain in the distribution of the ulnar nerve in angina pectoris.

Peripheral pain may be an early and suggestive symptom in organic disease of the brain and spinal cord. In meningitis the pains in the back and limbs may be very severe. The joints are frequently the seat of pain, which may be more or less constant or lancinating and paroxysmal.

Painful Crises.—Severe and prolonged attacks of pain, associated with functional disturbances and wholly independent of local organic disease, occur in some cases of locomotor ataxia and are known as the tabetic crises. They are (a) cardiac—intense precordial pain accompanied by a feeling of oppression and rapid and irregular pulse; (b) gastric, the most common—sudden severe pain in the epigastrium, with vomiting, rapid and irregular pulse, sometimes symptoms of collapse; there may be vomiting without pain or pain without vomiting; (c) laryngeal, which is comparatively rare—pain in the larynx with paroxysmal cough, inspiratory stridor and sensations of choking; (d) pharyngeal, also rare—painful acts of deglutition following one another at short intervals and lasting from some minutes to half an hour. Intestinal, rectal, urinary, and genital crises have also been described. Suddenness of onset, intensity, paroxysmal character, and abrupt termination are characteristic of these attacks. The absence of lesions in the affected viscera either during the attacks or in the intervals between them is of diagnostic importance. Errors in diagnosis are common.

Localization of Pain.

Superficial pains are mostly symptomatic of diseases of the underlying parts, but they may be referred.

Deep-seated pain attends inflammatory and ulcerative diseases of the viscera, mediastinal tumor, aortic aneurism, visceral cancer, and disease of the bones.

Pain may be unilateral or bilateral. The former usually attends morbid processes confined to the affected side; the latter those involving both sides or of central origin. This rule is far from being absolute. The pain caused by floating kidney is occasionally referred to the opposite side of the abdomen.

The more important local pains and their diagnostic significance are now to be considered.

Pain in the Head.—(a) **Headache** is a term used to designate pain referred to various regions of the head. It may be paroxysmal or continuous. The term *cephalalgia* was applied by the ancients to slight, limited, or transitory headaches; the term *cephalæa* to severe, deep-seated, and chronic pains in the head. Headache is in many cases a symptom of such importance and prominence that it overshadows all others and lends to the clinical picture its most characteristic feature, often at first sight its only obvious feature. Headache is a symptom very often significant when other phenomena are obscure. It thus acquires a high degree of diagnostic importance.

ORGANIC AND FUNCTIONAL HEADACHES.—Headaches due to lesions of the skull or intracranial disease are organic; those due to other causes are functional. In general terms headache is the manifestation of the irritation of sensory nerve-fibres caused by derangement of pressure or tension, inflammation, toxæmia, and reflex disturbances. It is probable that the meninges are chiefly concerned in the causation of headache. The substance of the brain in the lower animals does not respond to direct irritation by the manifestations of pain; and lesions of cerebral tissue not directly or indirectly involving the membranes may exist without causing headache. The meninges and especially the dura, on the other hand, are directly or indirectly implicated in those pathological processes which give rise to headache. The sensory nerve supply of the dura in the anterior three-fourths of its extent, that of the falx and probably that of the tentorium are derived from the trigeminus, while the dura mater of the posterior fossa is supplied with sensory fibres from the vagus. The trigeminus is the nerve of sensation to the scalp as far back as the vertex, while the posterior branches of the upper four cervical nerves supply the muscles and the skin of the back of the neck and the occiput. Sometimes headache is referred to the scalp; usually the pain is deep-seated and intracranial. In rare cases superficial headaches are essentially myalgic, the pathological condition involving the occipitofrontal, temporal, or sternomastoid muscles.

The following clinical considerations in regard to headache are important:

DISTRIBUTION OF HEADACHE.—This pain is usually bilateral. It may be frontal, occipital, parietal, and temporal, vertical or diffuse. The area most commonly involved is frontal, next in order of frequency is diffuse headache, then follow in the order named vertical, occipital, and temporal. Headache often shifts from one part of the head to another and is not always confined to regions limited by anatomical boundaries.

VARIETIES OF HEADACHE.—Headache, according to the character of the pain, may be: 1. Pulsating or throbbing. Headache of this kind is symptomatic of circulatory disturbances; it is often diffuse. 2. Dull, heavy. This is the headache due to toxæmia; it is usually frontal, sometimes occipital. 3. Binding or constrictive; the sensation is often described as that of a tight band around the head; the focus of intensity is referred to the parietal regions. This is the headache of hysteria and neurasthenia. 4. Burning or sore; forms of headache diagnostic of anæmia, rheumatism, and lithæmia. 5. Boring or sharp. These headaches are symptomatic of hysteria and allied conditions; they are usually localized; one form is known as "clavus"—the sensation as if a nail were being driven into the head.

Headache may be transient or persistent. In the latter case there may be exacerbations and remissions, or occasional intermissions which may last for days or weeks. There may be slight, continuous headache with exacerbations of varying intensity. Headache of this kind is symptomatic of forms of reflex irritation, especially those arising from defects of accommodation. Persistency is characteristic of organic headaches such as occur in cerebral tumor or abscess or pachymeningitis, or those which result from excesses in tobacco or alcohol, syphilis, and uræmia. The headaches which occur after sunstroke are persistent, with brief and irregular periods of remission.

The headache following cerebral concussion is severe and protracted. It may be circumscribed and limited to a region corresponding to the seat of the injury or to the opposite side of the head. It is commonly associated with tenderness on light percussion. The headache following injury may be, on the other hand, diffuse. It is apt to be associated with vertigo, lassitude, and indisposition to mental effort.

SIGNIFICANCE OF HEADACHE. — *Congestion.* — Headache may result from mechanical interference with the return of venous blood from the head. When produced by improper clothing it is slight and ceases upon removal of the cause; when due to venous obstruction from the pressure of tumors it is not usually severe. The headache caused by violent paroxysmal or frequently repeated cough is congestive.

Hyperæmia.—Headache is symptomatic of active cerebral hyperæmia such as follows excessive and prolonged mental effort, and results from the action of vasodilator drugs, as alcohol and the nitrites. This form of headache occurs in the initial stage of acute meningitis. The headache of cerebral hyperæmia, whether passive or active, is usually frontal or diffuse, often pulsating or throbbing.

Anæmia.—Headache occurs in the anæmia due to blood loss or other cause. It is a common symptom in chlorosis. Anæmic headache is commonly severe, usually frontal or diffuse, often attended by sensations of pressure and not rarely associated with vertigo and tinnitus aurium. The headache of anæmia is intensified by effort.

Inflammation.—Headache is characteristic of all forms of cerebral meningitis, both acute and chronic. It is usually at first localized, a fact of importance in the diagnosis of meningitis due to mastoid or ethmoid disease or disease or injury of the cranial bones. It, however, rapidly becomes diffuse. Meningeal headache is usually continuous with exacerbations of great severity. Headache in exceptional cases is absent in the early stages of gradually developing leptomeningitis. Sudden intense headache with painful rigidity of the muscles of the back of the neck and vomiting are early symptoms of epidemic cerebrospinal fever. Intense paroxysmal headache is a symptom of tuberculous meningitis. The headache of pachymeningitis is local at first, but later becomes generalized. Severe frontal headache, usually unilateral, is symptomatic of disease of the frontal sinuses.

Infection.—Headache is a common manifestation of infection. This headache is usually frontal, it may be occipital or general, is often neuralgic or superficial, soon becoming dull, deep-seated, and severe. Headache is an important symptom of the stage of onset of the acute febrile infections. It is early and severe in typhus and associated with pain in the back and limbs. After a time it is followed by stupor. It is a constant symptom in the early stages of enteric fever but subsides spontaneously during the second week of the disease. It occurs at the onset of relapsing fever and persists until the crisis, when it commonly ceases altogether. The headache of influenza is diffuse with points of intensity in the region of the frontal sinuses and behind the eyeballs. It may be a troublesome sequel. Intense headache characterizes the period of invasion of smallpox and is usually accompanied by excruciating pains in the back and joints.

Headache occurs in early syphilis. The headaches of late syphilis are usually symptomatic of arterial changes, gummata, or meningitis. Headache is common in hereditary syphilis. Paroxysmal headache is symptomatic of malaria. It occurs in the hot stage of the paroxysm. It is persistent and intense in estivo-autumnal fever. Periodical headache may be the chief symptom in estivo-autumnal infection.

Toxæmia.—Some intractable headaches are symptomatic of chronic uræmia. They are frontal or temporal, intense, usually continuous, with irregular exacerbations. Headaches of the same general character occur in diabetes and in those suffering from the gouty diathesis. To this group we may refer the headaches of chronic lead poisoning, those occurring in gastrohepatic derangements, and constipation. These headaches are intensified by alcoholic beverages and relieved by free purgation. Certain drugs cause headache. Full doses of quinine or the salicylates produce headache and tinnitus aurium. Opium causes distressing headache with floating sensations, nausea, and vomiting. All these symptoms are increased when the patient assumes the upright posture. Tense, vertiginous headache follows the administration of the nitrites in full doses. Headache is a significant symptom in chronic poisoning by lead, tobacco, alcohol, opium, and chloral. In the case of lead and of alcohol arterio-capillary sclerosis is coöperative. Opium and chloral headaches are often abstinence symptoms, occurring upon the withdrawal of the drug. Intense headache not unlike that of migraine frequently follows excesses in alcohol—the *acute alcoholism of debauch.*

Cerebral Abscess.—Headache is often very severe and persistent in cerebral abscess. It is apt to be associated with vertigo and pronounced mental dulness and irritation. Vomiting is common but not constant. Chronic brain abscess may present no other symptom than headache, vertigo, mental dulness, irritability, and physical depression. The pain is usually related to the region of the lesion; in ear disease it is referred to the parietal or the occipital region of the affected side. In abscess following disease of the nasal or ethmoid bones the pain is referred to the brow. In abscess from traumatism the focus of pain is located in the region of the injury.

Tumor.—Headache may be said to be a constant symptom of brain tumor. Its frequency and intensity vary according to the location of the new growth, the rapidity of its development, and in some degree to its character. Headache is more persistent and severe in cerebellar than in cerebral tumors; in those of the cerebral hemispheres than in those of the base and in those directly implicating the meninges. It is more prominent in tumors of rapid than in those of slow growth, without regard to the nature of the pathological process. In general terms the nature of the tumor formation has no direct relation to the intensity of the headache, the exception to this rule being that gliomata are less painful than other forms of coarse intracranial new growths. Headache in brain tumor is sometimes dull and boring, sometimes lancinating, usually intense, often agonizing. It is commonly continuous with periods of intensification, but sometimes recurs with a regular periodicity suggestive of malaria. The fact that it is commonly worse at night has some diagnostic value. The focus of the headache in cerebral tumor may be in the region involved, in the brow or in the occiput, or the pain may be diffuse. The headache of brain tumor may be localized when of moderate degree, diffuse during periods of intensification. Light percussion with the finger-tips may elicit tenderness in a region corresponding to the tumor. The headache of pachymeningitis interna hæmorrhagica is usually at first referred to the vertex; later it becomes generalized.

Aneurism.—Headache, either continuous or paroxysmal, is the most common symptom of intracranial aneurism affecting the larger arteries at the base. The location of the headache has in general no definite relation to the position of the aneurism, though aneurisms of the basilar artery usually occasion occipital headache. Headache occurs in caries of the bones of the skull.

Neurotic States.—Headache is a very common symptom in neuropathic conditions. In neurasthenia it is frontal, occipital, or diffuse; it is apt to be continuous and is aggravated by mental application and physical effort. Its intensity is moderate and it is attended by sensations of pressure in the head, aching in the back of the neck, and spinal pains. Headache is very common in the interparoxysmal periods of hysteria. It is often referred to the vertex and may be severe and persistent. Headache is common in emotional and precocious children. It is frequently associated with brow pains, pains in the back of the neck, and intolerance of bright light. Headaches of this kind are allied to the headaches of hysteria. Headache frequently enters into the symptom-complex of the epileptic paroxysm. It may precede or follow the convulsive attack. In the latter case it is associated with drowsiness and hebetude. Headache is common in petit mal. In many cases of epilepsy it constitutes an important symptom in the interparoxysmal state.

Reflex Headache.—This form is often troublesome and persistent. This is sometimes the case when the direct symptoms of the local disease are slight or absent. Errors of refraction constitute a common cause of reflex headache. The pain is usually frontal, sometimes temporal, often occipital. The patient is frequently unaware of any defect in visual accommodation. The headache is usually aggravated by close or prolonged use of the eyes. Reflex headache may occur as a symptom in chronic nasal disease especially in affections of the accessory sinuses. It usually involves the temporal region or the vertex. It is associated with sensitiveness of the nasal wall of the orbit and hyperæsthetic areas on the mucous membrane of the middle turbinate bone. Headache is an important symptom of adenoid vegetations in the nasopharynx. It constitutes one of the forms included under such terms as "school headaches," "headaches of the period of growth," and the like. Associated symptoms are mouth-breathing, mental dulness, and irritability. Carious teeth and exposure of the pulp not only cause toothache but occasionally also cause reflex headache. Disease of the auditory apparatus may be the unsuspected cause of persistent headache.

The headache of acute indigestion and gastro-intestinal catarrh is probably rather toxæmic than reflex.

The importance of headache as a manifestation of disease of the sexual organs is probably over-estimated; yet this symptom is very common in those of both sexes who suffer from actual disease of the reproductive apparatus or are the victims of psychical processes concerning such diseases. Very often these headaches are due rather to the attendant neuropathic condition than to reflex irritation.

ASSOCIATED SYMPTOMS.—Vertigo, nausea, vomiting, drowsiness, irritability, and hebetude are associated with headache with such frequency as to indicate a common causation. These symptoms are as a rule less constant and less severe in symptomatic than in organic headaches. Vertigo is a frequent attendant upon headache due to gastro-intestinal disorder; nausea and vomiting in acute toxæmia; somnolence in malaria, anæmia, and syphilis. In organic headaches the presence of this group of symptoms and their persistence are important and suggestive.

Headache is essentially a symptom and a careful examination and inquiry will reveal some general or local cause. Headache is to be differentiated from migraine—a paroxysmal neurosis.

Neuralgia differs from headache in the following points: The pain involves the trunk or branches of the nerve rather than its peripheral distribution. It is unilateral, localized, sharp, paroxysmal, and there are present the characteristic tender points of Valleix. Neuralgia affecting the first branch of the fifth nerve is sometimes attended with suffusion of the eye and œdema of the lids.

FUNCTIONAL AND ORGANIC HEADACHES.—The differential diagnosis between functional and organic headaches is of fundamental importance. Organic headache is commonly persistent, varying from time to time in intensity, sometimes undergoing violent exacerbations but rarely wholly absent. It often interferes with sleep. It is aggravated by mental or physical effort, by excitement, alcohol, and all conditions that increase intracranial hyperæmia. It yields less readily than functional headache to symptomatic treatment. It tends to progressively increase in severity and is in many cases ultimately replaced by the stupor, drowsiness or coma of the terminal stage of the disease. Associated symptoms, such as vomiting, vertigo, hebetude, and irritability, are of diagnostic importance, and double optic neuritis, convulsions, and localizing symptoms, as monospasm, cranial nerve paralysis, cerebellar titubation, forced movement, and hemianopsia, render the differential diagnosis between organic headaches and functional headaches in most cases an easy matter.

(b) **Pains in the Scalp.**—Myalgic pains have been already spoken of. They are usually frontal or occipital, increased by voluntary movements of the scalp and by pressure. Various affections of the skin are attended by itching and burning pains of moderate degree. Local dermatitis attended with pain sometimes results from the injudicious application of hair washes containing excess of cantharides and sometimes from the action of pediculi; also from burns and scalds, from erysipelas, and from traumatism.

Diffuse wandering pains are often experienced in various parts of the scalp and are associated with tenderness of the skin. These pains are not confined to the ramification of nerve-trunks and cannot be strictly regarded as neuralgic, but they very frequently alternate with true neuralgia. A patient under my observation compared these pains to sheet lightning.

(*c*) **Pains in the Face.**—The most important is trigeminal or facial neuralgia, known also as *tic douloureux* and *prosopalgia.* Neuralgia of the fifth nerve is much more frequent than all other forms of neuralgia. The pain is spontaneous, paroxysmal, and unilateral. Neuralgic pains involving the ophthalmic division usually affect the supra-orbital branch and are known as brow ague or supra-orbital neuralgia. The pain radiates over the front of the head from the supra-orbital notch. It may be felt in the eyelid or the eyeball or at the side of the nose. Tender points are found at or above the supra-orbital notch, in the upper eyelid, and on the side of the nose.

The neuralgic pain may be referred to the eyeball itself. It may occur spontaneously or as the result of over-use of the eyes. It is attended with dimness of vision and lachrymation and may occur alone or in connection with other neuralgic pain in the region of the fifth.

Neuralgia of the superior maxillary division of the fifth nerve is referred to the region between the orbit and the mouth and the side of the nose. Areas of special intensity are upon the side of the nose, over the prominent part of the upper jaw and along the gum. Paroxysms are frequently induced by the use of the tooth-brush. When the inferior maxillary division is involved a focus of pain is frequently found just in front of the ear, or in the temple or opposite the point of emergence of the nerve from the foramen, or in the region of the parietal eminence, and sometimes a point at the side of the tongue.

In intense paroxysms of trifacial neuralgia the whole side of the face and brow is involved and there is reflex facial spasm—tic convulsif. Supra-orbital neuralgias are occasionally attended with vasomotor disturbance. In other instances a herpetic eruption occurs which is probably the manifestation of an actual neuritis. Intractable neuralgias of the fifth nerve occurring late in life are known as degenerative neuralgias and are associated with changes in the ganglion of Gasser.

Severe pains in the distribution of the fifth nerve accompany cancer of the tongue, lingual ulcer, and caries of the inferior maxilla. Caries of the teeth and exposure of the pulp may give rise to pain referred to the ear.

(d) **Pain in the Eye.**—Inflammatory diseases of the eye cause local pain. In acute conjunctivitis there is pain in the eyelids, accompanied by photophobia and lachrymation; in iritis pain in the eyeball and intense supra-orbital pain, which may radiate in the distribution of the ophthalmic division.

The pain of glaucoma involves the distribution of the trigeminus, having its focus of intensity in the eyeball or at the supra-orbital notch. In the acute cases it is agonizing and associated with depression, pallor, nausea, and vomiting. In the chronic form it may be subacute with paroxysms of great severity. As the disease begins with great frequency on one side there is a misleading resemblance to migraine. Increase of the intra-ocular tension, irregular or dilated pupil, with inactive iris, haziness, anæsthesia of the cornea, and various visual derangements are suggestive symptoms.

(e) **Pain in the Ear.**—The pain of acute middle-ear disease is intense, throbbing, increased by pressure in front of the tragus and by gentle traction of the ear. It is subject to exacerbations and remissions and often radiates to the side of the face. Upon spontaneous or surgical perforation of the tympanic membrane the distressing feeling of tension is followed by immediate relief. Tinnitus is a common accompaniment. Pain referred to the ear and the side of the head is a prominent symptom in mastoid disease. It is accompanied by tenderness upon pressure and localized œdema.

(f) **Pain Referred to the Mouth.**—Pain is a symptom of various forms of stomatitis. It is intense in aphthous stomatitis, a very trifling affection, and often wholly absent in cancrum oris, one of the gravest of diseases. In mucous patches and syphilitic ulceration pain is less conspicuous than in tuberculous ulceration. In carcinomata pain is a persistent and distressing symptom. In inflammatory and ulcerative conditions of the pharynx pain is a prominent symptom. It is excited by mechanical irritation and by the contraction of the pharyngeal muscles in deglutition. Pain is not a prominent symptom in epidemic parotitis and parotid bubo. It is excited, however, by the movements of the parts involved and accompanied by great tenderness upon pressure.

(g) **Sinus Pain.**—Pain is a prominent symptom in disease of the accessory sinuses of the nose, especially in those cases in which there is an obstruction to the outlet. Under these circumstances the pain may be extremely severe and accompanied by marked systemic disturbance, as fever, chilliness, headache, and malaise. The sinuses usually involved are the antrum of Highmore and the frontal sinuses. Free discharge of mucus or pus is usually followed by immediate relief, but there are chronic forms in which the pain is apt to be of a dull character and constant, with exacerbations in damp weather and after exposure to cold. The diagnosis of antrum disease may be confirmed by transillumination with an electric light.

Pain in the Body.—(a) **Pain in the Back—Backache; Rhachialgia.**—Pain may occur in any part of the back. It is more common in the lumbar and sacral regions than elsewhere. Pain in the back of the neck extending between the shoulder-blades is a common symptom in neurasthenia and hysteria.

Acute pain in the small of the back attends the period of onset of many of the infectious febrile diseases, especially influenza, dengue, variola, and cerebrospinal fever. It occurs also in angina tonsillaris and acute nephritis. Acute pain in the back, much aggravated upon movements of extension, as in rising after lacing one's shoes, is characteristic of lumbago. Unilateral, deep-seated lumbar pain of great severity is symptomatic of renal colic. Persistent pain of this kind attends renal calculus. This pain is aggravated by pressure over the kidney or sudden jarring of the body. Pain in the back is often present in floating kidney. Sacral pains are symptomatic of disease of the pelvic organs, especially uterine flexions and displacements, ovarian disease, disease of the colon and rectum, hemorrhoids, and urethral stricture. Many of the pains in the lower part of the back are myalgic. Pains of this kind result from occasional or habitual overwork of the muscles or from traumatism in the form of contusion or strain, or finally from exposure to cold or damp, especially in lithæmic individuals. The pain of myalgia is increased by movement, cold, and pressure; it is relieved by rest in the recumbent posture and by hot applications.

Pain in the spine occurs in disease of the vertebræ. Traumatism, syphilis, tuberculosis, and caries from pressure, as in aneurism of the aorta, are common causes. The pain is local and corresponds to the segment of the column involved. It is increased by sudden pressure upon the head or shoulders, by jarring, by the application of heat, cold, and faradism, and is relieved by the recumbent posture and in some cases by suspension and a properly applied spinal jacket. Rigidity results from muscular spasm in the earlier stages and from ankylosis in the later. Various deformities occur. Pain is present in that form of arthritis deformans which involves the vertebræ—*spondylitis deformans—spondylose rhizomélique.* There are associated nerve-root symptoms, as anæsthesia and muscular atrophy.

Pain attends various diseases of the spinal meninges. It is local and often intense. There are symptoms of irritation in the course of the nerves. The more common causes are hemorrhage into the spinal membranes and meningitis. Muscular spasm and rigidity are present.

Diseases of the cord are more apt to cause radiating and referred pains than pain in the spine itself. The latter is felt in the lumbar region; the former, as nerve-root irritation, as girdle pains, and in the lightning pains of tabes.

(b) **Pain in the Side.**—1. The pain may be symptomatic of injury or inflammation of the skin, as abrasion, contusion, local dermatitis, or furunculosis. The last is common in the axillary region. In rare instances phlegmon or subcutaneous extravasations of blood may be the cause of severe pain. An inspection of the parts is necessary in all cases.

2. Myalgic pains are not uncommon. Pleurodynia affects the muscles on one side, usually the intercostals, sometimes the pectorals and the serratus magnus. It is more common on the left than on the right side. It is especially distressing since the muscles are in constant use in respiration. The movements are restricted on the affected side, but deep breathing, coughing, and forced lateral movements increase the pain. Tenderness is present often in a limited area. This affection may suggest intercostal neuralgia, from which it is to be distinguished by the more circumscribed area involved, the paroxysmal character of neuralgic pain, and the well-defined tender points. It is sometimes mistaken for pleurisy, but the absence of friction sounds is of diagnostic importance. Violent spasmodic flexion to one side is an occasional though rare manifestation of tetanus and is attended with great pain in the affected muscles. Side pains referable to the muscles are observed in some cases of trichiniasis.

3. Pains due to injury or disease of the bones may be referred to the side. Fracture of the ribs, periostitis, osteosarcoma, rickets, and some cases of osteitis deformans are to be considered. The diagnosis demands a careful examination of the area involved by inspection, palpation, auscultation, and in obscure cases by the Röntgen rays.

4. The pain of plastic pleurisy is referred to the inframammary region or the side. It is sharp or stabbing,—the stitch in the side,—increased on deep breathing and accompanied by friction sounds, in some cases friction fremitus and a dry cough. It may occur in previously healthy individuals, or be accompanied by slight fever and presently disappear; it is a secondary process in croupous pneumonia and develops during cancer, abscess, and gangrene when the surface of the lung is involved. It is a very common phenomenon in tuberculosis of the lungs and may be basic or apical.

5. Pain in the side may be due to visceral disease. Sudden tension of the spleen, as often occurs in boys after running, is accompanied by intense pain in the infra-axillary region of the left side. Heavy, dull, dragging pains are symptomatic of the splenic tumor of leukæmia and the malarial cachexia—*ague cake*. Renal colic is characterized by an extension of the pain from the lumbar region to the affected side and thence downward toward the groin. In biliary colic the pain frequently extends to the right side of the chest. A dull heavy pain in the side sometimes attends upward pressure upon the diaphragm such as occurs in an overloaded stomach or distended colon, rapidly developing ascites, or an enormous abdominal tumor. Pain, paroxysmal in character but not extremely intense, occurs in the early stage of some cases of pyelitis. Intense pain in the lumbar region, aggravated by pressure, is a symptom of perinephritic abscess. It is often referred to the hip-joint or the adjacent region or the inner aspect of the thigh. This pain is attended with fixation of the thigh, which is flexed to relax the psoas muscle, and the patient in walking stoops and throws his weight upon the sound side. The pain of hepatic abscess is usually referred to the back or shoulders; it may be most severe in the right hypochondrium. A duller, dragging pain is felt in the right side when the patient turns upon the left. The pain of angina pectoris is occasionally referred to the left side—fifth, sixth and seventh and even eighth and ninth dorsal areas.

6. Pain in the side is very often the manifestation of disease of the nerves themselves. Neuralgia may be the result of nutritional changes in the sensory nerve-roots, the course of the nerve, or its peripheral distribution. Intercostal neuralgia is very common. Women are more liable than men; adults far more liable than children. The left side is more frequently involved than the right. Neuropathic individuals especially suffer. Intercostal neuralgia is encountered in anæmic conditions, general malnutrition, gout, lead poisoning, malaria, cachexia, and chronic nephritis. The attack may follow exposure to cold. The pain is paroxysmal and burning or lancinating and there are characteristic *points douloureux*. Trophic or vasomotor phenomena may occur, as local œdema or erythema. The posterior branches of the lumbar plexus may be involved with pain in advance of the crest of the ilium extending along the inguinal canal and spermatic cord to the scrotum—irritable testis—or the labium majus. The pain of herpes zoster is intense and often persistent. It corresponds to the distribution of the eruption. The pain in caries of the vertebræ and aneurism of the descending aorta is referred to the distribution of the intercostal nerves.

(c) **Pain in the Chest and Abdomen.**—1. The skin may be the seat of pain in inflammatory diseases, burns, severe eruptions, and herpes zoster. Painful burns sometimes result from the unguarded use of sinapisms or hot-water bags. An inspection of the part is necessary.

2. Myalgia of the abdominal muscles may result from continuous cough. The epigastric pain in children suffering from measles is due to the cough. Muscular pain attends tetanus and some cases of strychnia poisoning. Trichiniasis is to be considered.

3. Periostitis and necrosis of the sternum, costal cartilages, and ribs cause pain in the anterior wall of the thorax. Resorption and ulceration from aneurism, malignant disease, syphilis, and enteric fever are common causes of painful lesions in these structures. Contusions, fractures, and dislocations cause pain.

4. Many visceral diseases cause pain in the chest and abdomen. It is an important sign of aneurism of the aorta. It is usually dull and persistent with frequent paroxysms in which it is sharp and lancinating. It is frequently severe when erosion of the chest wall or vertebræ is taking place. Anginose attacks may occur. Pain may be absent. Broadbent has spoken of aneurism of the ascending arch as the aneurism of physical signs; of the transverse arch as the aneurism of symptoms. Pain is the chief symptom in aneurism of the abdominal aorta. It is epigastric, paroxysmal, and radiates to the back and sides. Severe epigastric pain occurs in aneurism of the cœliac axis and the splenic artery. Pain may occur in mediastinal tumor, but it is much less common than in aneurism and does not have the radiating character so common in the latter affection. The pain of mediastinal abscess is substernal, throbbing, and usually associated with chilliness and profuse sweating. In plastic pericarditis pain may be absent. When present it is variable in intensity, usually mild, exceptionally severe, and frequently intensified by the pressure of the stethoscope. It is felt in the precordia or at the base of the ensiform cartilage. The pain of pericarditis with effusion is sharp and lancinating and intensified by pressure over the ensiform cartilage. It may be dull and dragging. Pain is not a symptom of endocarditis. It occurs in chronic valvular disease, especially aortic insufficiency, in which it is sometimes persistent and distressing. It is usually precordial, dull, and aching; sometimes sharp and radiating to the neck and down the left arm. Pain is much less common in aortic stenosis and is not a prominent symptom in mitral disease so long as compensation is maintained. Angina pectoris is characterized by paroxysmal, agonizing pain in the region of the heart, radiating into the neck and arms, especially into the ulnar distribution of the left arm, and often attended with the fear of impending death. Chest pain is common and severe in croupous pneumonia, pleurisy, and pulmonary abscess. It may occur in any part of the chest but is most common in the inframammary and mammary regions. In some cases of severe acute bronchitis substernal pain is a distressing symptom. Pain may be absent in diseases of the liver. It occurs in acute infectious cholecystitis and is paroxysmal and severe. It is referred to the region of the liver but may have its focus of intensity as low as the appendix or in the epigastrium. Intense paroxysmal pain is met with in cancer of the bile passages. Biliary colic is of common

occurrence in gall-stone disease. There is agonizing pain in the region of the gall-bladder, extending into the lower thoracic, epigastric, and upper abdominal zones and radiating to the right shoulder. Dull dragging pain with intense exacerbations associated with nausea or vomiting is encountered in so-called hypertrophic cirrhosis. Pain of a dull, aching character and radiating to the back and right shoulder occurs in hepatic abscess. Pain and uneasiness in the right hypochondrium are present in some cases of cancer of the liver. In pancreatic disease pain may be a prominent and suggestive symptom. It occurs in hemorrhage, acute pancreatitis, and abscess and is referred to the upper zone of the abdomen. It is intense and persistent with agonizing paroxysms. Painful colicky attacks with nausea and vomiting have been noted in pancreatic cysts and the passage of calculi has caused pancreatic colic. A dull pain under the sternum is present in inflammation and in spasm of the œsophagus. In cancer it may be persistent or only present upon attempts to swallow food. The pain of gastralgia is usually deeply seated; that of gastritis more superficial. Cardialgia is a term used to designate the uneasy and painful sensations in chronic gastritis, sometimes caused by the taking of food, sometimes present when the stomach is empty. Pain is a distinctive symptom of gastric ulcer. It is gnawing, burning, paroxysmal, induced by taking food, and referred to the epigastrium. It is also in some cases felt in the back at the level of the tenth dorsal vertebra. In peptic ulcer of the duodenum the pain is sometimes located in the right hypochondrium and may come on two or three hours after eating. Pain is an early symptom in cancer of the stomach and occurs at some period in almost all cases. It is usually epigastric but may be felt in the back or loins. It is usually burning or gnawing and rather continuous than paroxysmal, though it is aggravated after food. The gastric crises of tabes consist of intense paroxysmal pain in the stomach accompanied with vomiting and an excess of intensely acid gastric fluid. Intestinal diseases are accompanied by pain which may be colicky when the small intestine is involved and bearing-down when the colon is affected—the tormina and tenesmus of the older physicians. Abdominal pain of variable intensity occurs in acute and chronic catarrh, ileocolitis, proctitis, malignant disease of the intestines, obstruction, intussusception, ileus, and appendicitis. It is the first and most distinctive symptom of peritonitis. Inframammary pain upon the left side is a common symptom of fecal accumulations in the sigmoid flexure of the colon in women, and is relieved by free purgation. Renal colic may extend well into the abdomen upon the affected side. Pyelitis may cause suprapubic pain. Displaced kidney is usually a source of much discomfort; often of distressing pain. The paroxysmal pains known as *Dietl's crises* occur in this condition.

5. Lead colic, the referred pain of diaphragmatic pleurisy felt in the right hypochondrium, and the girdle sensations of disease of the spinal cord are abdominal pains of purely nervous origin. The last may be a mere sensation of a cord or belt around the waist or it may constitute an actual pain. It is usually upon the level of the umbilicus or higher but may be lower. The pain is sometimes much less marked upon one side than upon the other and may suggest a unilateral new growth or other form of one-sided abdominal disease.

Pains in the Extremities.—In general terms the diagnostic significance is the same for the arms and hands and for the legs and feet. The exceptions are mainly as follows: The pain of angina pectoris extends to the arms and especially to the left arm and involves the ulnar distribution. The pain in writer's spasm and other occupation neuroses involves the forearms and hands. It consists of irregular darting pains in the affected muscles and the usual pains attending the spasm upon effort. The pains of dactylitis, onychia, and paronychia involve the fingers. Gout occasionally affects the fingers, but usually the foot and especially the great toe. A group of painful affections are due to improper foot wear—ingrowing toenail, corns, bunions and metatarsalgia. The pains of flat-foot, varicose veins and varicose ulcer are to be considered in regard to the habitually erect posture. The especial liability of the knee and ankle to troublesome painful affections and the greater frequency of venous thrombosis in the lower extremity are due to postural conditions and the greater distance of the blood-vessels from the heart. Referred pains are common in the lower extremities. The pain in hip-joint disease and obturator hernia is often referred to the inner side of the knee; that of ovarian and uterine disease, fecal impaction, aneurism, and other abdominal tumors, to the inner surface of the corresponding thigh, and in rare instances that of acute disease of the prostate gland to the sole of the foot. Pains in the limbs associated with numbness and tingling have occasionally been observed in the prehemiplegic stage of cerebral hemorrhage. Pain in the toes, due to peripheral neuritis, is an occasional affection after enteric fever. The affection is not attended by the signs of inflammation and passes away in the course of some days. Painful muscular cramps in the post-dormitium usually involve the lower extremities and in particular the muscles of the calf of the leg. They occur in pregnancy, in gouty subjects, and in persons otherwise in good health. Similar painful cramps may attend violent exertion and exposure to cold, as in swimmers.

The painful affections common to the upper and lower extremities, aside from traumatism and the action of cold, as in frost-bite, involve the muscles, nerves, blood-vessels, articulations, and bones.

1. Pain is symptomatic of myalgia from unaccustomed or habitual overwork. It shows itself in athletes, dancers, horseback riders, pedestrians and soldiers after forced marches and is without diagnostic significance. Muscular pain occurs in various forms of myositis and especially in trichiniasis. General muscular pain is a symptom of rickets: It occurs in scurvy and is distinctive of infantile scorbutus, in which it is a prominent symptom upon both voluntary and passive movement of the legs. Painful cramp upon muscular effort—intermittent claudication—occurs in thrombosis and arteriosclerosis of the lower extremities.

2. Nervous pain is symptomatic of neuralgia—tender points; paroxysms, pressure aggravation; neuritis either intrinsic or from pressure; peripheral neuritis or neuromata. Diffuse pain below the knees is especially common in alcoholic neuritis. Sciatica, as well as brachial neuritis, which is the same thing in the upper extremity, is in some instances a neuralgia; in others a neuritis of the nerve or its plexus. It is almost always unilateral. Lightning pains occur in spinal disease, especially tabes. They are more

common in the legs than in the arms. They are sometimes localized. Bilateral neuralgic pains in the arms and legs are due to spinal cord disease as sclerosis, to general toxic conditions as lead or arsenic, to vertebral disease, or in the lower extremities to pressure upon the nerve-roots of the cauda equina.

3. Venous thrombosis—*milk-leg, phlegmasia alba dolens*—is often extremely painful. It occurs in lying-in women and as a sequel to enteric fever and other infectious diseases. A similar condition may occur in consequence of local pressure in the upper extremity. Pain, usually tingling or burning in character, occurs in the early stages of local gangrene, in ergotismus, diabetes, and Raynaud's disease.

4. The joints are especially liable to pain. Exquisite pain is experienced in the joint affection of rheumatic fever. The wrists, elbows, knees, and ankles are especially liable to involvement. Another exquisitely painful joint affection is gout. Arthritis deformans is attended by occasional outbreaks of pain, each of which results in an increase of the previously existing deformity of the joints. Many of the cases described under the term chronic rheumatism belong to this category. The pain in gonorrhœal arthritis is persistent and rebellious to treatment. That of ordinary synovitis is of moderate intensity. Pyæmic joints are usually exquisitely painful. Postfebrile arthritis closely resembles the joint affection of rheumatic fever. In spinal arthropathies—*Charcot's joints*—and in tuberculous joints pain is not always a conspicuous symptom.

5. All forms of periostitis are accompanied by pain. The subperiosteal hemorrhages of scurvy are attended with pain, which is also a common symptom in osteomyelitis and a group of cases of osteitis deformans.

TENDERNESS.

Tenderness is pain upon pressure. It usually but not invariably accompanies spontaneous pain. Intestinal colic and some forms of neuralgia are relieved by pressure. Tenderness may be present in the absence of spontaneous pain. This symptom is often of considerable diagnostic value, but being purely subjective it is liable to the uncertainties which modify the diagnostic significance of spontaneous pain. It is attended by objective manifestations, as wincing, flinching, exclamations of suffering, and the like. As in the case of spontaneous pain the allegations of the patient cannot always be depended upon. In certain cases tenderness may disappear when his attention is directed to other objects, or it may be present under the influence of suggestion or expectant attention, or finally it may be simulated in malingering.

A distinction is to be made between tenderness, which is pain upon pressure, and hyperæsthesia, which is an exaggeration of the sensibility of the skin. Tenderness is (a) superficial, namely, pain upon a very light touch; or (b) deep, that is, pain excited by pressure sufficiently firm to extend to underlying parts. Superficial tenderness is closely allied to hyperæsthesia and is usually coupled with a diminution of the power to recognize the nature of the agent by which the impression is caused—loss of tactile sensibility.

For practical purposes tenderness, like pain, may be best studied in relation to the parts in which it is localized and the anatomical structures involved.

The Head.—Tenderness of the scalp occurs during and after the attack in migraine, occipital neuralgia, and in hysterical conditions. Light pressure or the use of the comb or brush may excite pain. Local tenderness is present in traumatism, especially contusions, and subcutaneous effusions of blood. Diffuse tenderness may be elicited in myalgia of the occipito-frontalis muscle. Tenderness attends periostitis and caries of the skull. It is present also in gumma. Tenderness with or without local œdema is symptomatic of infection of the mastoid sinuses—*suppurative mastoiditis.* Localized pain is produced by tapping upon the skull in some cases of meningitis, tumor, and abscess of the brain—a symptom of minor importance.

The Face.—Tenderness immediately in front of the tragus is present in acute inflammation of the middle ear. Tenderness over the malar bone is symptomatic of abscess and malignant disease of the antrum of Highmore. The tender points in trifacial neuralgia are found at the emergence of the branches from the bony foramina and their penetration of fasciæ. There is occasionally also sympathetic tenderness at the occipital protuberance and over the upper cervical spines. Exquisite hyperæsthesia is encountered in some cases of neuralgia of the fifth nerve.

The Neck.—Localized tenderness is found in acute inflammatory conditions, as mumps, cellulitis—*angina Ludovici*—acute adenitis; in myalgia, the spastic rigidity of meningitis; in caries of the cervical vertebræ and in cervico-occipital and cervicobrachial neuralgia.

The Thorax.—Tenderness in the course of the spine occurs in meningitis, spondylitis, arthritis deformans involving the spine, periostitis, and in some cases of myelitis. It is a symptom of importance in neurasthenia, hysteria, and spinal irritation, and in lumbago. Pressure upon the tender points produces not only pain but also marked acceleration of the pulse—*Mannkopff's symptom.* Thoracic aneurism causing erosion of the vertebræ is a cause of tenderness in the dorsal or lumbar spine. Spinal tenderness may frequently be found in lumbar, subphrenic, and perinephric abscess, and has been observed in acute inflammation of the bronchial glands and in some cases of tumor of the mediastinum. In these conditions pain may be also called forth by sudden pressure upon the shoulders of the patient or by jarring the body, as by a misstep.

Tenderness attends periostitis and caries of the clavicles, sternum ribs, and cartilages. It may be present in these structures in the painful form of osteitis deformans, especially early in the course of the disease. It is found in abscess of the wall of the thorax, perforating empyema, and eroding aneurism. Tender points are present in intercostal neuralgia. Tenderness upon percussion is not uncommon in the infraclavicular regions in phthisis. The mammæ sometimes are tender at the menstrual period, in early pregnancy, in the condition known as irritable breast, which is a syndrome of hysteria, and in adenoma and malignant tumor. Tenderness is a symptom of pericarditis.

Abdominal tenderness is a very common symptom. It may be general, as in peritonitis, or local. The latter is usually present in a limited area, as the epigastric, hypochondriac, umbilical, hypogastric, or iliac regions; or the tenderness may be found in one of the quadrants of the abdomen. Sometimes the tenderness is distinctly focal, as in peptic ulcer, the McBurney point in appendicitis, the region of the gall-bladder, or pyosalpinx. In other cases it is diffused, with or without circumscribed areas of intensity.

Epigastric tenderness is a symptom in acute and some cases of chronic gastritis, pancreatitis, pericarditis, acute yellow atrophy of the liver, and disease of the gall-bladder and bile passages. It may be found in some cases of Addison's disease. One or more tender points are present in peptic ulcer. Tenderness in this region attends the myalgia of persistent cough and may be observed in hysteria and hypochondriasis.

Tenderness in the right hypochondrium is encountered in various diseases of the liver, as perihepatitis, congestion, acute hepatitis, abscess, cancer, acute yellow atrophy, and in diseases of the gall-bladder and bile-ducts, including cholelithiasis. In the last group of cases the tenderness may be confined to the region of the gall-bladder, or diffused over the hepatic area or even more widely; in the left hypochondrium in acute distention of the spleen, infarct, perisplenitis, pancreatitis, and fecal impaction; in either hypochondrium in diaphragmatic pleurisy; in both in influenza, relapsing fever, and the gastrohepatic form of estivo-autumnal malarial fever. Tenderness in the umbilical region may be elicited in peritonitis, enteritis, and enteric fever; in the right iliac region in enteric fever, appendicitis, renal calculus, fecal accumulations in the hepatic flexure of the colon, and in cancer; in the left iliac region in cancer of the sigmoid flexure and in some cases of membranous colitis; in either in pelvic inflammations and diseases of the tubes and ovaries; in both when any of these conditions are bilateral, and in hysteria. Hypogastric tenderness may be symptomatic of cystitis, inflammation of the pelvic organs, dysmenorrhœa, and hysteria.

The Extremities.—Cutaneous hyperæsthesia may be due to peripheral neuritis, especially the alcoholic form, neuritis involving a nerve-trunk in the course of which there are tenderness upon pressure and *points douloureux*, crural thrombosis, varicose veins; to periostitis, osteitis, osteosarcoma, arthritis, myalgia, myositis, rickets, scurvy, trichiniasis, or tetanus. Forms of arthritis especially characterized by pain and tenderness are encountered in rheumatic fever, the acute process in arthritis deformans, the gonorrhœal joint infection, gout, sprain, and tuberculosis. The hysterical knee is usually exquisitely painful upon pressure.

PARÆSTHESIA.

Paræsthesia is a condition of modification of normal sensibility. The phenomena are due to irritation of the sensory nerves in their course or distribution. They depend upon nutritive disturbances of the nervous system or the action of toxic or irritating substances in the blood. The itching of mild morphine intoxication is an example. Similar symptoms occur in gout and lithæmia and are met with in neurasthenia and hysteria. Sensations of numbness, burning, stinging, itching, and formication are common. Coldness, weight, tenesmus, the girdle sensation, precordial constriction, tightness, throbbing, sinking, faintness, and debility also belong to this group of symptoms. The sensations are closely allied to pain and are often described as painful by the patients. They are wholly subjective and their value in diagnosis rests entirely upon the ability of the physician to estimate the patient's accuracy of expression and desire to communicate the truth. There is no objective method of testing his statements.

These perversions of sensibility are very common and in many instances constitute the principal if not indeed the only symptom of which patients complain. They are much more common in women than in men and in the well-to-do than in the poor. Common associated conditions are defective digestion, constipation, anæmia, and general malnutrition. Important etiological factors are overwork, worry, irregular or indifferent meals, the stress of life, too frequent child-bearing, prolonged lactation, and enteroptosis. Remarkable forms and combinations are described by women passing through the grand climacteric. Forms of general and local paræsthesia constitute important epiphenomena of many chronic morbid states.

Cerebral Paræsthesiæ.—Sensations of heat, fulness, pressure, and other abnormal sensations in the head—the so-called *cerebral paræsthesiæ*—occur in neurotic individuals and over-taxed brain workers. These abnormal sensations do not amount to actual pain, though they frequently alternate with it. They are often distressing and sometimes intense. They occur in adolescence and early adult life and are especially common in women about the time of the grand climacteric. They are, however, more common in men than in women and in those given to intellectual pursuits and of sedentary habits than among the laboring classes. They occur with great frequency in lithæmic and gouty individuals. These sensations are sometimes general, sometimes localized to the vertex, occiput, or forehead. They frequently persist for long periods of time, in some cases preserving the same character, in others varying. They are augmented by mental effort and by disagreeable emotions and intensified by introspection and attempts on the part of the patient to explain them to his physician. They are, on the other hand, minimized by diversion and suggestion.

Forms of Paræsthesia.—The paræsthesiæ may be best studied in respect of their character, since almost any of them may be referred to various parts of the body and all parts at different times. They are described in the most varied combinations, so that numbness and tingling, itching and formication, burning and stinging, coldness and tension, tightness and throbbing, and many others occur.

NUMBNESS.—This is a common symptom in superficial injuries of the skin from cold or heat; the action of corrosive substances, as the mineral acids and carbolic acid; overdoses of certain drugs, as aconite and the bromides; injuries of nerves, neuritis, neuralgia in the stage of access and decline and in the remissions of pain; herpes zoster; peripheral neuritis from any cause and in the endemic form of the tropics, beriberi; hysteria, neurasthenia, tetany, tabes, the early stages of myelitis, and in cerebrospinal fever. Numbness may be a localizing symptom in coarse lesions of the brain, as tumor or abscess. It may occur as a premonitory symptom in apoplexy and as the aura in epilepsy. The sensation is sometimes described as like that produced by a very mild faradic current. It is common and distressing in myxœdema and may be a troublesome symptom affecting the hands and feet in arthritis deformans. Numbness in the hands and feet constitutes the condition known as *acroparæsthesia.* Waking numbness occurs at or about the menopause. It involves the extremities and usually passes off as the day goes on and ceases when the patient becomes adjusted to the non-menstrual life. Numbness is sometimes associated with or alternates with burning and tingling.

Itching or Pruritus.—This form of paræsthesia is frequently associated with formication and is sometimes so severe as to be described as pain. It is also associated with burning, especially in inflammations of the skin such as occur in the exanthemata, as measles and scarlet fever. Itching of the scalp is a symptom of seborrhœa; of the lips and nose a symptom of herpes; of the eyelids a symptom of beginning conjunctivitis; of the anal region a symptom of hemorrhoids or ascarides; of the external genitalia in both sexes a symptom of saccharine diabetes, in the female of leucorrhœa and neurotic states. Itching of the whole surface is a troublesome symptom in aged persons, in certain subjects in winter, in others who are lithæmic or gouty, in hysteria, neurasthenia, and many organic diseases of the nervous system. It is a symptom of jaundice, and sometimes follows the administration of morphine, copaiba, and other drugs. Pruritus is an occasional symptom in chronic interstitial nephritis and chronic lead poisoning.

Coldness.—A common form of paræsthesia. It is often general, as in the chill, rigor, or shivering which marks the onset of an acute febrile infection as pneumonia, or constitutes the initial stage of ague. Under these circumstances the internal temperature is elevated. Sensations of coldness with a normal or subnormal temperature occur in myxœdema, profound asthenia from any cause, especially after hemorrhage, hysteria, neurasthenia, and in some forms of spinal cord disease, as tabes, lateral sclerosis, and syringomyelia. Coldness in the back is often experienced by persons who are suffering from pulmonary tuberculosis in the period of incipiency. Subjective sensations of coldness in the extremities are usually associated with actual low temperature and often with some degree of cyanosis. In other cases the sensation of coldness is referred to a circumscribed area, usually in the leg or thigh. The affected region feels as though in contact with a piece of cold metal or even a piece of ice. This symptom occurs in neuropathic persons usually in middle life and commonly in men. It has been observed in local injury to a nerve-trunk and in spinal diseases.

Heat.—Heat as a subjective sensation not dependent upon general or local elevation of temperature constitutes a common and distressing paræsthesia. When it amounts to pain it is known as *causalgia.* It is mostly localized. Flushing is accompanied by the sensation of heat. Flushes of heat are common in stout women at middle life, at or about the menopause, and in nervous persons with weak heart. Subjective sensations of heat are sometimes associated with the girdle sensation.

Weight.—This paræsthesia is likewise of common occurrence. It occurs in the chest as a symptom in severe acute bronchitis, asthma, pleural and pericardial effusion, and mediastinal tumor; also in great cardiac hypertrophy and dilatation and in valvular disease upon rupture of compensation. Substernal weight and oppression may be a symptom of acute indigestion or of an overloaded stomach and may precede hæmatemesis. The sensation may be referred to the epigastrium. Weight upon the chest occurs in hysteria and neurasthenia and constitutes the *incubus* in nightmare. It is symptomatic of enteroptosis and splanchnoptosis, ascites, and abdominal and pelvic tumors.

Tenesmus or Bearing Down.—This form is frequently so distressing as to amount to actual pain. The milder forms are encountered in over-distention of the bladder, straining at stool, and some varieties of dysmenorrhœa.

Precordial constriction or stenocardia accompanies the pain of angina pectoris. Similar sensations but much less intense are sometimes experienced in cardiac asthenia, myocarditis, fatty heart, pericarditis, and when the heart is displaced upward by large ascites or abdominal tympany. It belongs also to the wide group of sensations in hysteria and neurasthenia.

Throbbing.—Sensations of throbbing are felt in conditions characterized by vascular relaxation and nervous excitement. Among these are aortic regurgitation, anæmia, and paroxysmal states in hysteria and neurasthenia. Almost every part of the body may be the seat of these sensations. They affect the head in migraine and other intense headaches; the neck in front and laterally in cardiac hypertrophy and exophthalmic goitre; the precordia in palpitation; the epigastrium in the pulsating aorta of neurasthenia; and constitute a local symptom in phlegmon and aneurism. Throbbing is commonly associated with objective pulsations. Purely subjective sensations of fluttering are described by nervous women. They are often referred to the left inframammary region.

Faintness.—Faintness is a sensation attendant upon enfeeblement of the heart's action, whether due to physical or emotional causes. Hence it occurs in dilated heart, myocarditis, fatty heart, and all forms of anæmia, especially upon exertion; in hemorrhage, shock, collapse, and upon the too sudden withdrawal of fluid by the trocar or aspiration; and finally in fatigue, excessive heat, and intense pain. Faintness attends sudden depressing emotion and mental shock. Weakness and debility are attended by subjective sensations which are characteristic and important, since they are often danger signals in the absence of the objective phenomena of oncoming disease. Sudden sensations of weariness out of all proportion to effort—*fatigue symptoms*—are suggestive of neurasthenia.

XIV.

GENERAL SYMPTOMATIC DISORDERS OF THE NERVOUS SYSTEM (CONTINUED): VERTIGO; CONVULSIONS; TREMOR; FIBRILLARY TWITCHINGS.

VERTIGO.

Vertigo—literally a turning—is a symptomatic derangement of the nervous system governing the relationship of the body to external objects. It is of two kinds: *objective vertigo,* characterized by sensations of movement on the part of surrounding objects which are really at rest, and *subjective vertigo,* characterized by sensations of movement on the part of the individual himself. It is popularly known as *dizziness* or *giddiness.* This symptom attends organic intracranial disease, but is more common in peripheral or functional disturbance. Vertigo in which no underlying pathological condition is discoverable is known as *essential.* Vertigo is a common nervous symptom. It is often associated with headache. It may occur (1) in mild cerebral concussion; (2) circulatory disturbances, as cerebral anæmia and hyperæmia; (3) local nerve irritation, as mechanical irritation of the external auditory meatus, inflammation of the middle ear, or the application of electrical currents to the head. A special form of vertigo—*true auditory vertigo*—occurs in labyrinthine disease. (4) Vertigo is a common symptom in toxæmic conditions and is associated with headache in the period of onset of the acute infections, in many cases of acute and subacute gastrohepatic derangements, and in lithæmia. It is symptomatic of narcotic poisoning, especially that produced by alcohol, tobacco, opium, and the nitrites. It occurs also in aniline poisoning. (5) It is a common symptom in arteriosclerosis and (6) in valvular disease of the heart, especially aortic insufficiency, and in forms of degenerative myocarditis; (7) in neuropathic conditions, especially neurasthenia and epilepsy; (8) in reflex disturbances, such especially as arise from diseases of the visual apparatus or the stomach; (9) in organic disease of the brain, especially in tumor, cerebellar disease, in meningeal irritation and meningitis, and in brain syphilis. Finally, (10) vertigo results from mechanical causes, such as swinging, certain unusual postures, rapid rotary movements, and sea-sickness.

Vertigo varies in intensity from a trifling sensation of imperfect equilibrium—mere swimming of the head—to the most active and distressing sensations of rapid or irregular movement or whirling of the body or of surrounding objects.

The equilibrium of the body is maintained by muscular action. The nicely adjusted and constantly varying motor impulses necessary to equilibrium are determined in cerebral centres in response to sensory impressions which are as continuous as the motor impulses which respond to them. These sensory impulses are visual, aural, muscular, articular, cutaneous and visceral. Anything which suddenly deranges the continuous and systematized though unconscious sensory impulses from these structures causes a derangement of the nervous mechanism by which the body is maintained in its relation to external objects. This derangement manifests itself as vertigo. These sensory impressions are not felt in normal consciousness, but when they are interrupted or when the cortical processes by which they are converted into motor impulses are deranged consciousness in regard to them is perverted and vertigo results. For this reason vertigo implies a disturbance, not a loss of consciousness. In true vertigo consciousness is always retained.

Vertigo comes on suddenly and is commonly of short duration. In the objective form the floor or the bed on which the patient is lying appears to rise and sink and objects whirl around, usually in a definite direction. In subjective vertigo the patient himself appears to be whirling around or rising and sinking in space. These sensations are often accompanied by compensatory movements on the part of the patient which may result in a fall. Mental confusion, faintness, a sense of alarm, and nausea or vomiting are associated symptoms, which vary in intensity but are almost always present. When the vertigo is severe consciousness is impaired but not lost. The attacks continue to recur whilst the causal condition persists. The term *status vertiginosis* has been applied to persistent vertigo.

The following forms of vertigo demand separate consideration:

AURAL VERTIGO.—This symptom frequently arises from the pressure of accumulated cerumen in the external auditory canal or from the pressure of air against the tympanic membrane by a blow upon the ear, or the entrance of water in diving or surf bathing, or the too forcible use of the ear syringe. It may also occur, though it is not a common symptom, in cases of middle-ear disease or from the use of the Eustachian catheter. Vertigo occurring under the above circumstances is usually slight and transitory. Labyrinthine vertigo is the chief symptom in Ménière's disease.

TOXIC VERTIGO.—Vertigo which attends the onset of the acute infections is of no great importance and usually quickly passes away. That which occurs in gastrohepatic catarrh is commonly annoying on rising in the morning in persons of bilious temperament and sedentary lives, especially if they be addicted to the pleasures of the table. This symptom occurs also in acute indigestion and in lithæmic conditions. Vertigo is a very common drug symptom, which is, however, much influenced by habit and idiosyncrasy.

CARDIOVASCULAR VERTIGO.—Vertigo is a symptom of cerebral anæmia. It occurs in sudden blood loss, cardiac asthenia, excitement, or sudden effort during digestion, upon sudden effort in myocarditis, valvular disease, and in particular aortic insufficiency. It occurs also in pernicious and other forms of anæmia, chlorosis, and leukæmia. Associated with tinnitus aurium it is very common in sclerotic changes in the branches of the cerebral arteries.

NEUROTIC VERTIGO.—Vertigo sometimes occurs in epilepsy as an aura. It is not rare in petit mal. Vertigo is a common and distressing symptom in neurasthenia. The attacks are frequent but not commonly severe or prolonged. They are attended with nausea, though vomiting is not common. It is usually subjective and frequently reflex. Stumbling or paralyzing vertigo has been observed in exophthalmic goitre and as an endemic condition in certain cantons of Switzerland during the summer. There is a sudden loss of power in the legs with impairment of consciousness. Paroxysmal vertigo may occur in nervous individuals after excitement or fatigue. It is very distressing, occurring suddenly, accompanied with nausea and vomiting, and lasting sometimes for hours.

Reflex vertigo may be associated with the brow pains and other forms of headache which are symptomatic of errors in refraction or want of harmonious action in the ocular muscles.

Mechanical Vertigo.—This symptom attends sudden lowering of the head, whirling around, or swinging in individuals not accustomed to it, and is a very important part of the symptom-complex in sea-sickness and car-sickness. Mild persistent vertigo has been observed in elevator boys.

Vertigo of Intracranial Disease.—This is a very common symptom in diseases of the brain and its meninges. It is sometimes distressing but as a rule is of secondary importance to the headache, vomiting, and mental dulness with which it is commonly associated. It occurs at some time during the course of meningitis, cerebral abscess, thrombotic softening, tumor of the brain, and cerebellar disease. This form is of considerable importance in the diagnosis of cerebral syphilis.

Laryngeal vertigo, better called laryngeal epilepsy, usually manifests itself in neurotic adults. The paroxysm begins with tickling or irritation in the larynx, cough, partial loss of consciousness, and dyspnœa. Light tonic or clonic movements occur. The patients suffer from laryngitis, bronchitis, asthma, or pulmonary phthisis. The attacks recur as often as once a day or at longer intervals.

CONVULSIONS.

The term convulsion is used to designate a paroxysm of involuntary and more or less violent muscular contractions involving the voluntary muscles in general. The word spasm is frequently used in a more limited sense to indicate similar involuntary contractions of the muscles of particular parts of the body. We speak of general convulsions and local spasms. This distinction is, however, not always observed.

General Convulsions.—Convulsions are tonic and clonic. A tonic convulsion is an involuntary muscular contraction which is continuous and intense. It may be of brief duration, as in the beginning of the epileptic paroxysm; or prolonged, as in tetanus. A clonic convulsion is characterized by the rapid alternation of contraction and relaxation, as in the second stage of the epileptic paroxysm or in infantile eclampsia. The posture in tonic convulsions is forced and immovable; in clonic convulsions it is constantly changed. The arms and legs are alternately flexed and extended with more force than in ordinary movements, the body is violently tossed, and the muscles of the face contorted. The chief centre for convulsions is the cerebral cortex. Tonic and clonic convulsions may succeed each other, as in epilepsy, or may alternate, as in hysteria. Consciousness is often preserved in general convulsions of the tonic type, as strychnine poisoning and tetanus, and usually lost in those of clonic type, as epilepsy and uræmia. A spasm may be confined to a muscle or a group of muscles; or it may extend to an entire limb or the whole of the body. A cramp is a painful tonic spasm affecting a single muscle or group of muscles, as the well-known cramp in the calves of the legs.

Etiology.—From the standpoint of etiology convulsions are symptomatic of (1) local irritation; (2) general cortical irritation, (a) from causes wholly unknown, (b) from the toxæmia of infection, (c) from various intoxications; (3) circulatory derangements; (4) inflammatory and degenerative processes involving the cerebral cortex; (5) convulsions are very often of reflex origin.

Convulsions are essentially paroxysmal. Even though the cause is persistent, the motor centres become exhausted and there are intermissions, as in uræmia. Again the paroxysms occur as storms, the cause exhausting itself in a single paroxysm or series of paroxysms and only again asserting itself after an interval more or less prolonged, as in ordinary epilepsy. In infancy, in the children of neurotic parents, and in neuropathic individuals convulsions frequently arise from the action of causes not capable of producing them at a later age or in normal individuals.

1. LOCAL IRRITATION.—The motor areas may be directly involved in fracture, hemorrhage, cicatrix, or neoplasm, as in focal or Jacksonian epilepsy. The initial symptom may be a local spasm, involving the leg, arm, or face, the convulsion becoming generalized in the course of a few seconds or longer. Again the local irritation may be transmitted from a distance, as in tumor, abscess, or sclerosis.

2. THE IRRITATION MAY BE GENERAL, (a) from causes wholly unknown, as in epilepsy. The paroxysm is frequently preceded by an aura; it begins with tonic spasm and loss of consciousness and is characterized by clonic convulsions. So characteristic is the latter stage, that general convulsions due to other causes are described as epileptiform or epileptoid. The paroxysm is followed by hebetude, drowsiness, or stupor, and may be replaced by a maniacal outbreak or other mental disturbance—*the psychical epileptic equivalent.* (b) The toxæmias of infection. General convulsions very commonly attend the onset of the infectious diseases in childhood. They occur at this period of life as the equivalent of the initial chill in the adult and are frequently seen at the onset of scarlet fever, measles, and pneumonia, and in other not well defined infections. They are frequent in rickets, which is the most important predisposing cause of infantile convulsions. They are early symptoms of that disease, and when convulsions occur in infancy without manifest cause rickets is to be considered. The convulsions of tetanus, strychnine poisoning, and hydrophobia are to be considered under this heading. (c) General convulsions occur in poisoning from aconite, prussic acid, and veratrum viride, and in chronic alcoholism and lead poisoning. Under this heading are to be included the convulsions of uræmia, puerperal eclampsia, and asphyxia.

3. CIRCULATORY DERANGEMENTS are sometimes the cause of general convulsions which occur after profuse hemorrhages, and in the cerebral anæmia which immediately precedes dissolution. Violent general convulsions occasionally occur during the coma following sunstroke.

4. INFLAMMATORY AND DEGENERATIVE PROCESSES involving the cerebral cortex give rise to general convulsions. Under this heading are to be considered the convulsions of cerebrospinal fever and other forms of meningitis, cerebral syphilis, general paresis, and pachymeningitis hæmorrhagica.

5. CONVULSIONS are very often of reflex origin. Painful affections and excitation in the region of a sensory nerve may produce spasms. Examples of reflex convulsions are those following severe injuries, burns, those associated with renal or intestinal colic, a foreign body in the ear, intestinal strangulation, retention of urine, and phimosis. Dentition and intestinal worms are less common causes of convulsions than is generally supposed. Indigestion is a cause of convulsions in infants and older children. In whooping-cough convulsions are very common. They result from the asphyxia attendant upon a prolonged paroxysm, cerebral congestion, or hemorrhage resulting from such a paroxysm. In other cases they are to be attributed to the depressed condition of the nervous system caused by the disease itself. General convulsions have been attributed to enlargement of the thymus gland as a result of pressure either upon the pneumogastric or upon the trachea. They frequently occur in children in whom no cause can be discovered and may in such cases be regarded as idiopathic. In infants in whom an attack of convulsions has once occurred a predisposition seems to be established, so that similar attacks occur from indifferent or not recognizable causes. In infantile convulsions the attack is commonly preceded by restlessness, fretfulness, grinding of the teeth, and slight twitching. It may occur suddenly without premonitory symptoms. The initial cry so common in epilepsy is usually absent, nor are the successive stages so well defined. The spasm begins in the hands; the eyes are fixed and staring or strongly turned upward; the body rigid, and the face congested. The convulsion is at first tonic, so that respiration is suspended, but presently clonic convulsions set in, the eyes are moved from side to side, there are violent twitchings or alternate flexions and extensions of the limbs, contortion of the face, and retraction of the head. There is spastic flexion of the fingers, the thumb being against the palm—*clenched fingers.* These movements gradually cease and the child passes into a condition of stupor. There is usually slight elevation of temperature. Convulsions arising from indigestion and those which usher in an infectious disease are commonly single, but those due to rickets recur in series. In some instances one attack succeeds another until death ensues.

When the attack occurs in a healthy child, it may be due to acute indigestion or some form of peripheral irritation; when accompanied by high fever and vomiting it may be the forerunner of an acute infection, as scarlet fever, or of infantile hemiplegia; when it occurs in badly nourished or rickety children it is apt to be incomplete and to recur. The convulsions of infancy do not of necessity run on into epilepsy, but general convulsions occurring without apparent cause at irregular intervals in young children otherwise healthy are in a limited proportion of the cases epileptic from the beginning.

Uræmic convulsions may be preceded by headache and restlessness. Sometimes they come on without warning. The epileptic cry does not occur, but in other respects the attack may resemble true epilepsy. The convulsions are often recurrent and prolonged, the seizures being separated by periods of coma or deep stupor. The temperature is usually subnormal; exceptionally it is elevated. The condition is recognized by the characters of the urine, the presence of œdema, the condition of the heart and arteries, a urinous odor, and the history of the case.

Puerperal convulsions present the same clinical picture as those which occur in ordinary nephritis.

Hysterical convulsions are to be distinguished from epilepsy by the emotional state which precedes the attack, the *globus hystericus*, the difficult respiration, the alternating laughter and tears. Sensations may be described which suggest the epileptic aura, as precordial, abdominal, or pelvic uneasiness or distress. The patient does not fall to the floor in instant and complete unconsciousness as in epilepsy, but gently or by preference upon a sofa or couch in such a way as to do herself no harm. The movements are irregular and clonic but usually much less violent than in epilepsy. The tongue is not bitten. The attack gradually subsides and the patient becomes conscious and emotional again. At the close of the attack a large amount of light-colored urine of low specific gravity is often voided. The more violent convulsions, manifestations of hystero-epilepsy, include grinding of the teeth, tonic spasm, opisthotonus, and other forced attitudes, clonic spasms, and more or less profound unconsciousness. The attack is more prolonged than in epilepsy and is followed by contortions and cataleptic poses and in some instances by attitudinizing suggestive of various passionate states.

In tetanus the earliest symptoms are slight stiffness of the neck and some embarrassment in mastication. These symptoms gradually increase until the condition of trismus or lockjaw develops. The spasm extends and involves the muscles of the body, causing the rigid attitudes known as opisthotonus, orthotonus, pleurotonus, and emprosthotonus. Respiration is interfered with by the muscular spasm and asphyxia may threaten from closure of the glottis. The convulsive paroxysms are excited by the slightest irritation and are of variable duration. Complete relaxation may not occur during the intervals. There is usually a history of trauma.

The resemblance of strychnine poisoning to tetanus is close. Trismus is absent as a rule and the relaxation between the convulsive paroxysms is complete. There is a history of the ingestion of the poison.

Tetany is characterized by the peculiar position of the hands and feet, the involvement of the extremities, less often the face and neck, and the presence of Trousseau's symptom—the reproduction of the paroxysm by compression of the affected part either in the direction of the principal nerve-trunks or over the blood-vessels; or of Chvostek's symptom—an increase in the mechanical irritability of the motor nerves, a slight tap over the nerve-trunk being sufficient to throw the muscles into active spasm. The history of the case is quite different from that of both tetanus and strychnine poisoning.

TREMOR.

Tremor is a rhythmical to-and-fro movement of limited range due to the alternate contraction and relaxation of opposing muscles. The movements are involuntary and differ from fibrillation in that they cause locomotion of the parts involved. It is due to nutritive alterations in the motor neurons both of the cortex and spinal cord. A distinction is made between *intention* or *volitional tremor*, which shows itself only upon intentional movements, and *passive tremor*, which occurs when the parts are at rest. The former is sometimes spoken of as paralytic; the latter as spastic tremor. In the examination the patient is to be observed at rest, in intentional movement, and in attitudes which require sustained tonic contraction of the muscles, as horizontal extension of the arms and hands, separation of the fingers, or protrusion of the tongue.

The following forms of tremor are of diagnostic importance:

1. The Intention Tremor of Multiple Sclerosis.—This form of tremor does not occur during rest, but shows itself upon intentional movement, usually at first slight, then progressively more rapid and with wider oscillations, so that the intended movement is greatly hindered. The movements in some cases are so great and so irregular as to suggest ataxia. The rate of the tremor in disseminated sclerosis is given by Peterson at 7.9 to 8.1 per second for the earlier stages and 4.6 to 6.3 for the later stages.

2. The tremor of paralysis agitans is distinctly slower. It continues during rest, becomes less marked upon movement, and upon determined impulse of the will may disappear for a brief period. The rate is from 3 to 6 per second. This form of tremor disappears during sleep. It usually first appears in the hands and is characterized by rhythmical movements of the index finger against the thumb which suggest pill rolling. The tremor of paralysis agitans very seldom affects the head.

3. Senile tremor is in its more moderate forms an intention tremor; in well developed forms a tremor of rest. The hands and arms are more commonly involved, but the head is often affected and the under jaw and lips. The rate is from 4 to 6 oscillations per second.

4. The tremor of exophthalmic goitre is best manifested in the hands when extended and the fingers separated. It sometimes affects the head. The rate is rapid—8 or more per second—and the excursion limited. Upon intentional movements the tremor is sometimes increased. This form of tremor is common in hysteria, in which, however, every form may be encountered. It is seen also in tuberculous meningitis, in lesions of the corpora quadrigemina, and rarely in disease of the cerebellum. Similar tremors occur in the acute febrile diseases. The tremor of enteric fever is an example. It occurs even in mild cases and is most noticeable in the tongue when it is protruded for examination. At first fine, it becomes coarser as the exhaustion increases. The lips are affected and in severe cases the hands. It is more marked in persons who are addicted to alcohol. Murchison regarded excessive tremor as one of the signs of deep ulceration of Peyer's patches.

5. The toxic tremors are usually fine. They are intensified upon intentional movement. The more common causes are alcohol, tobacco.

morphine, and mercury. In alcoholic tremor first the hands and then the lips are affected, and it is temporarily intensified upon the withdrawal of alcohol and diminished by its administration in increased doses.

6. Tremor due to miscellaneous causes, as intense emotion, excessive or prolonged muscular effort and extreme cold may occur in healthy persons and is without diagnostic importance. Popular phrases are *trembling with anger* or *fear* or *cold*, and *buck fever*, in the inexperienced hunter.

FIBRILLARY TWITCHING OR FIBRILLATION.

This is an involuntary, brief, sluggish contraction of groups of muscular fibres rather than of an entire muscle. It is manifested as a wave-like movement of feeble intensity just under the skin, not involving the muscle as a whole and producing no movement of the parts to which the muscle is attached. It may occur in a limited number of fibres at long intervals, or in successive groups of fibres in rapid succession. There are cases in which fibrillary contractions do not occur spontaneously but can be excited by tapping the skin overlying the muscle with the finger, and in those cases in which they occur infrequently they may be produced in the intervals by the same manœuvre. They often occur in healthy persons upon exposure of the surface of the body to cold air. Fibrillation is probably caused by a lesion which at once weakens and irritates the cell-body of the peripheral motor neuron in the anterior horn of the spinal cord (Lloyd). It is, therefore, symptomatic of progressive degenerative processes involving and gradually destroying the large ganglionic motor cells, and occurs in paretic and atrophic muscles when those changes are of nuclear origin. This phenomenon is especially seen in anterior poliomyelitis and in bulbar paralysis. It may be present in traumatic neuroses without paresis or atrophy.

Other morbid motor phenomena are discussed in the chapter upon the Examination of the Nervous System.

XV.

PSYCHICAL CONDITIONS, EMOTIONAL STATES, DERANGEMENTS OF CONSCIOUSNESS, INSOMNIA AND OTHER DISORDERS OF SLEEP.

PSYCHICAL CONDITIONS.

The consideration of abnormal mental phenomena comes properly within the scope of psychiatry. Mental derangements constitute at times, however, important symptoms in almost every department of internal medicine. The degree of intelligence, defects of memory, emotional states, and irritative and depressive derangements of consciousness are to be considered. Closely allied are insomnia and other disorders of sleep.

Intelligence.—The age, education, and social surroundings of the patient are to be considered. Derangements of intelligence are frequently associated with impaired consciousness but may occur independently of it. Both vary greatly in degree. Slight intellectual defects often not recognized in the ordinary demeanor and conversation of the patient become apparent upon further knowledge or upon taking a careful clinical history. The lower grades, designated by such terms as dulness and stupidity, or an extreme degree, as idiocy and dementia, are immediately apparent in the facial expression and behavior of the individual. Not infrequently a falling off in intelligence is manifest in persons suffering from chronic incurable affections, as valvular disease of the heart, nephritis, tuberculosis, and cancer. Not only is the nutrition of the cerebral cortex impaired but the patient's range of thought becomes progressively more circumscribed. His interest in general affairs or the particular objects of his previous intellectual activity diminish in proportion as his interest in his symptoms and in the narrow life of the sick-room increase. Graver derangements amounting to absolute indifference, stupidity, or dementia are on the other hand observed in cerebral diseases, especially in tumors of the brain, progressive bulbar paralysis, multiple sclerosis, hemorrhage, thrombosis, embolism, and softening. In other cases stupidity or dementia may be the expression of a developmental anomaly of the brain, as in idiocy and cretinism. Of special interest is the derangement of intelligence which occurs in myxœdema, both that form which develops spontaneously and in the *cachexia strumipriva*. In this condition, which is closely allied to cretinism or indeed practically identical with it, the derangements of intelligence vary in degree from moderate apathy and indifference with slowness of thought associated with slowness of speech to a state bordering upon dementia. A transient abnormal exaltation in mental activity with a rapid flow of ideas and unusual facility of expression may attend hectic fever, the action of alcohol, and excitement due to other causes. A corresponding depression in mental activity is observed in the period of reaction. The patient who has been restless and talkative in the febrile period is depressed and silent during the sweating that attends the defervescence; the exhilaration of alcohol is followed by the depression of a physical if not a moral remorse; fervor of speech and energetic action give place to dulness and abstraction.

Mental dulness or confusion occurs independently of derangements of consciousness. Confusion of thought attends grave neurasthenia, cerebral [illegible] sclerosis, old age, and profound malnutrition. S[illegible] and awkwardness in expression are usually char[illegible] intelligence, but may indicate lesions of the nervous [illegible] expression, as in forms of aphasia.

Memory.—[illegible] becomes less accurate and [illegible] in good health and intelligence [illegible]. At earlier periods of life [illegible] same conditions of [illegible] therefore very often [illegible] brain such as result [illegible] necessarily asso[illegible] memory is very [illegible] demanding attention [illegible] Individuals recovered from severe traumatic neurasthenia very often have but faint recollection of the events associated with and following the injury. Loss of memory occurs in epilepsy, bromidism, and chronic alcoholism, is common in insanity, and often complete in terminal dementia.

EMOTIONAL STATES.

Mental depression is very common in chronic and incurable diseases. It is sometimes purely symptomatic. More commonly it arises from pain and suffering or from apprehension in regard to the future. A high grade of depression characterizes hypochondriasis and melancholia. In deep jaundice, especially when chronic, depression is very common. Mental depression frequently attends diseases of the stomach, particularly those in which pain is prominent. Periods of depression occur during the menopause and in pronounced neurasthenia, hysteria, and in cerebral disease.

Emotional exaltation characterizes acute and chronic mania and is an important element in active delirium. During anæsthesia by chloroform, ether, and nitrous oxide the early derangement of consciousness is manifest by emotional excitement which is often intense. A similar condition is characteristic of alcoholic intoxication.

Instability of temper, irritability, and sensitiveness are very common in invalids. The testiness and outbursts of anger which occur in gout and the fretfulness and impatience of uterine disease are well known. Emotional instability and similar changes in disposition are frequently observed in pregnancy.

DERANGEMENTS OF CONSCIOUSNESS.

These may be irritative or depressive. Irritative derangements of consciousness vary in degree from mild emotional excitement to furious homicidal mania; in extent from perversion in a limited region of consciousness relating to a single idea or group of ideas to systematized delusions influencing the whole life of the patient. Irritative frequently alternate with depressive derangements of consciousness. Delusions, illusions, and hallucinations are irritative derangements of consciousness.

A delusion is an unfounded conviction or belief. It is very often absurd or ridiculous. Delusions that are persistent and based upon false ideas having a logical interdependence or sequence are known as organized. An expansive delusion is an insane belief in the individual's own greatness, power, or goodness. No evidence or demonstration is sufficient to convince a person of the falsity of his delusions. Examples of delusions that are common are the belief that individuals, almost always unknown, are conspiring to do the patient a serious harm, or that the patient is the Christ or Solomon or Queen Victoria.

An illusion is a false or misinterpreted sensory perception. The phenomena upon which it is based actually exist. A patient who mistakes the nurse for an officer of the law, or a bundle of rags for her baby, or ordinary household sounds for the voice of God is the subject of an illusion. Illusions are very often transient or momentary.

An hallucination is a sense perception not founded on objective reality. Hallucinations may relate to any of the senses. The patient who sees the figures of bystanders or hears whispering voices, or perceives a disagreeable odor or unpleasant taste, or feels upon his shoulder the pressure of a hand when none of these objects exist, suffers from an hallucination. Hallucinations are frequently persistent and distressing.

The Obsessions.—An obsession is an idea which dominates consciousness often to the exclusion of other thoughts and ideas. It comes unbidden and cannot be dismissed by any effort of the will. Nevertheless its nature and unreasonableness are usually fully understood by the subject.

Obsessions very commonly take the form of definite systematized fears relating to certain objects or conditions. These constitute the so-called *phobias*, as *kenophobia*, the dread of large or open spaces; *claustrophobia*, the fear of closed or narrow spaces; *agoraphobia* (ἀγορά, a market place), the fear of a crowd; *aichmophobia*, the fear of pointed instruments or weapons or the dread of being touched by anything; *metallophobia*, a terror of touching or handling a metallic object; *pyrhophobia*, a morbid dread of fire; and many other forms of persistent and dominating fear.

Doubt constitutes a common form of obsession. The mental uncertainty may be restricted to a single subject or set of subjects or embrace almost every affair of life from the simplest to the most important, recurring with intolerable insistence and refusing to be allayed by the demonstration of the actual conditions to which they relate.

Another group of obsessions consists in a morbid exaggeration of the activities of life. Those who are subject to them are possessed of a demon of unrest and are irresistibly impelled to be continually doing something or going somewhere, usually aimlessly and without fixed purpose, and always ready without adequate motive to change from one occupation to another or from the selected course to a different one.

Closely allied to this group of obsessions are those which consist in an irritable impulse to touch a spot or an object—*folie de toucher*—or to repeat certain movements, as returning to pass through a door two or three times before departing from it, and the like.

Fixed ideas are closely allied to obsessions and the terms are often used interchangeably. There are those, however, who distinguish between these two derangements of consciousness, namely, that an obsession is recognized by the patient as an abnormal train of ideas without basis in fact, while the subject of a fixed idea is convinced that it is based upon the conditions as they exist and perfectly normal under the circumstances.

The foregoing derangements of consciousness are permanent symptoms in insanity. They occur also in hysteria and neurasthenia and constitute important elements of delirium.

Delirium is an irritative derangement of consciousness characterized by restlessness, excitement, and incoherence. Periods of delirium may alternate with somnolence, stupor, or convulsions. There are two forms of delirium. In the *active* or *maniacal* the patient is wild and noisy. He sings, screams, shouts, tries to get out of bed, struggles with his attendants, and has to be restrained by force. His face is congested, his eyes bright, his expression alert, excited, even fierce. The second form is *low* or *muttering*. The patient lies quiet, murmuring in a low tone, holding incoherent and often whispered conversation with imaginary persons, or occupied with vague fancies and taking no notice of what goes on around him. If aroused he may give a rational but brief reply to questions, quickly relapsing into his wandering dreams. This form of delirium is sometimes associated with restlessness. The patient moves in bed, may even try to get up, but is easily restrained. Between these two there are transitional forms attended with moderate restlessness and excitement. The patients are irritable, disturbed by trifles, and at times incoherent, though not boisterous.

Delirium develops very readily in persons of neurotic temperament and in early life. It may occur in any severe illness. It is especially common in fever and usually indicates a grave infection. In febrile diseases children are more liable to delirium than adults, just as they are more liable to high temperature. In general terms, there is no constant relation between particular diseases and forms of delirium. Active delirium is, however, frequently associated with the acute infectious fevers. The delirium of pneumonia is sometimes violent; in inflammatory diseases of the brain and in acute mania it is often furious. In fevers of ordinary intensity the delirium is of moderate type. It is muttering or wandering in the exhaustion of the low fevers and in the later stages of other acute diseases. Delirium may be present in uræmia and in poisoning by belladonna, cannabis indica, hyoscyamus, and opium, and a loud and boisterous delirium quite different from delirium tremens sometimes occurs in acute alcoholism. In enteric fever the headache usually ceases as delirium develops.

The onset of delirium may be abrupt or gradual. An outbreak of maniacal delirium has in rare instances been the first manifestation of an acute infectious disease, as enteric fever, typhus, or pneumonia. Cases have occurred in which under these circumstances individuals have been regarded as insane and placed in an asylum. Much more commonly delirium develops gradually, showing itself first in a certain confusion of thought upon awaking from sleep. In some cases delirium is absent during the day, coming on again and increasing as night approaches. Mild nocturnal delirium is sometimes seen during convalescence from pneumonia, enteric fever, and septic conditions.

The delirium of inanition occurs in wasting diseases and in starvation. It is not very rare in malignant disease of the œsophagus or stomach and occurs in cases characterized by intractable vomiting. The delirium of convalescence is probably a delirium of inanition. In this form of delirium the outbreak is sudden, usually in the early morning. There is feebleness of pulse and a relaxed and sweating skin with cold hands and feet. It is very often of brief duration, yielding in the course of some hours or a day or two to the proper administration of nourishment and stimulants. Maniacal delirium not uncommonly follows the epileptic paroxysm—*postepileptic mania*—or may develop as the psychical *equivalent* of the paroxysm.

The delirium of alcoholism—*delirium tremens*—is very characteristic. It is almost always associated with hallucinations which take the form of large numbers of small objects, as mice, bugs, serpents, which continually approach the patient and inspire abject and pitiable terror, or there are animals running around his bed or crawling upon the walls. The delirium is busy. The patient is restless, his hands are constantly moving, he tries to get out of bed, but is usually tractable. Associated symptoms of diagnostic importance are tremor and sleeplessness, which are almost constantly present. A condition not unlike delirium tremens may develop in other drug habits. It has been observed after prolonged excesses in morphine, chloral, and paraldehyde.

CARPHOLOGIA, literally a gathering of chaff, the picking at the bedclothes, seen in the wandering delirium of grave fevers and profound exhaustion, is of unfavorable prognostic significance. The patients lie quiet, wholly oblivious of their surroundings, plucking with feeble hands at the bed-covering or grasping at imaginary objects in the air. These movements are dependent upon hallucinations.

Delirium is sometimes simulated by malingerers. Feigned delirium is to be recognized by the absence of other signs of illness, the want of the characteristic incoherence, and by the continuing sameness and limited range of the manifestations.

Depressive derangements of consciousness vary in degree from simple clouding of the ordinary consciousness to complete unconsciousness. They affect the entire field of consciousness. Loss of consciousness may be sudden or gradual, and is a symptom of great diagnostic importance.

SOMNOLENCE is the term used to describe the mildest degree. The individual is dull, drowsy, and indifferent, but retains an appreciation of his surroundings and can respond more or less intelligently when addressed. Naturally there is no sharp line of demarcation between this and the following progressive conditions.

SOPOR, literally a sound or heavy sleep, indicates a more profound impairment of consciousness. [illegible] surroundings but can be aroused [illegible] replies in [illegible] has a con- [illegible] again into a [illegible]

[illegible] The patient [illegible] The ex- [illegible]

[illegible]

or follow intense or prolonged muscular effort, or accompany hemorrhage or shock. It is important in all cases to make the differential diagnosis between suddenly on-coming coma and syncope.

LETHARGY OR TRANCE is a condition of unconsciousness, more or less complete, which occurs in hysteria. It has been observed in rare instances after excessive mental application or exhausting disease and cases have been noted in which it has occurred in individuals otherwise apparently in good health. It differs from coma in resembling a deep and protracted sleep from which the patient in some instances may be partially aroused. The patient is usually pallid, the extremities relaxed, the eyelids closed, the eyes turned upward or to one side. The pupils vary in size but react to light. Respiration and circulation are greatly enfeebled. The temperature is subnormal. The attack varies in duration from some hours to several weeks. Cataleptic rigidity, or convulsions, may develop.

CATALEPSY is a condition of impaired consciousness characterized by rigidity affecting the voluntary muscles. A limb or the body of the patient may be maintained continuously for some time in the same posture. The position of the limb may be passively changed with slight resistance, remaining in the posture in which it has been placed. This condition of increased muscular tonus has been termed "*waxy flexibility.*" The attack may last for a few minutes or for several hours. It is attended with complete anæsthesia of the skin and deeper tissues. The rhythm of the respiration is disturbed, the circulation feeble, the surface temperature depressed, and the reflexes impaired. The eyes are usually open; the pupils are dilated but react to light. The attitudes are sometimes bizarre and grotesque. As the attack passes away the power of muscular movement is fully regained. Catalepsy is a rare symptomatic disorder. It is encountered in hysteria, occurs in hypnotic states, and has been observed in cerebral disease, as tumor and meningitis, and in forms of insanity, as melancholia.

Coma may be easily recognized. Its diagnostic significance is often obscure. It occurs not only in cerebral disease but in the most varied constitutional conditions. It may be symptomatic of the following:

(a) Organic disease of the brain, either general, as acute encephalitis, cerebral syphilis, multiple sclerosis, and general paresis; focal, as intracranial hemorrhage, embolism, thrombosis or softening, tumor, abscess and thrombosis of the cerebral sinuses; disease of the meninges, as inflammation, the pressure from exudate, and subdural hemorrhage or tumor; or, finally, it may occur in the course of disease of the cranial bones. (b) Traumatism of the head, producing cerebral commotion or compression. (c) The pre-agonistic state in all diseases terminating fatally. (d) The fully developed febrile infectious diseases. Only exceptionally is coma under these circumstances complete. Early and complete coma occurs in the malignant forms. (e) Uræmia, in which it commonly alternates with convulsions. (f) The last stage of diabetes. (g) Forms of auto-intoxication analogous to diabetic coma in which β-oxybutyric acid or its derivatives are present in the blood. (h) Rare cases of septicæmia, pyæmia, carcinoma, and acute yellow atrophy of the liver. (i) Narcotic poisoning, especially by alcohol, morphine, chloral, and various poisonous gases, and the surgical anæsthesia produced by the administration of ether, chloroform, nitrous oxide, etc. (j) General convulsions, infantile eclampsia, and the epileptic paroxysm. (k) Drowning and asphyxia from other causes. (l) Sunstroke and similar conditions produced by exposure to excessive heat. (m) Hysteria.

The Associated Phenomena in Different Morbid States Characterized by Coma.—The diagnosis of the underlying condition is always important, often difficult, sometimes impossible. When the previous history can be obtained from the patient's friends the diagnosis is simplified. A child is seized with convulsions and vomiting and falls presently into coma. The fact that other children in the family suffer from scarlet fever justifies a provisional diagnosis of malignant scarlet fever. A man in middle life complains of headache and becomes comatose, with twitching of the face and general convulsions. Information to the effect that he has had polyuria with low specific gravity, small amounts of albumin, and casts, warrants a diagnosis of uræmia. A girl is found unconscious, pallid, with irregular respiration and occasional twitching of the face or extremities. It is of diagnostic importance to learn that she has been a highly nervous person who has just passed through some emotional stress and that the coma was preceded by tears and outbreaks of laughter—phenomena characteristic of hysteria.

The anamnesis is not always conclusive. It frequently happens that a patient suffering from chronic nephritis becomes comatose from cerebral hemorrhage and that a man who has been drinking falls into a coma not the manifestation of alcoholism but [illegible] of fracture of the skull. The causal diagnosis of coma is attended with increased difficulty in ambulance cases and patients concerning whom no history can be obtained, seen for the first time in a [illegible] condition.

CEREBRAL DISEASE.—Coma occurring in the course of organic disease of the brain is usually preceded by such general symptoms as headache, vomiting, delirium, and stupor, with varied local symptoms which depend upon the position and extent of the lesions and may be either irritative or paralytic.

A[illegible]—[illegible] symptoms are [illegible]

[illegible]

pernicious forms of malarial infection. In the last there is the history of exposure in intensely malarial localities and of one or two recent well characterized paroxysms of ague.

URÆMIA.—Uræmic coma may occur in acute or chronic nephritis.

DIABETES.—In saccharine diabetes coma very often attends the terminal condition, particularly in the young. Three forms of diabetic coma are recognized: (a) The patient after exertion is seized with sudden weakness, syncope, and somnolence which gradually deepens to coma and is followed in a few hours by death. (b) The early symptoms are due to pulmonary or gastric derangement or there may be some local affection, as pharyngitis, phlegmon, or carbuncle. The attack begins with nausea and vomiting. The breath has the peculiar sweetish, fruity odor of acetone. The onset of coma is gradual. Death occurs in the course of one to five days. (c) The patient without special previous symptoms is suddenly seized with violent headache and the sensation of profound illness and rapidly falls into deep and fatal coma. There are cases of diabetes in which the coma is due to some accidental cause, as uræmia, apoplexy, or meningitis.

NARCOTIC POISONING.—In coma from opium and its derivatives the face is pallid, dusky, and slightly cyanotic, respirations and pulse slow, pupils equal and contracted, skin natural, and temperature normal.

In alcoholic coma the face is commonly flushed, sometimes pallid, occasionally cyanotic. The respirations are usually normal in depth and frequency. They are sometimes stertorous. The odor of the breath is characteristic, the pulse is at first frequent and full, later small and feeble. The pupils are equal, sometimes normal, more frequently dilated. The skin is usually cool and moist and the surface temperature lowered, especially under circumstances of exposure to cold or damp, when heat dissipation is favored. Convulsions are not common.

POISONOUS GASES.—Coma develops under circumstances that make the diagnosis clear. It may result from the inhalation of carbon dioxide, as in disused wells, and carbon monoxide—illuminating gas, charcoal fire—a very common cause of accidental death and suicide. There can be no question about the coma of surgical anæsthesia. During this state various accidents may occur. Asphyxia has resulted from the falling back of the base of the tongue and from pulmonary œdema. Progressively deepening coma may terminate in death from failure of the cardiac or respiratory centres, and apoplexy may occur.

CONVULSIONS.—Coma is very often preceded by general convulsions or alternates with them. It is frequently preceded by convulsions in the malignant forms of the infectious diseases, especially in children, and sometimes in dentition and the digestive disorders in young infants—*infantile eclampsia.* It follows the general convulsions of epilepsy. Coma and convulsions may alternate in cerebral syphilis, general paresis, and some forms of alcoholism. The alternation of coma and convulsions is characteristic of uræmia.

The coma of sunstroke is very often preceded by convulsions. The skin is excessively hot and dry, the face flushed, the respiration labored, the pulse frequent and full. The temperature ranges extremely high and may become that of hyperpyrexia. Upon venesection the blood is dark, thick, and flows slowly from the vein. The diagnosis is usually easy.

Epilepsy.—The diagnosis of postepileptic coma rests upon the history of the case, the convulsive seizure, the bitten tongue, the foam upon the lips, and the sudden profound loss of consciousness of no very long duration. The congestion of the face, stertorous breathing, urinary incontinence, and general muscular relaxation may suggest apoplexy, but the signs of hemiplegia are lacking.

Hysteria.—The unconsciousness of hysteria is commonly incomplete —lethargy or its intensification, trance. Its duration may extend over several days or weeks. True hysterical coma which is a further intensification of the foregoing is very rare. A condition of impaired consciousness suggestive of coma not infrequently enters into the symptom-complex in the grand attack of hysteria. It is usually preceded by the ordinary phenomena of the hysterical paroxysm: laughing, crying, convulsions, extravagant muscular movements, and the like.

For practical purposes the differential diagnosis between the coma resulting from opium, traumatism, alcohol, apoplexy, and uræmia is of imperative importance. Only in a correct diagnosis are to be found the indications for treatment. These are often immediate and urgent. Furthermore the diagnosis may have to do with questions of medico-legal interest. Definite diagnostic phenomena are to be systematically sought for.

Such points in the anamnesis as are available are to be obtained from the patient's friends or the bystanders. The immediate investigation demands an examination of the scalp and head for evidences of traumatism; of the eyes with reference to pupillary conditions and reactions, strabismus, and conjugate deviation; the face for blood extravasations, flushing, pallor, cyanosis, œdema, puffing of the cheeks, the presence of foam upon the lips, a bitten tongue, relaxation of the jaw, the odor of the breath, and the presence upon the lips or face of the stains of corrosive or other poisons. The character of the respiration is to be studied, the frequency, volume, and tension of the pulse, the sounds of the heart. The occurrence of fecal or urinary incontinence is to be noted, catheterization should be performed, and the urine examined for the presence of albumin, blood, sugar, acetone, etc. The signs of hemiplegia are to be sought in the position of the head and eyes—conjugate deviation—in the greater relaxation of the mouth and cheek upon one side and the complete loss of muscular tonus in the arm and leg. The temperature must be taken in the axilla, and if found to be very low, in the rectum also. The signs of antecedent disease, general anasarca, great emaciation, various specific and other eruptions and scars, and the general condition of the viscera as determined by the methods of physical examination, such as the presence of effusions in the serous sacs, great enlargement of the liver or spleen, and the like, are to be in turn rapidly investigated. The stomach pump is often necessary for the diagnosis. If the conditions suggest the possibility of pernicious malarial fever an examination of the blood should be made for Laveran's bodies.

Not every case demands such comprehensive and elaborate investigation. Very often the condition underlying the coma is obvious at a glance. In other cases it is speedily revealed. Once in a while the true condition is not discovered without careful and prolonged study, and there are obscure cases which tax the resources of clinical medicine.

INSOMNIA AND OTHER DISORDERS OF SLEEP.

Insomnia—Abnormal Wakefulness.—These terms are used to designate a disturbance of the nervous system characterized by habitual incomplete sleep or periods of entire absence of normal sleep. Sleep varies with age, sex, and individual peculiarity. In very young babies sleep is practically continuous; a healthy child two years old passes half its time in slumber; the adult requires from seven to eight hours out of twenty-four; and aged persons not more than five or six hours. Women need more sleep than men. Workers in the open air require longer hours of sleep than those of sedentary habits. Insomnia may be functional or symptomatic. Functional insomnia occurs in neurotic individuals and over-taxed brain workers. Symptomatic insomnia is an important element in the symptom-complex of a great variety of morbid conditions. It occurs in painful diseases, as cancer, aneurism, and the intractable neuralgias. It is common in acromegaly. Insomnia is a very troublesome symptom in neurasthenia and various forms of insanity. It is an important element in acute delirium. Advanced disease of the heart is very often attended by sleeplessness due in part to cerebral anæmia, in part to the condition of the blood, but chiefly to the inability of the patient to lie down. As the condition progresses wakefulness gives way to somnolence and stupor. Tea and coffee have in many persons the power of inhibiting sleep. Complete insomnia is a conspicuous phenomenon in delirium tremens and alcoholic mania. Insomnia occurs with some degree of frequency also in the early stage of enteric fever, influenza, and croupous pneumonia. It is met with in cases of malaria and is a troublesome symptom in trichiniasis. It is not uncommon during the convalescence from acute disease. Insomnia may take the form of troubled and unrefreshing sleep of short duration or broken by intervals of distressing wakefulness, or sleep may be absent for days together. The patient may fall asleep upon going to bed but awakes in the course of two or three hours and lies absolutely awake or lightly dozes until morning. There is often great and irregular mental activity, especially in neurasthenia, and the cares, anxieties, and worries of the day are rehearsed with torturing iteration. Such insomnia is associated with restlessness, which is also present in the insomnia of insanity. Insomnia is rare in children but when present significant of profound disturbance of the nervous system. The sleeplessness of the aged is usually tranquil and unaccompanied by excitement or irritability.

Dreams usually have their starting-point in some sensory impression arising from local causes, as an uncomfortable posture, a sound which is perceived but which does not arouse, an over loaded stomach, a distended bladder or rectum, or a condition which interferes with the action of the heart and lungs. *Nightmare* is a frightful dream accompanied by sensations of oppressive weight upon the chest, intense fear, horror, or anxiety, and inability to move or cry out. The attack ends in a groan and the recovery of consciousness. It is mostly symptomatic of indigestion.

Night Terrors—Pavor Nocturnus.—This condition, which presents points of resemblance to nightmare and somnambulism, is a paroxysmal disturbance of sleep in young children. It differs from nightmare in the gradual subsidence of the attack and the persistence of terror and distress after waking. It differs from somnambulism in the gradual waking, the less complete automatism, and the terror. The child starts up in bed screaming with fear and seeks protection, trembling and sobbing. The dream images are often indefinite, sometimes the creatures of imagination, suggested by the tales of the nursery. Night terrors occur commonly in neurotic and badly nourished children. They are sometimes symptomatic of eye-strain, the cutting of the second teeth, intestinal parasites, or indigestion.

Sleep drunkenness is a rare condition resembling maniacal delirium which appears upon waking from profound sleep. There are delusions of immediate danger to life or liberty. The sufferer fails to recognize his surroundings. He is excited, incoherent, and boisterous. The attack is usually of short duration.

Somnambulism—sleep-walking—is a disorder of sleep in which consciousness and volition are suspended but the activity of certain nerve-centres is exerted and coördinated movements are automatically performed. It occurs in adolescents and young adults of neurotic temperament and is more common in females. It is due to causes which ordinarily give rise to dreams, including indigestion, faulty attitude during sleep, intense excitement, or violent distressing emotion during the period preceding sleep. The attacks are frequently recurrent and may become habitual. They are of brief duration but may continue an hour or two, during which time difficult and complicated actions are performed, apparently with conscious intention. The eyes are closed or, if open, are staring and fixed. There is complete indifference to sound and the expression is blank and impassive. The patient on waking has no recollection of his wanderings.

Morbid Sleep.—Drowsiness may be symptomatic of cerebral malnutrition or toxæmia. It is common in aged persons with feeble heart and diseased blood-vessels, in the obese, and in malaria, anæmia, and diabetes. It is caused by the impure air of crowded assemblies. Cases have been reported in which prolonged deep sleep has ceased after the discharge of lumbricoid worms. Morbid sleep is a symptom by no means uncommon in organic cerebral disease, as syphilis, tumor, and arteriosclerosis. It is common in insanity, both in the prodromal period and the developed state.

Narcolepsy is abnormal deep sleep occurring in spells which may be of short duration or prolonged and continuous. The cause is unknown. In some instances the sleep has progressively advanced to deep and fatal coma.

Waking numbness—sleep palsy.—This is a form of paræsthesia occurring upon waking. There is a sensation of numbness and tingling. The distribution involves one or more extremities, usually the hands and arms. It is commonly of brief duration, disappearing in an hour or two. It resembles the forms of paræsthesia which occur about the grand climacteric.

Paroxysmal disturbances of the nervous system, both physiological and pathological, are common during sleep. Seminal emissions, the venereal orgasm, and urinary incontinence are accidents of sleep. Epileptic seizures—nocturnal epilepsy—are not uncommon, and the paroxysms of asthma and migraine frequently come on in sleep.

PART IV.

OF THE CLINICAL APPLICATIONS.

I.

DIAGNOSIS OF THE SPECIFIC INFECTIONS.

I. ENTERIC OR TYPHOID FEVER.

Definition. — An acute general infection caused by the Bacillus typhosus, characterized clinically by fever of prolonged duration, a scanty rose-colored, maculopapular eruption, enlargement of the spleen, abdominal tenderness, diarrhœa and tympanites, profound asthenia, and rapid wasting; anatomically by hyperplasia and ulceration of the lymph follicles of the intestines, enlargement of the mesenteric glands, and diffuse parenchymatous changes in the viscera.

Etiology. — **Predisposing Influences.** — Enteric fever is the prevalent febrile infection of the present historical epoch, just as typhus was that of the three hundred years preceding the beginning of the nineteenth century and the plague that of the Middle Ages. It owes its present wide distribution and great prevalence to faulty disposal of sewage and neglect of the simplest sanitary laws. Defective drainage and contaminated water and milk constitute the means by which the specific infecting organisms are distributed. CLIMATE.—Enteric fever prevails in all parts of the world but is especially common in temperate climates. SEASON.—It is most common in the autumn months but occurs at all seasons of the year. Of personal causes: SEX.—Males and females are equally liable to the disease. The mode of life of the individual exposes males to the infection to a greater extent than females. In early childhood the sexes suffer alike; in late childhood and adolescence, boys more than girls. More men than women are admitted to hospitals. The conditions of camp life in military campaigns especially favor the spread of the infection. In the Spanish-American and South African wars enteric fever prevailed most disastrously. AGE. — Enteric fever is especially a disease of youth and early adult life. The period of greatest liability is from the fifteenth to the twenty-fifth year. Exposure and immunity are to be considered in this connection. Exposure to the infection is probably greater after adolescence and the immunity acquired by the attack more general after the twenty-fifth year. Cases occasionally occur after sixty. OCCUPATION AND SOCIAL STATE.—These conditions are without predisposing influence. IMMUNITY.—Not all who are exposed contract the disease. An unrecognized or forgotten attack in childhood may have conferred immunity. There are families which show in successive generations an especial susceptibility. The immunity acquired by the attack is usually life-long. Second attacks have occurred in the course of several months or years and three attacks in the same person have been noted by competent observers.

The Exciting Cause.—BACILLUS TYPHOSUS.—This organism, the bacillus of Eberth, is constantly present in enteric fever. It is a short, thick, flagellate, motile bacillus with rounded ends, growing readily on various culture media. It can now be differentiated from certain other bacteria to which it bears a close morphological resemblance, especially the bacillus coli communis, with which it is liable to be confounded. These organisms colonize with preference in the lymphoid tissues. They are distributed in recent infection in the solitary follicles and Peyer's patches of the intestines, in the mesenteric glands, spleen, bone-marrow, liver, and in the bile. They have been isolated from foci of suppuration in various parts of the body, from meningeal and pleural exudates and vegetations upon the endocardium. Their presence in the blood and rose spots may be demonstrated. In the course of the second week and afterwards they have been isolated by culture methods from the stools. They are met with in the urine and sputum and have in a few instances been found in the sweat.

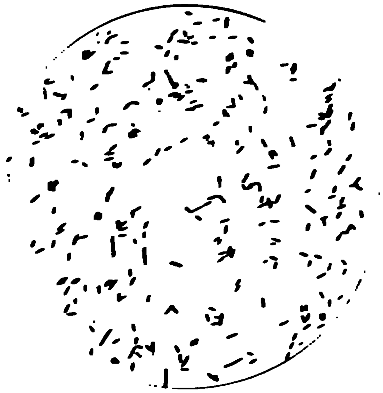


Fig. [illegible].—Bacillus typhosus.

Outside the body typhoid bacilli retain their vitality in water, snow, ice, the superficial layers of the soil, dust, and in fæces for periods varying according to circumstances from several days to many months. In milk they undergo rapid growth without changing its appearance. They retain their vitality for three months in sour milk and for shorter periods in butter made from infected cream.

The above facts are of great use in the etiological diagnosis of enteric fever, especially when the disease occurs in local outbreaks. It is most important in this connection that the infecting principle is discharged from the body of the patient in the urine and especially in the fæces. Its conveyance by means of water, milk, or other articles of food contaminated by such discharges in consequence of faulty sanitation is the source of the sporadic, endemic, and epidemic prevalence of the disease. It may be stated positively that enteric fever is not contagious in the ordinary sense, *i.e.*, transmissible from the sick to others by contact or by the exhalations from the body, when the introduction of bacilli-containing dejecta or secretions does not take place.

Infected water is the ordinary means of transmission. By means of such water the infection of milk, uncooked vegetables, salads, oysters, and clams occurs. It has recently been shown that lobsters also may become infected when kept in cages in sewage-contaminated water awaiting a market. Natural ice and vegetables grown upon soil fertilized by sewage, eaten raw, may convey the germs. Flies and atmospheric dust play an important part in the dissemination of the disease. The open latrines on the one hand and the unscreened mess table on the other were largely responsible for the terrible epidemics among our recruits in the practice camps at the time of the Spanish-American War.

When the bacilli find their way into the intestinal tract the evolution of the disease is as follows: Being resistant to dilute acids they are by no means wholly destroyed by the hydrochloric acid of the gastric secretion. If acid be absent or the water or other fluid containing them be ingested when hydrochloric acid is not secreted they pass into the intestine and colonize and multiply in the lymph structures, finding their way into the mesenteric glands and thence by means of the blood to the spleen, liver, kidneys, and bone-marrow. In these locations and elsewhere they form soluble toxins which, circulating in the blood, exert their influence especially upon the nervous system and the nutrition and thus give rise to the fever and other constitutional symptoms. Brieger isolated from cultures a toxin belonging to the ptomaine group to which he gave the name typhotoxin. Later with Fraenkel he isolated a toxalbumin. Of greater importance is the poison derived from the bacilli—protein toxin—by R. Pfeiffer.

Period of Incubation.—The duration of the time from the entrance of the pathogenic principle until the appearance of the phenomena of the fever varies upon the average between two and three weeks. This period runs its course without symptoms, provided prodromes are not included.

Stage of Prodromes.—The onset is very rarely abrupt. It is as a rule preceded by a period of impaired health characterized by malaise, feebleness, indisposition to bodily or mental effort, loss of appetite, headache, vertigo, and disturbed sleep. Abdominal uneasiness, even pain, and diarrhœa in the absence of laxatives are often present at this time. Slight but transient rises of temperature may occur. Bleeding at the nose is common.

The course of the attack varies between twenty-one and twenty-eight days. It may be divided into the stage of onset, the fastigium or fully developed disease, and the stage of decline upon which supervenes the convalescence. As, however, there are anatomical changes in the lymphoid structures of the intestines which run their successive courses in periods of five to seven days and correspond to changes in the symptom-complex of similar duration, it is convenient for purposes of description to divide the course of the attack into four periods of a week each.

Course of the Disease.—**First Week.**—The attack of enteric fever begins with a distinct and sustained elevation of temperature. This rise is frequently attended with chilliness, which may be repeated, but rarely by a pronounced chill. The patient now in the majority of instances betakes himself to his bed. During the first four or five days the temperature rises in the evening from one to two degrees higher than upon the previous evening and each morning a degree or more above that of the preceding morning. At the end of this time the temperature has reached its fastigium, 103°-105° F. (39.5°-40.5° C.), and with slight morning remissions remains, in the absence of complications, at this level until the end of the second week. During this period there are lassitude, headache, anorexia, thirst, a hot, dry skin, diminished urine, and restless sleep. The headache becomes progressively more severe and is attended with tinnitus aurium and delirium, at first mild and present usually upon waking. Epistaxis frequently occurs. It is usually slight but may be free. The tongue is coated and is seen to be of a bright red color at the edges and tip—"red tongue fever." At this time constipation is the rule but laxatives act with unusual energy. Toward the end of the first week spontaneous diarrhœa often occurs. There are cases, however, in which constipation continues throughout the attack. The spleen is found upon palpation to be enlarged toward the end of this period; there is slight tympanitic distention of the abdomen and tenderness in the ileocæcal region. The gurgling upon pressure, often observed in this region, is wholly without diagnostic value.

A few scattered, medium-sized, dry râles may usually be heard upon auscultation of the lungs. The pulse is rapid, 90–110, but less so in proportion to the rise of temperature than in many other acute diseases. It is full in volume, of low tension, and often dicrotic.

Second Week.—The fever now assumes the subcontinuous type, the range between the evening rises and morning remissions not greatly exceeding those of health. The symptoms become progressively more severe. The pulse is rapid and gradually loses its dicrotism. About the tenth day the headache spontaneously ceases and is replaced by somnolence and stupor, which alternate with delirium usually wandering but sometimes noisy and active. The facies is dull, faintly flushed, sometimes slightly cyanotic. The lips and tongue are dry and there is a tendency for sordes to collect upon the teeth and gums. The abdominal symptoms, diarrhœa and tympanites, are aggravated; there is decided enlargement of the spleen and between the sixth and tenth days the eruption makes its appearance, commonly upon the upper abdominal or lower thoracic regions anteriorly. Traces of albumin are now to be found in many of the cases. The signs of bronchitis are more pronounced. Fine subcrepitant and crepitant râles are heard at the bases posteriorly and slight dulness upon percussion may be found in this region.

Third Week.—The temperature assumes the remittent type showing morning remissions of increasing length. The pulse becomes more feeble and frequent, 110–140. The first sound of the heart is faint and may be inaudible. There is muscular tremor. Diarrhœa and tympanitic distention of the abdomen may increase or, if previously absent, now appear. There is often retention of urine and sometimes involuntary discharges of urine and fæces. Weakness is most marked and wasting conspicuous. Stupor and delirium continue. The rash, which has continued to appear and fade in successive crops, now as a rule gradually diminishes and does not in most cases again appear. This is especially the period of severe complications, hypostatic pneumonias, bed-sores, parotitis, hemorrhages, and perforation.

Fourth Week.—The type of the fever is now intermittent, the morning remissions gradually falling to normal or slightly subnormal ranges and the evening rises progressively diminishing until they no longer transcend the normal. The tongue becomes clean and moist, the diarrhœa ceases, and there is a rapidly increasing and urgent desire for food. The spleen undergoes involution. The tympanites subsides. The pulse becomes stronger and fuller and the first sound and impulse of the heart more distinct. The convalescence may be postponed by the occurrence of various sequels or by relapse, and in some instances the symptoms of the fourth or even of the fifth week may continue to be the same which were present during the third—progressive asthenia, rapid and feeble pulse, abdominal distention, involuntary discharges, dry tongue, muttering delirium, stupor, and subsultus.

The foregoing sketch represents a severe attack of enteric fever unmodified by treatment and terminating in recovery. But from this typical picture of the disease there are many variations. The problem in diagnosis is to determine not the nature of a well-defined typical case of enteric fever—that any tyro in medicine can readily do—but to recognize the atypical cases and to differentiate them from the affections to which they present features of resemblance and to do this successfully at the earliest possible moment, since failure in this respect leads to indifference in regard to the search for the cause of the attack on the one hand and neglect in the proper treatment and disposal of the evacuations on the other.

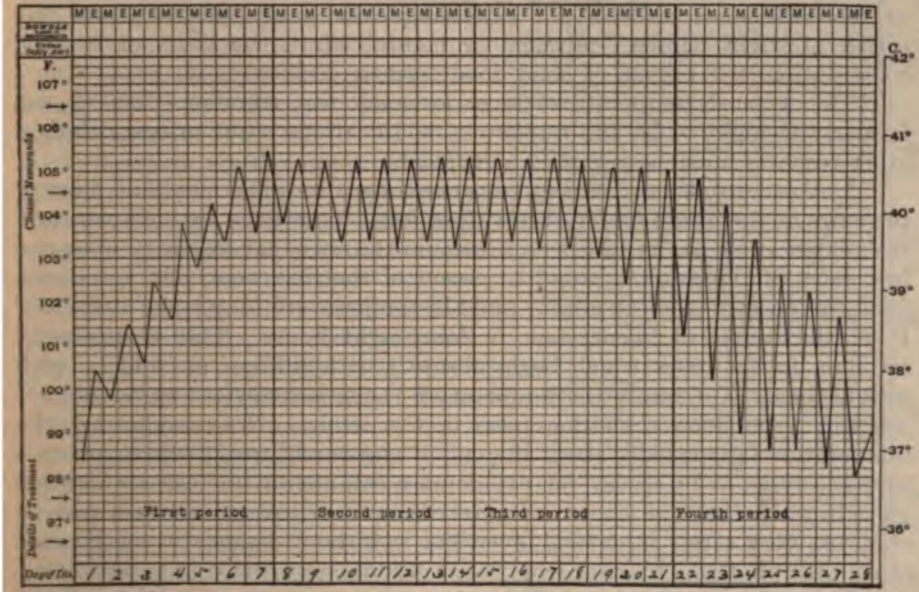


FIG. 207.—Temperature range in enteric fever.—Wunderlich.

Symptoms of Especial Importance in Diagnosis.—Before entering upon the consideration of the varieties of enteric fever it is important to give our attention in greater detail to certain of the principal symptoms the association of which is of cardinal importance in diagnosis. These symptoms relate to (a) the fever, (b) the pulse, (c) the rash, (d) the abdominal organs, and (e) the nervous system.

(a) **The Fever.**—1. REGULAR, SO-CALLED TYPICAL COURSE.—The temperature rises by regular step-like gradations, with marked evening exacerbations and slight morning remissions, until it reaches a range of

103°-105° F. (39.5°-40.5° C.) by the fourth or fifth day. From this period to the end of the second week its range is subcontinuous, the oscillations between the morning minima and the evening maxima but little exceeding those of health. During this time the temperature is scarcely modified by ordinary antipyretic measures and only gradually yields to systematic cold bathing. Toward the end of the second and throughout the third week the type becomes remittent with diurnal oscillations of gradually increasing length until in the fourth week the type is distinctly intermittent, the diurnal oscillations progressively diminishing at the expense of the evening rises until subnormal ranges are reached. The defervescence is by lysis. From this level the temperature again in the course of a few days rises to normal, but it remains for a time unstable and is liable to recrudescences of 1°-3° F., extending over a day or two, from the action of slight causes,—physical or mental effort, the visits of friends, constipation, the eating of solid food,—*febris carnis.*

This typical temperature range is much less common than it was formerly thought to be and numerous modifications occur in cases that in other respects must be regarded as typical.

2. Variations in the Temperature Range.—The fact that most of the cases do not come under observation until some days have elapsed and the temperature has attained its fastigium throws some obscurity upon the frequency of the gradual step-like ascent of the first week. In cases seen from the onset it is often absent and the temperature may reach 104°-105° F. in the course of twenty-four or forty-eight hours. This is especially common in the cases which begin abruptly with chills or in children with convulsions. Not rarely the temperature range is remittent throughout—infantile type, seen sometimes also in the adult. Not very rarely the temperature falls rapidly about the end of the second week,—defervescence by rapid lysis, or by crisis,—an event more common in the cases which begin abruptly than in those of gradual onset. Inverse temperature is observed very rarely in enteric fever and is without diagnostic significance other than that which arises from its occasional occurrence in tuberculosis.

The course of the temperature is sometimes interrupted by sudden falls. These declines may amount to 8° or 10° F. in the course of a few hours. They occur from hemorrhage from any cause and especially accompany intestinal hemorrhage. As a rule they are followed in the course of several hours, when the bleeding has ceased, by a rise to the former range, but the temperature often remains unstable; exceptionally the temperature stays low and the patient enters upon an early convalescence. Hyperpyrexia is not common in enteric fever. In very rare instances collapse may occur in the absence of hemorrhage or perforation.

Recrudescences of fever from trifling causes, occurring as a manifestation of the instability of the heat-regulating mechanism which follows the infection, are of no great importance. They are, however, to be differentiated from the symptomatic fever of an inflammatory complication, as pneumonia or pleurisy or venous thrombosis. In this connection the local phenomena and an increase of the leucocytes are of importance.

Subfebrile States in Convalescence.—In children and neurotic individuals there may be evening fever, 100.5°-102° F. (38°-39° C.), for weeks after the symptoms of the disease have disappeared and convalescence is in other respects fully established. I have several times seen this condition in laboring men in hospital wards. It has been described as bed-fever. It disappears when the patient is allowed to sit up. A similar evening rise may be the sign of a latent complication.

The subnormal temperature of early convalescence is not important. It is especially liable to occur in feeble or greatly emaciated individuals but may be encountered in persons making a good recovery. In the course of a week or ten days the temperature rises to normal and regains its stability. The hypothermia of hemorrhage and the morning remissions of the later stages have been already described. That of cold bathing and other antipyretic treatment is transient and without diagnostic importance.

Relapses are characterized by a febrile range like that of the primary attack, except that the respective periods are shorter just as the relapse is shorter—ten days to two weeks. The gradual ascent, subcontinuous fastigium, and defervescence by lysis with remittent and intermittent curves are important from the standpoint of diagnosis.

Afebrile Typhoid.—Cases have been described in which the general constitutional symptoms and the duration of the case together with the eruption, enlargement of the spleen, dicrotism, and the diazo reaction have been present without fever or at most with only a trifling elevation of the evening temperature to subnormal ranges, and in which the etiology of the disease and the existence of a local epidemic have rendered the diagnosis of enteric fever in the highest degree probable. The diagnosis will become positive in the event of the occurrence of intestinal hemorrhage or upon post-mortem examination resulting in the demonstration of the characteristic intestinal lesions. In the absence of those events the diagnosis may then be made by the finding of the bacilli in the urine and stools or in blood cultures.

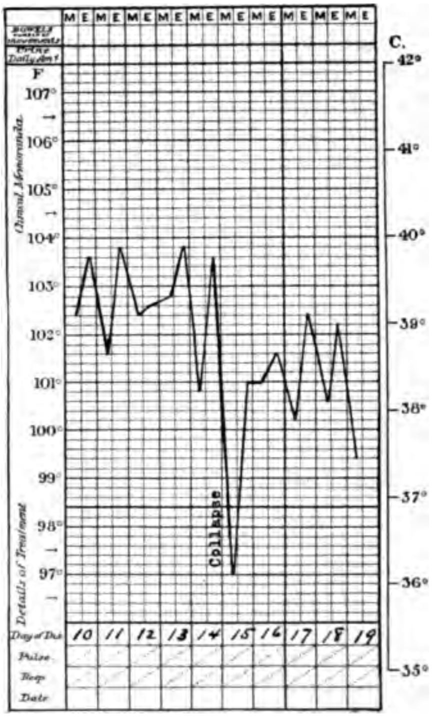


FIG. 208.—Collapse in enteric fever, 14th day. No hemorrhage. Ultimate recovery.

3. CHILLS.—Chilliness is not uncommon in the period of onset, but rigors are rare in enteric fever. Chills occasionally, however, occur with the initial rise of temperature; at irregular intervals during the course of the attack, followed by profuse sweating; sometimes upon the development of complications; after the administration of internal antipyretics, and in septic conditions. Chilliness and shivering are frequent at the end of tub-baths.

(b) **The Pulse.**—The characters and frequency of the pulse in relation to the fever are of diagnostic importance. The pulse is commonly full and of low tension in the beginning of the attack. Dicrotism appears early and is not only more common but also more marked than in any other acute disease. The frequency is increased but not proportionately to the elevation of the temperature. A pulse of 100–120 may within the second week be associated with a temperature of 104°–105° F. (40°–40.5° C.). Later the pulse becomes more frequent and feeble and its volume is much diminished. In grave cases with great prostration it may reach 160 and such a degree of enfeeblement as to be scarcely countable.

(c) **The Rash.**—The eruption occurs in at least four-fifths of the cases. It consists of lenticular, hyperæmic papules, slightly elevated above the surface of the skin, of a pale rose color, one to four mm. in diameter and disappearing upon pressure or when the skin is made tense. These roseolous maculopapules may be felt by the finger. They must not be confounded with the dense papules or small pustules of acne which are common upon the trunk, especially the back, of young persons. They appear in successive crops and vary in number from two or three, found only upon careful search, to a somewhat copious roseola distributed upon the surface of the trunk and extremities. Contrary to the general opinion, I am now of the belief that cases characterized by an abundant rash frequently run a severe course. The eruption first makes its appearance between the end of the first and the middle of the second week and commonly in the epigastric zone upon the anterior surface of the body. It is also frequently noted upon the back, between the shoulder-blades. It may in rare instances be seen upon the face, especially in young persons of fair skin. The spots are circular or oval with well-defined borders. They gradually fade in two or three days, leaving an area of pigmentation the degree of which varies according to the complexion of the individual. In the majority of cases no new crops appear after the defervescence begins, but exceptionally the spots continue to appear after the temperature has fallen to normal.

Other points of value in diagnosis in connection with the skin are the out-cropping of sudamina as the fever begins to decline; the occasional occurrence of purpura; an infrequent erythematous eruption at the outset, resembling that of scarlet fever; a fine branny desquamation in children; the presence of the *tache cérébrale*, and the great infrequency of herpes labialis in comparison with malaria, croupous pneumonia, and cerebrospinal fever.

(d) **The Symptoms Relating to the Abdominal Organs.**—Splenic enlargement may be made out upon palpation, the border of the organ extending below the ribs, especially on deep inspiration. The results of percussion are obscured by the meteorism which is common after the beginning of the second week. A splenic tumor is demonstrable in more than eighty per cent. of the cases.

Diarrhœa is a variable symptom. It is present at some time in the course of the majority of the cases, often alternating with constipation. There are, however, epidemics in which constipation throughout is the rule. Diarrhœa is more common in the later course of the attack. It is caused by the associated catarrh rather than by the ulcers and is indicative of extensive rather than of deep ulceration. The number of the stools varies from two or three to eight or ten in twenty-four hours. They are usually large, thin, grayish-yellow in color and of a granular composition. They very often contain one or more soft scybalous masses of the size of a walnut. The reaction is alkaline and the odor foul. On standing, the fluid and solid constituents separate into two layers, the upper containing albumin, salts, and coloring matter, the lower epithelial débris, cellular elements, fat crystals, triple phosphates, and later in the disease sloughs from the necrotic Peyer's patches, and microscopic blood. In many cases the bacilli of Eberth may be found after the middle of the second week. This separation of the stools into layers is not often seen in other forms of diarrhœa, but cannot be regarded as pathognomonic. In truth it cannot be said that the stools of enteric fever are characteristic in any diagnostic sense. The familiar comparison with pea-soup is inexact and misleading and might well be discarded with a multitude of other unscientific, traditional, false phrases from the language of descriptive medicine.

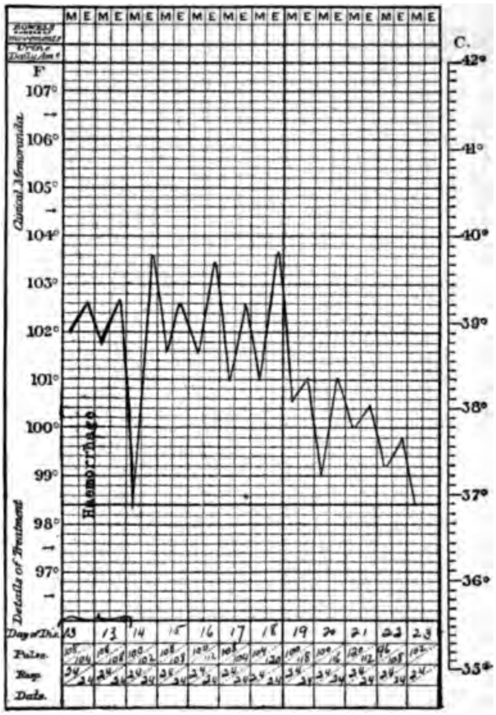
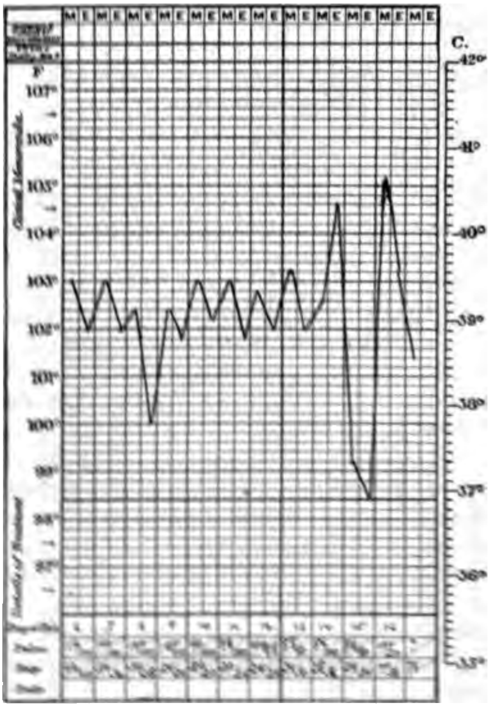


FIG. 209.—Enteric Fever. Hemorrhage on the 13th day; recovery. Woman, aged 23.

TENDERNESS AND PAIN.—Tenderness in the ileocæcal region is common in the early course of the attack and important in the differential diagnosis between enteric fever and appendicitis. The tenderness is in some instances confined to the umbilical region; less often it may be elicited upon pressure over almost any part of the abdomen. Tenderness and pain in the abdomen may be symptomatic of an over-distended bladder, pleurisy, crural phlebitis, or other acute affection. In a large proportion of the cases no definite cause for these symptoms can be discovered. Pain and tenderness are occasionally associated with intestinal hemorrhage but are present and intense in almost all cases of perforation.

INTESTINAL HEMORRHAGE.—Hemorrhage from the bowel is a serious accident, occurring in from three to ten per cent. of all cases. It varies in amount from a trace of blood in the stools to a profuse and fatal blood loss. Large hemorrhages most commonly occur about the time of the separation of the sloughs, namely, between the close of the second and the beginning of the fourth week. The slighter hemorrhages which take place earlier than this period are due to oozing from the hyperæmic Peyer's patches; those which occur after it are to be ascribed to the mechanical disturbance of unhealed ulcers by the peristaltic movement of the bowel and the contact of the intestinal contents. Intestinal hemorrhage usually comes on without premonitory symptoms. It may reveal itself at once by the discharge of blood from the anus, with faintness, feeble pulse, pallor, and a rapid fall of temperature to the extent of several degrees, or by collapse symptoms which may terminate in death before the blood appears in the stools—concealed hemorrhage. Intestinal bleeding may be symptomatic of a general hemorrhagic condition manifested also by petechiæ and hæmaturia or oozing from other mucous surfaces.



[illegible]

PERFORATION.—Intestinal perforation is the gravest accident that occurs in enteric fever. Its frequency is about three per cent. of all cases. It may happen in otherwise apparently mild cases but is more common in severe cases in which diarrhœa and meteorism are marked or in which hemorrhage has occurred. Nearly fifty per cent. of the cases occur in the third or fourth week. The symptoms are first those caused by the perforation itself and the escape of the contents of the bowel into the peritoneum, and second, those of the resulting peritonitis. Of the first group, sudden, sharp pain in the right lower quadrant of the abdomen, increasing in severity, attended by general [illegible] tenderness, is the most [illegible]. Next in order of [illegible] which become spastic [illegible] micturition are [illegible] fall in temper[illegible] surface with sweat[illegible] perforation may [illegible]

[illegible] consecutive peri[illegible] save the life of the patient [illegible] account of muscular rigidity [illegible] nature of the respiratory [illegible] liver and splenic dulness [illegible] cavity, fulness and tenderness upon digital examination per rectum; absence of the peristaltic murmur upon auscultation, ·friction sounds in the upper hepatic area or elsewhere, and in the majority of the cases a rapidly progressive leucocytosis. These phenomena are not all present in every case but the presence of several of them justifies the diagnosis of typhoid peritonitis in its early stage. Their progressive development is soon followed by the occurrence of more ominous signs. The spastic contraction of the muscles of the abdominal wall is replaced by tympanitic distention which progressively increases. Percussion in the flanks reveals flatness in the most dependent region, the sign of an accumulating exudate. The pain is less urgent; the tenderness less acute. Further displacement of the liver and splenic dulness in an upward direction is noted and the impulse of the heart may be found as high as the fourth interspace. In a small number of the cases perforation and peritonitis occur with a flat or even retracted abdomen.

The general symptoms indicate the gravity of the condition. They consist of pallor, an expression indicative of pain, which develops later into the facies Hippocratica, profuse clammy sweating, a feeble, thready, frequent pulse, hiccough, and vomiting. The respiration is shallow, the heart sounds indistinct, the temperature, which may have fallen upon the occurrence of perforation, rises again, and the kidneys secrete little or no urine. This formidable array of symptoms, the forerunners of the approaching fatal issue, may be masked by those of an antecedent overwhelming toxæmia.

Perforative peritonitis without the escape of air into the peritoneal sac may arise from the rupture of a pseudo-abscess, softened mesenteric gland, or a splenic or hepatic abscess. A general or local peritonitis may occur in consequence of infection through the thinned base of a deep ulcer without rupture.

(e) **The Nervous System.**—There is nothing characteristic in the nervous symptoms of enteric fever; yet their association with the phenomena mentioned in the foregoing pages constitutes a symptom-complex that is in the highest degree significant.

Headache, sleeplessness, and a condition of the nervous system which renders physical and mental effort alike difficult characterize the period of prodromes. The headache may involve any part of the head or be general. Its most constant peculiarity is that it becomes worse toward night. It usually ceases spontaneously about the middle of the second week and is replaced by somnolence and stupor—a change which is of diagnostic value. Tinnitus aurium frequently accompanies the headache and deafness develops with the progress of the disease.

Delirium sometimes begins early and is usually of mild character. The patient can be roused, his attention fixed and his replies become apparently rational. The delirium is at first nocturnal. Later it becomes continuous and marked. It may not appear until the second or even the third week. In other cases it is noisy and restless and attended with efforts to get out of bed. In hard drinkers it may be associated with tremor and the hallucinations peculiar to delirium tremens. Abrupt changes in the form of delirium occur. The patient who has been apathetic and wandering may without warning develop an active and purposeful delirium with suicidal tendencies. The enteric fever patient who has become delirious must under no circumstances be left alone. Somnolence and hebetude are common and in the severe cases these symptoms deepen into a stupor from which the patient cannot be roused. Coma vigil, tremor, subsultus tendinum, and carphologia are among the most ominous symptoms of the disease.

Convulsions are rare. They may occur in children at the onset. In the course of the attack and especially in adults they may be hysterical, uræmic or symptomatic of some nervous complication, as encephalitis or thrombosis of cerebral arteries or veins.

Varieties.—Enteric fever is in the strictest sense a disease of the whole organism. Scarcely an organ escapes; not a function goes on normally. The duration of the attack with profound derangements of nutrition exposes the defenceless tissues to the secondary invasions of pathogenic organisms both local and general. In different cases various organs bear the brunt of the attack according to the personal predisposition of the individual. Variations in the intensity of the infection,—virulence of the bacilli,—the resistance of the individual,—integrity of the tissues, degrees of hereditary or acquired immunity or predisposition,—complications, sequels, relapse, the management of the case and the surroundings likewise modify the severity of the attack. Hence a most diverse and complex symptomatology. A full account of enteric fever in all its relations would constitute an epitome of the Practice of Medicine. It is important to remember that the disease presents the widest variations from a typical course alike in its mode of onset, its intensity, the prominence of certain symptoms, and in its duration. Errors in diagnosis in doubtful cases are to be avoided only by the routine employment of every resource of clinical medicine with due regard to the teachings of the clinical laboratory and the post-mortem room. From the standpoint of the infection the following varieties may be recognized:

(a) **Ordinary Form with Well-developed Intestinal Lesions.**—This group includes the great majority of the cases. The lesions of the lymph structures are well marked and more or less extensive; the mesenteric glands and spleen are enlarged, parenchymatous changes are present in the viscera. The anatomical diagnosis can be made in the absence of a history of the symptoms.

(b) **Cases Characterized by Slight Intestinal Lesions.**—The changes in the lymphoid structures of the intestines may be superficial and limited in extent and therefore readily overlooked, or if death has occurred late in the attack the ulceration may have already healed. The symptom-complex may be that of an ordinary attack with or without mild intestinal symptoms, of a general sepsis with high fever and marked nervous symptoms,—so-called typhoid state—typhoid septicæmia,—or of an affection of one or more viscera with profound constitutional disturbance. The organs especially involved may be the liver, gall-bladder, lungs, pleura, kidneys, endocardium, or meninges. It is in the last group that the cases are found which are described as pneumotyphus, in which the attack sets in with pulmonary symptoms; pleurotyphus, beginning with an acute pleurisy; nephrotyphus, in which the general symptoms and urinary findings of an acute nephritis are present at the onset, and the cerebrospinal form in which the attack begins suddenly with urgent symptoms of disturbance of the nervous system. In all of these unusual forms the symptoms which dominate the clinical picture at the onset shortly, and mostly in the course of the first week, become subordinate to those characteristic of enteric fever and the attack generally runs its subsequent course in the usual way and time. In many of these cases, however, the intestinal lesions are well developed. To elevate these groups of cases into separate varieties is to increase the difficulties of the student and teacher alike without any compensatory advantages of classification.

(c) **Cases Characterized by the Absence of Intestinal Lesions.**—In some of the cases the true nature of the affection has not been demonstrated; in others lesions such as those of tuberculous ulceration, by which the bacilli of Eberth may have found access, were present. There remain, however, a limited number of cases in which the Bacillus typhosus has been demonstrated in the organs, the symptoms have been characteristic, and death has occurred at a time when the lesions of the gut are commonly conspicuous, yet none have been discovered. The possibility that the bacilli have found entrance by way of the intestinal wall without giving rise to demonstrable lesions has been suggested. Infection by way of the respiratory passages has not been demonstrated. Even in the cases of pneumotyphus the absence of intestinal lesions to which the early lung affection may have been consecutive has not been established.

(d) **Mixed or Secondary Infections.**—The conditions caused by the Bacillus typhosus impair the powers of resistance. A secondary invasion of colon bacilli, streptococci, staphylococci, or the pneumococci may occur with the development of consecutive local and constitutional phenomena. This true mixed infection may take place in any disease and is to be discriminated from other specific infections which occur as complications or intercurrent affections, as infection with Bacillus tuberculosis, Bacillus diphtheriæ, Streptococcus Fehleisen or, the malarial parasite.

(e) **Cases Presenting the Symptoms of Enteric Fever but due to other Organisms—Paratyphoid.**—Researches conducted since 1896 have shown that a symptom-complex not to be distinguished from enteric fever may be caused by organisms other than the Bacillus typhosus, which stand in their cultural and agglutinating properties between B. typhosus and B. coli communis, and that B. coli may perhaps play the same etiologic rôle. This fact does not, however, impair the universal belief in the specific nature of B. typhosus and enteric fever.

As regards intensity the following forms may be described:

(a) **The Mild Form—Typhus Levissimus.**—The fever is moderate, not exceeding 102°-103° F. (39°-39.5° C.) in the evening. The symptoms characteristic of the ordinary form are present but are of mild intensity. Headache, weakness, epistaxis, rose spots, and the signs of splenic enlargement are present, but the illness is so slight that it is difficult to make the patient realize its true nature. Diarrhœa is not common. These cases are often regarded as simple continued fever, febricula, or gastric fever. Their duration varies from eight to fourteen days.

(b) **The Abortive Form.**—The onset is abrupt and marked by shivering or a chill. The temperature rises abruptly and may reach 104° F.

(40° C.). Rose spots appear early, often before the fifth day. At the end of the first week, or early in the second, the temperature falls by rapid lysis or even by crisis with profuse sweating and the patient enters upon convalescence. These cases are sometimes seen in epidemics. The recognition of the true nature of the mild and abortive cases is of the utmost importance from the standpoint of prophylaxis.

(c) **The Latent or Ambulatory Form—Walking Typhoid.**—The symptoms are slight and the patient continues to attend to his affairs as usual. There is feverishness and a feeling of illness. Diarrhœa is commonly present but not urgent. The rose spots and enlarged spleen are often found in the routine examination of walking typhoid patients; or sudden delirium, hemorrhage, or perforation may occur. Cases belonging to this group are more common in men than in women and among laboring men, tramps, and others who habitually give little attention to their subjective symptoms. They are also encountered with some frequency among school-boys.

(d) **The Grave Form.**—The symptoms may at first be of moderate intensity. More commonly they are severe from the onset. The infection is intense. The temperature is high, 105°-106° F. (40.5°-41° C.), with very trifling remissions, and the duration of the fever may be protracted into the fifth or sixth week. To this category must be assigned the cases of mixed or secondary infection and the cases beginning with severe symptoms referable to the lungs, kidneys, and nervous system.

Modifications of the course of the attack as determined by anatomical and physiological conditions peculiar to the individual give rise to the following forms:

(a) **Enteric Fever in Children.**—This disease is not common in infancy. The nature of the food and doubtless the presence of an immunizing substance in the milk of the mother protects sucklings. Cases have, however, been reported in the first week of life and occasionally in the first year. Enteric fever is not at all uncommon after the second year. The onset may be insidious; commonly it is abrupt with high temperature. The type of the fever in a majority of the cases is remittent throughout—infantile remittent of the early writers. Nose-bleeding and diarrhœa are comparatively infrequent but bronchial catarrh begins early and is often moderately severe. There is nothing peculiar about the rash, which may be sparse or plentiful. Tympanites is commonly slight and intestinal hemorrhage and perforation much less common than in adults. Nervous symptoms are often prominent. The attack may begin with convulsions. Drowsiness alternating with insomnia, and mild delirium interrupted by sudden outcries and spells of fretfulness are observed. Aphasia, usually transient, and noma are prominent sequels. The mortality is much lower among children than in older persons. The marked differences in the course of the disease in childhood and after puberty, and especially the very common occurrence of fever of remittent course in connection with the symptom-complex just described, warrant the division of the cases of enteric fever into two great groups, those of the Infantile and those of the Adult Type. Those of the infantile type are milder than those of the adult type and the prognosis is more favorable. Cases of the former sometimes occur among adults; of the latter among children. The prognosis is less favorable in an attack of adult type in a child; more so when the infantile type occurs in later life. This is in accordance with a long recognized fact, namely, that in the absence of complications the prognosis is more favorable in proportion as the morning remissions are longer, *i.e.*, as the temperature curve conforms to the remittent type of fever. It has been found also that treatment which systematically brings about large oscillations between the morning and evening temperatures, as the cold bath treatment, also renders the prognosis more favorable.

(b) **Enteric Fever in the Aged.**—The course of the disease is much modified when it occurs in middle life or in elderly persons. The temperature range is irregular and not so high. The rose rash and splenic tumor are often absent. Diarrhœa and tympany are often troublesome and there is a marked tendency to complications, especially those affecting the respiratory tract—pneumonia, bronchitis.

(c) **Enteric Fever in Pregnancy.**—The pregnant woman enjoys no immunity against the disease. The fever may develop at any time, but is more commonly met with in the first half of pregnancy. Abortion or premature labor occurs in a large proportion of the cases. The maternal mortality is high—sixteen to twenty per cent. Infection of the fœtus does not always follow, but when it occurs the child dies either in utero or shortly after delivery. Recent investigations have shown that the bacilli may pass by way of the placenta to the child and cause a typhoid septicæmia without intestinal lesions. The positive Widal reaction has been observed with fetal blood.

Complications and Sequels.—Complications and sequels are more common in enteric fever than in any other acute infectious disease. A recognition of this fact is of great importance in diagnosis, since cases occur in which the prominence of a complication may mask the symptoms of the primary disease.

The following more important complications are to be considered:

(a) **Complications Involving the Digestive and Abdominal Organs.**—Ulcerative stomatitis occasionally occurs. Phlegmonous and pseudomembranous angina is a rare complication, which may develop in the third week and usually proves fatal. Parotid bubo, usually single, sometimes double, is a grave but not necessarily fatal complication in severe cases. It may be followed by extensive sloughing or by angina Ludovici, venous thrombosis or pyæmia. Hæmatemesis is of extremely rare occurrence in enteric fever. It may result from the specific lesions implicating agminate follicles present in the gastric mucous membrane or from a peptic ulcer.

The enlargement of the spleen may attain such a degree that the capsule may burst. Rupture of this organ is more likely to be the result of abscess formation following infarct. The latter condition owes its occurrence to embolism or venous thrombosis.

The liver itself is rarely the seat of changes which attract attention. Jaundice is of very infrequent occurrence. Hepatic abscess is exceedingly rare. Cholecystitis is, on the other hand, common. Pain, tenderness, and muscular rigidity in the region of the gall-bladder may be noted in most of the cases. Distention of the viscus—gall-bladder tumor—may be recognized upon nice palpation and percussion. Perforation may occur with the symptoms of intestinal perforation—extreme pain, tenderness, rigidity, fall of temperature, collapse symptoms, and the general and local signs of peritonitis. A suppurative cholangitis may occur. More commonly the symptoms gradually subside and recovery follows. There may, however, be remote consequences. The bacilli frequently give rise to chronic cholecystitis with recurrent paroxysms and to cholelithiasis.

Persistence of Bacillus typhosus After Recovery.—In by far the greater number of cases inflammation of the bile-ducts and gall-bladder terminates in recovery, but in about 2 per cent. it persists, especially in the gall-bladder, and the bacillus continues to multiply in the latter for an indefinite period. From this viscus it passes from time to time in considerable quantities into the gut and may be recovered from the fæces. These cases constitute a group of individuals now known as "typhoid carriers" who are a constant source of danger to the public, since they may spread the infection without giving rise to suspicion. Many of the sporadic cases, the origin of which has been involved in obscurity, are now attributed to these "carriers." Many of them are women and the subjects of cholelithiasis. The urine for several weeks may be in the absence of symptoms laden with virulent typhoid bacilli.

Intestinal hemorrhage and perforation have already been considered. These events are so directly due to the specific lesions of enteric fever, they occur with such frequency, and require such a degree of importance in the consideration of the subject that it seems more in accordance with the facts to regard them not as complications, but rather as accidents in the disease.

(b) **Complications Affecting the Respiratory Organs.**—Laryngeal ulceration is common in the severe cases. It may consist merely of superficial erosion and run its course without symptoms. It may, on the other hand, give rise to hoarseness, pain and difficulty in deglutition. Finally, it may produce perichondritis, in the course of which œdema of the glottis may occur. Bronchitis is prominent in infancy and often severe in old persons. Hypostatic pneumonia and deglutition pneumonia are almost always present in severe cases after the middle of the second week. Pulmonary œdema is a terminal condition.

Lobar pneumonia occurs, (1) as an initial condition—pneumotyphus. The onset is abrupt with chill, high temperature, pain in the sides. Cough and bloody sputa occur. After a day or two the signs of consolidation occur and the case presents the complete clinical picture of an ordinary croupous pneumonia. Crisis does not occur and by the end of the first or the middle of the second week rose spots appear and the symptoms of enteric fever are unmistakable. In the absence of rose spots, the uncertainty as to whether the case is one of croupous pneumonia with so-called typhoid symptoms resulting from secondary infection or enteric fever with early pulmonary localization can only be cleared up by a bacteriological diagnosis—presence or absence of Eberth's bacilli in the sputum, urine, and pus, blood culture, Gruber-Widal test. (2) Croupous pneumonia is a common and serious complication—intercurrent disease—in the second or third week. It usually occurs in cases already otherwise severe. The symptoms are not usually well developed. Cough is slight, rusty sputum may be absent, and the presence of the pulmonary consolidation may only be discovered upon routine examination. Secondary gangrene of the lung may develop. Pulmonary gangrene in enteric fever is more frequently the result of the breaking down of an infarct. Abscess of the lung constitutes one of the less common complications.

Pleurisy is by no means rare. It may be fibrinous, serofibrinous, or purulent. The effusion is often small and circumscribed. It may follow the signs of an infarct—local pain and dulness, increased fever, and hemorrhagic sputa. Typhoid bacilli have frequently been found in both the serous and purulent pleural exudates.

Pulmonary tuberculosis is not rarely an associated affection. The patient may be already phthisical, in which case the enteric fever plays the rôle of an intercurrent disease, or a latent tuberculous process may become active. Hæmoptysis may be profuse, even fatal. Pneumothorax has been observed. It may result from the rupture of a peripheral abscess of the lung.

(c) **Complications Affecting the Circulatory Organs.**—Pericarditis is exceedingly uncommon. It has been observed in children and in connection with pneumonia. Endocarditis is likewise rare.

Myocardial changes are on the other hand most frequent. They begin early and are often well established by the end of the second week. The heart is soft, flabby, and of a pale yellowish—faded-leaf—color. Upon the table it often flattens into a formless mass. Microscopically the changes are those of parenchymatous degeneration and interstitial myocarditis. There may be fatty degeneration of high grade. Feebleness of the pulse, faint, even inaudible first sound, profound asthenia, fatal collapse may be the manifestations of the changes in the myocardium. Acute dilatation with relative insufficiency, thrombus formation and visceral engorgements likewise arise and are attended with their usual symptoms and signs. There is a direct relationship in patients who recover between the myocardial changes of enteric fever and chronic myocarditis, the symptoms of which develop later in life, as can be learned from the anamnesis.

Vascular occlusions occur both in the veins and arteries. They may result from embolism or thrombosis. Obliteration of the femoral artery may occur with gangrene of the foot and leg. Obliteration of both femorals with extension of the clot into the aorta has been observed. The condition has been ascribed to local arteritis with thrombus formation.

Venous thrombosis is of comparatively frequent occurrence—two to four per cent. of the cases. It is usually unilateral, sometimes bilateral, the left side being first affected, the right later. In far the greater number of cases the femoral vein is the seat of the occlusion; less frequently the popliteal or the long saphenous. The clot may extend along the vein from the point of formation. The greater liability of the left femoral vein has been attributed to the relative retardation of the blood-flow in the left common iliac vein caused by the pressure of the right iliac artery which crosses it. The occurrence of venous thrombosis is attended by elevation of temperature, pain, tenderness, enlargement, and tense œdema of the leg. It may come on later in the attack or not until after defervescence. Eberth's bacilli have been found in the clot and in the wall of the vein. Suppuration and pyæmia may occur. A fragment of the clot swept into the blood stream may cause sudden death by plugging of the pulmonary artery. Gangrene does not result from venous thrombosis. As the collateral circulation is established the enlargement of the leg subsides, but many patients are obliged to wear an elastic stocking for months or even years.

Thrombosis of the cerebral sinuses is a rare accident. Infarction of the lungs, spleen, or kidneys occurs as the result of arterial occlusion, due more commonly to thrombosis than embolism.

Gangrene as a sequel of enteric fever most commonly affects the feet and legs. Gangrenous areas occur less frequently upon the face, neck, and trunk. The genitalia, especially in girls, the nose and ears may also be affected.

The blood undergoes important changes. In the third week the erythrocytes and hæmoglobin are reduced. A gradual increase to normal takes place during convalescence, the hæmoglobin, which has suffered a relatively greater reduction than the corpuscles, regaining the normal more slowly than the latter. These changes are without diagnostic value.

Very important, however, is the fact that there is a reduction of the leucocytes during the whole course of the attack—leucopenia. This condition is of actual diagnostic value in the differentiation between enteric fever and septic states, and other infectious diseases which resemble it more or less closely. A leucocytosis occurs when in the course of enteric fever a local inflammation arises or pneumonia or some other affection characterized by an increase in the number of the leucocytes occurs as an intercurrent disease. The large mononuclear and transitional forms are increased and the polynuclear neutrophiles are greatly diminished. In contrast to these changes, the polynuclear neutrophiles are increased in inflammatory conditions, as in abscess formation or perforation, a fact of diagnostic value.

(d) **Complications Affecting the Nervous System.**—The nervous system, as has been pointed out above, in all cases manifests to a greater or less degree the effects of the intoxication. These effects are apparent at the onset of the attack and vary in intensity from the headache and indisposition to mental effort seen in the mildest cases to the furious symptoms of meningitis. The latter cases, which fortunately are extremely rare, are characterized by intense headache, photophobia, painful retraction of the muscles of the back of the neck, muscular twitchings, rigidity, and in some cases convulsions. The onset is abrupt and vomiting may occur. At the end of a week the symptoms become less intense, in a considerable proportion of the cases almost as suddenly as they appeared. The headache ceases, rose spots appear, there is palpable enlargement of the spleen, and the case presents the clinical features of an ordinary attack of enteric fever which runs the usual course. These are the cases described as cerebrospinal typhoid. In fatal cases of this group the lesions of meningitis are not found.

Inflammation of the meninges of the brain has been observed. It is an extremely rare complication. Typhoid bacilli have been isolated from the exudate in pure culture. Kernig's sign was present in a case of enteric fever with meningeal symptoms recently observed. Lumbar puncture may be necessary in the differential diagnosis between this form of enteric fever and cerebrospinal fever.

The rare cases of thrombosis of the cerebral veins and sinuses are characterized by local and general convulsive movements, active delirium, and rapidly developing coma.

Neuritis is an infrequent complication. It is usually confined to a single nerve; sometimes it is symmetrical. Less frequently there is a general peripheral neuritis. Neuritis affecting a single nerve area may develop during the course of the attack, or, as is more commonly the case, after convalescence has begun. There is severe pain in the affected limb with exquisite tenderness over the trunk of the nerve. There may be swelling and redness. The extensors are more commonly involved and wrist-drop or foot-drop may occur.

Tender toes constitute the manifestation of a form of neuritis not very uncommon. This distressing but not very serious condition may occur under any treatment but is said to be more frequent in cases treated by systematic cold bathing. The tips of the toes, their pads, and sometimes the pads at their bases are painful and exquisitely sensitive so that it is impossible for the patient to bear the weight of the bedclothes. There is neither redness nor swelling, and the condition—which begins toward the end of the attack—usually disappears in the course of a week or ten days.

Multiple neuritis, sometimes giving rise to paraplegia, palsies of individual nerve-trunks from neuritis and poliomyelitis, hemiplegia from thrombosis or meningo-encephalitis, and tetany may develop during convalescence. These sequels are of extremely rare occurrence and of secondary importance in the diagnosis.

Postfebrile insanity is encountered more frequently after enteric fever than any other acute infection. It belongs to the group of confusional insanities and is the manifestation of profound nutritional derangements in neurotic individuals. It has occurred in my experience in cases in which active delirium has been present during the attack. There are persistent hallucinations, melancholia, and speech derangements. The prognosis is good. A majority of the cases recover in the course of six or eight months. After that period the outlook becomes progressively less favorable, though I have seen a case recover at the end of a year.

Complications relating to the eye are exceedingly rare. Of those affecting the ear, otitis media is common. There have been years in my service in the Pennsylvania Hospital in which no cases of this complication have occurred, and years in which it has been extremely frequent. The otitis commonly develops insidiously but it sometimes comes on with a chill and increase of fever. The ears should be examined as a matter of routine. Mastoid disease may occur.

(e) **Other Complications.**—Febrile—toxic—albuminuria is of very frequent occurrence as in the other serious infectious diseases. Hyaline and granulohyaline casts may be found in the centrifugated urine. This condition passes away with convalescence. Eberth's bacilli are demonstrable in the urine in a large proportion of the cases. In some cases they have been found in the urine of persons months, even years, after the attack.

The attack may begin with the symptoms of an acute nephritis—nephrotyphus. The toxic albuminuria occasionally leads to the development of an acute nephritis with scanty urine of high specific gravity. a large amount of albumin, and microscopical blood. This complication may be the cause of a fatal termination of the case. Otherwise it gradually subsides and complete recovery ensues. Polyuria during the course of the attack is an extremely rare complication. Chronic nephritis is a rare sequel. The multiple foci of round-celled infiltration—lymphomatous infiltration—which may lead to the formation of miliary abscess does not as a rule give rise to symptoms and is rather of pathological than of clinical interest.

Pyelitis is occasionally observed. It may develop in the later stages of the attack or during convalescence. It may involve one or both kidneys. This infection reveals itself by free blood together with pus and later by a condition in which there is an abundant pyuria. If one kidney only is affected the pyuria may be intermittent, with an almost pus-free urine in the intervals.

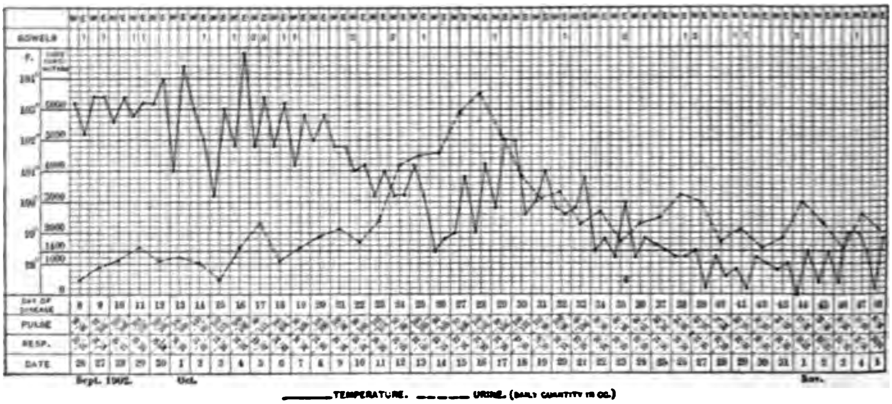


FIG. 211.—Polyuria in enteric fever.

Catarrh of the bladder or an acute cystitis may be the cause of the urinary pus. These conditions are somewhat common in enteric fever. especially after repeated catheterization. The more common pyogenic micro-organisms are B. coli, B. typhosus, and staphylococci. Orchitis occasionally occurs, as a rule in association with a purulent urethritis. In women œdema and gangrene of the vulva and acute mastitis are very rare complications. Menstruation is not likely to occur during the attack. When it does occur it is usually profuse.

Hemorrhagic cases of enteric fever have been reported. Of these I have seen several. Hemorrhages into the skin are more common than from mucous surfaces. The general symptoms are severe.

Furunculosis, local gangrene of peripheral parts, periostitis, caries, and necrosis with abscess and sinus formation are complications and sequels often encountered. The bone lesions of enteric fever are notable for their persistence. Typhoid bacilli are present in a majority of the cases. The tibiæ, ribs, and costal cartilages are most commonly involved. Arthritis involving the knee and hip is among the infrequent complications. It is usually septic.

Typhoid Spine.—Spinal symptoms are sometimes observed in the later course of severe attacks or during convalescence. They consist of pain in the lumbosacral region, aggravated by movement, tenderness upon pressure, stiffness, and inability to execute movements requiring flexion, extension, or rotation of the spine. Nervous symptoms are prominent in some cases. There is no rise of temperature and physical signs are absent. The condition is usually a neurosis—hysterical spine—and terminates in recovery. There may be a spondylitis. I have seen a fatal case of vertebral tuberculosis following enteric fever in a lad of seventeen, which for a period of several months was regarded as an instance of typhoid spine.

Parenchymatous degeneration of the voluntary muscles, which especially affects the recti abdominalis and the adductors of the thigh, sometimes leads to the rupture of the mass of the muscle and may also lead to hemorrhage and abscess formation.

The Association of other Diseases with Enteric Fever.—The frequency of croupous pneumonia as an intercurrent disease has been noted above. Erysipelas occurs in about two per cent. of the cases, more commonly in the period of convalescence. The exanthemata, especially measles and varicella, may be associated with enteric fever.

Malarial fever and enteric fever may coexist. There is no such thing as a hybrid, as indicated by the term typhomalarial fever. Such cases are usually enteric fever or estivo-autumnal fever without well-marked paroxysms.

Tuberculosis as an associated disease has already been mentioned. The various phases of this relation will be fully considered later.

The Effect of Enteric Fever upon Certain Chronic Diseases.—During the attack of enteric fever the paroxysms of epilepsy frequently cease, the irregular movements of chorea and the allied affections are in abeyance, and sugar frequently wholly disappears from the urine in diabetic subjects. In all these conditions the effect is only temporary and the symptoms of the antecedent disease recur with convalescence or shortly afterwards.

Relapse.—The frequency of relapse varies, according to different observers, between three and eighteen or twenty per cent. Relapse occurs with greater frequency in cases treated by systematic cold bathing. It is obvious that a therapeutic method which reduces the mortality will increase the number of convalescent cases in which relapse may occur.

The ordinary form of relapse begins after the defervescence is complete and presents the picture of a repetition of the primary attack, usually shortened and moderated in intensity. The onset is somewhat more rapid; sometimes abrupt with a chill. At times, however, the relapse is even more severe than the original disease and occasionally it terminates in death. The interval between the defervescence and the relapse varies from two or three to twenty days. I have seen a case in which it was five weeks. The question as to whether a repetition of the attack after a prolonged period constitutes a late relapse or an early second attack is purely academic and without practical importance. The relapse is commonly single; occasionally multiple: two are by no means rare; three are infrequent; five have been observed. The diagnosis of relapse rests upon the range and duration of the fever and its association with rose spots, recurrence of splenic tumor, cessation of the hunger which follows defervescence, and other factors in the enteric fever symptom-complex. It is to be distinguished from recrudescence—a transient fever dependent upon the instability of the heat-regulating apparatus during convalescence and due to trifling causes; from so-called bed-fever,—an unimportant clinical manifestation,—and from the symptomatic fever which may be the first

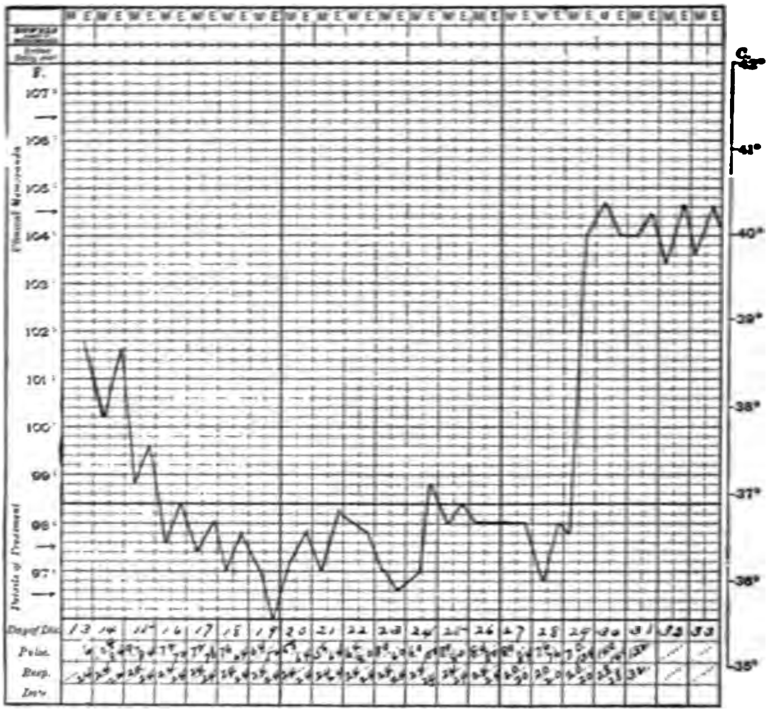


Fig. 212.—Enteric fever. Relapse beginning in abrupt rise of temperature on the 29th day after the onset of the primary attack and the 14th after complete defervescence. Duration of relapse 13 days. Recovery. Girl, aged 11 years.

indication of some sequel or fresh infective or inflammatory process, to which the depraved nutrition of the organism singularly disposes it.

Intercurrent Relapse.—This form of relapse receives its name from the fact that it begins before the primary attack comes to an end. The patient seems to be doing well; the temperature has assumed the remittent, even the intermittent, curve; the tongue begins to clean off; the nervous symptoms ameliorate, and convalescence appears assured, when the fever again rises and becomes subcontinuous, and with recurrence of the symptoms the attack repeats its previous course. Relapses of this form are often severe. They explain a large proportion of the protracted cases.

Theory of the Relapse in Enteric Fever.—In a majority of the cases the circumstances under which relapses occur render it practically impossible for the reinfection to have come from outside. Neither the water nor the food, including the milk, can be incriminated. The theory of reinfection from within the organism is the only alternative. Of the actual conditions favoring its occurrence little is known. As a working hypothesis, reinfection from the gall-bladder and bile passages as suggested by Chiari appears to meet the requirements of a majority of the cases. The persistence of the bacilli in the mesenteric glands, the spleen, and the gall-bladder, and their presence in the stools for a long time after defervescence favor this view. The immunity conferred by the attack is apparently of slow development. The fact that the greater number of relapses occur within a week or ten days after the fever has subsided, at a time when solid food in increasing amounts is being taken, lends support to this hypothesis. It is at least possible that increased peristalsis may cause the sudden discharge of large quantities of bacilli-laden bile into the intestines at a time when immunity is not yet complete. Too much food and unwholesome food may cause recrudescences of fever, but that they can, in the absence of reinfection, cause relapse is a proposition not to be considered. The hypothesis of Durham, that groups of bacilli of similar species but not identical cause the attack, and that the antitoxin formed in the blood does not neutralize all these groups, those remaining active giving rise to single or multiple relapses, is highly ingenious but lacks proof.

Diagnosis.—(1) The causal or etiological diagnosis; (2) the direct diagnosis; (3) the differential diagnosis; (4) diagnosis by exclusion; and (5) a provisional diagnosis are to be considered.

The diagnosis is a simple matter in well-characterized cases after the first week. The atypical cases are obscure and the more widely they depart from the type the more uncertain does the diagnosis become. The remarkable variations in the disease itself, the great number of complications and sequels, and the irregularity of its course add to the difficulties. The diagnostician must know not only enteric fever but he must also know the many maladies to which in its multiform aspects it bears a close or superficial resemblance.

1. **Causal or Etiological Diagnosis.**—In sporadic cases in which the true nature of the attack remains obscure and the association of cardinal symptoms essential to a clinical diagnosis is lacking, it is of importance to ascertain whether or not the patient has visited a district in which enteric fever is endemic or in which the water supply is tainted; whether he has been in a house in which there were one or several cases or he is a newcomer, and to ascertain the length of time since such probable exposure. The question of acquired immunity is to be considered. A clear history of a previous attack is presumptive but not positive evidence against enteric fever in a doubtful case. In a majority of sporadic cases the source of the infection cannot be traced.

In local epidemics every effort should be made to find the starting-point of the outbreak. Has a case been the source of contamination? Has a sewer burst and discharged its contents into a water reservoir? Do the cases follow the distribution of the milk from a particular dairy? Are they limited to those who have eaten oysters or other shell-fish from beds in sewage-defiled waters? If in a standing camp is the water to be incriminated, or badly constructed latrines and swarms of house-flies? Upon the answer to questions like these often depend many lives. Diagnosis and prophylaxis, the work of the physician and of the sanitarian, are inseparably bound together. Bacteriologically, the presence of the bacilli in the blood or discharges is of conclusive importance.

The isolation of B. typhosus from the blood by means of culture methods has become in recent years a practical diagnostic procedure. The quantity of blood necessary is considerable but not so great as to do harm in any case. Its withdrawal from a vein is almost painless. This method yields positive results early in the attack, even before the appearance of the eruption. It is especially valuable in septic cases. A recent method is that of Peabody, and consists in the employment of ox-bile as a culture medium for the Bacillus typhosus. The quantity of blood required is small—two cubic centimetres being sufficient. The blood is inoculated into a small quantity of sterile ox-bile which is incubated for twenty-four hours. A portion of this culture is then transferred to Löffler's blood-serum medium and incubated for another twenty-four hours. The isolation of a motile bacillus may be regarded as presumptive evidence of the existence of enteric fever, but further cultures are necessary to establish the identity of the organism.

Blood removed from the rose spots contains the bacilli. The procedure is attended with pain and is useless for diagnostic purposes, since the eruption itself constitutes a cardinal diagnostic criterion. The isolation of the bacilli from the urine is now a practical method of diagnosis. Their demonstration is conclusive as regards the nature of the disease. Their presence has been noted in some cases at an earlier period than a positive agglutination test.

The isolation of the bacilli from the stools has been rendered practicable by the more recent culture methods, but the technical difficulties are considerable and the results uncertain.

Of the foregoing methods, blood cultures and the examination of the urine are more practical than the others and are in use when the necessary technical skill and the facilities of a clinical laboratory are at hand. The majority of practitioners are not, however, in a position to avail themselves of them.

THE AGGLUTINATION TEST—WIDAL TEST.—This diagnostic procedure is of very great value. It depends upon the property of the blood-serum of an enteric fever patient, when added to a fresh culture of the bacilli, to cause an arrest of the movement of the latter and their agglutination in clumps. The test requires a definite dilution of the serum and time limit. The microscopical examination is made by means of the hanging drop. A dilution of 1–50 and time limit of an hour are in general use. The result is decisive if loss of motility and clumping occur within this period. The dried serum is convenient but its use does not permit of accurate dilution. Small glass bulbs may be used for the collection of the serum. When the test yields a positive result an equally prompt and energetic response to an increased dilution renders the diagnosis even more certain.

The results of the Widal test are to be taken into consideration in connection with the following facts:

A positive result has been obtained some time in the course of the attack or convalescence in about 97 per cent. of cases in which the clinical or post-mortem data rendered the diagnosis certain and in about 93 per cent. of similar cases in which the test was made before the eighth day. In a limited number of cases the diagnosis has been negative in the early course of the attack and become positive in the third or fourth week, or not until after the defervescence or upon the occurrence of a relapse. A well-characterized case of enteric fever with enlarged spleen, rose spots, diarrhœa, even intestinal hemorrhage may give a negative result throughout. The agglutinating power is retained by the serum for an indefinite time, sometimes months, even years, after the attack. A positive reaction may be therefore misleading, especially in a patient in whom the disease has run its course in an irregular manner or has been of the mildest or of the ambulatory variety—and does not therefore appear in the anamnesis. The blood-serum of healthy persons and of persons suffering from other specific infections, undiluted or in much lower dilution than is used in the Widal test, sometimes causes loss of motility and clumping of the bacilli in cultures, but not with the same promptness and energy, and this property in non-typhoid blood is lost when higher dilutions, 1–50, 1–100, are employed. In a limited number of cases presenting the clinical picture of enteric fever but in which the Widal test has remained negative, a positive reaction has been obtained with cultures of paratyphoid bacilli—paratyphoid.

Caution is therefore necessary, except in the prompt and marked reaction on the one hand and in the cases, on the other hand, in which the reaction does not occur at any time, in drawing either positive or negative conclusions. If, however, due regard be paid to the above mentioned peculiarities in the reaction, the Widal test is of great value in diagnosis.

The Widal test, important as it is, cannot be made available for the general practitioner because of the difficulty of having constantly on hand fresh cultures of the bacilli. Only in communities in which there are well-equipped clinical laboratories can its full usefulness be realized.

MACROSCOPIC METHOD.—This method is based upon the fact that typhoid bacilli are susceptible to agglutination after devitalization by certain antiseptics. The killed bacilli are suspended in narrow test-tubes, forming a homogeneous, slightly cloudy fluid. Upon the addition of typhoid serum agglutination takes place, and flocculi appear which gradually settle as a white precipitate, leaving the supernatant fluid clear. If the reaction is incomplete, flocculi are formed which still float. When the reaction is negative the fluid remains uniformly cloudy, as in the control tube. Suitable outfits with full directions are made and marketed by the biological laboratories.

THE OPHTHALMIC REACTION IN THE DIAGNOSIS OF ENTERIC FEVER.—Chantemesse (1907) announced a new characteristic reaction for the diagnosis of enteric fever. The test is performed by instilling into the conjunctival sac a solution prepared from virulent typhoid bacilli by washing, drying and trituration, precipitation with alcohol, and again drying and reducing to powder. A positive reaction consists in the occurrence, in from three to eighteen hours, of conjunctivitis, which attains its maximum in a few hours and subsides in the course of twenty-four to seventy-two hours. In control cases there is at most a slight hyperæmia which passes off in the course of five or six hours.

The urotoxic coefficient in enteric fever is high and has been found to be increased in cases treated by systematic cold bathing. This fact which is intimately related to the bacteriology of the disease is not used for diagnostic purposes.

2. **The Direct Diagnosis.**—The presence of B. typhosus in the blood or excretions or a prompt response to the Widal test renders the diagnosis positive independently of the clinical phenomena. The direct diagnosis then rests upon the bacteriological diagnosis. In the vast majority of cases the diagnosis can be made with precision at the bedside—a fortunate matter, since the elaborate procedures of the bacteriological laboratory are not always available.

The association of the four following symptoms is of cardinal importance:

(a) THE GRADUALLY ASCENDING TEMPERATURE.—This phenomenon is available only when the observations have been begun early in the attack—second or third day. This happens only in a limited number of the cases; but a temperature of 103°–104° F. (39.5°–40° C.) of subcontinuous type and a history of febrile symptoms of gradually increasing severity and several days' duration are significant. A curve of remittent type in the third week and distinct intermissions with progressively falling evening maxima are of great diagnostic value in a case in which other diagnostic criteria have been uncertain. The duration of the febrile movement is important, but it is in the doubtful cases that we find a shorter or, more commonly, a longer course than in those which are typical.

(b) THE PULSE-FREQUENCY, which is increased, but not in proportion to the temperature. With evening maxima of 104° F. the pulse-frequency may not exceed 90–110 per minute. This derangement of ratio is observed in very few of the acute febrile infections. Yellow fever is conspicuous, but there can scarcely be any question as to the discrimination between that disease and enteric fever. There is nothing characteristic in the pulse itself. Dicrotism occurs under other conditions, but its early appearance is very suggestive.

(c) ENLARGEMENT OF THE SPLEEN.—This is a constant phenomenon, but cannot always, on account of the distention of the bowel, be demonstrated. It occurs in the other acute infections. Nevertheless enlargement of the spleen at the end of the first week, associated with the other cardinal symptoms, is of great diagnostic value in the direct diagnosis.

(d) THE ERUPTION.—The appearance of the rose spots at the close of the first or in the course of the second week is an event of the highest diagnostic significance. A single spot is without great value, and the single spot is usually a "doubtful" spot; but successive crops of papulo-macular, rose-pink spots, distributed over the upper abdominal or lower thoracic regions, disappearing upon pressure or when the skin is made tense, each spot fading in the course of three or four days, do not occur in any other febrile infection and may be regarded as rendering a provisional diagnosis positive.

SYMPTOMS OF MINOR DIAGNOSTIC IMPORTANCE are epistaxis, the furred tongue, red at the borders and tip, diarrhœa with thin, ochre-colored stools containing now and then one or two hard masses, and separating on standing into a thin upper and a thicker sedimentary layer, tympany, the nervous phenomena, and bronchitis. Intestinal hemorrhage or perforation confirms the diagnosis in a doubtful case. The tendency to complications is characteristic of enteric fever, especially in the later course of the attack. Furunculosis, abscess formation, parotitis, bed-sores, septic phenomena are of merely suggestive importance. A subnormal temperature and eager hunger after defervescence are of very common occurrence. The absence of leucocytosis is of value, but it is precisely in the doubtful cases that inflammatory processes or obscure pus collections impair the value of this method of diagnosis.

Among the symptoms which militate against the diagnosis but do not negative it entirely are marked coryza, herpes, initial sweating, early arthritis and endo- or pericarditis; the continued absence of abdominal symptoms, as diarrhœa, especially if resistant to laxatives, a retracted abdomen and only slight enlargement of the spleen. The continuing absence of the diazo reaction is not common in enteric fever.

3. **Differential Diagnosis.**—The data for a positive diagnosis of enteric fever are rarely present during the first week. A provisional diagnosis only is possible. This is especially the case when the patient is seen for the first time. During the fastigium if the four cardinal symptoms of subcontinuous high temperature, slow pulse in proportion to the temperature, enlarged spleen, and rash are present the nature of the malady is beyond question. But it often happens that one or more, even all of these symptoms, are wanting. The fever may be irregular, the pulse rapid, the enlargement of the spleen not demonstrable, and eruption absent. In the later course of the attack septic phenomena, various complications, or an intercurrent relapse may again render the diagnosis obscure. It is instructive, therefore, to consider under the above heading (a) the diseases which resemble enteric fever in the first week, (b) those which resemble it during the fastigium, and (c) those which resemble it at later periods.

(a) DISEASES WHICH RESEMBLE ENTERIC FEVER IN THE FIRST WEEK.—We must consider first the diseases which sometimes resemble it in its normal course—influenza, febricula, the exanthemata, febrile enteritis and gastro-enteritis, appendicitis—and those which resemble it in certain of its varieties—cerebrospinal fever, pneumonia, central pneumonia, acute nephritis, and septic conditions.

Influenza can in some of the cases be excluded only after several days, especially when the attack has begun with a gradual, step-like elevation of temperature, diarrhœa, and enlargement of the spleen. I have many times seen epistaxis at the onset of an attack of epidemic influenza. The initial nervous symptoms are also much alike. In favor of influenza are a more abrupt onset, catarrhal symptoms, especially coryza and conjunctivitis, the intensity of the headache and its localization in the orbital regions, a pulse-rate proportionate to the rise in temperature, and the fact that the attack runs its course and defervescence is complete by the end of a week.

Febricula.—Every practitioner sees cases of transient fever with headache, malaise, anorexia, and sometimes barely recognizable enlargement of the spleen. If the symptoms last twenty-four hours and disappear altogether, the attack is known as ephemeral fever; if they continue longer, to six or seven days in the absence of local trouble, it is designated febricula. Many of the cases are examples of the mildest variety of enteric fever as shown by the Widal test. The diazo reaction may also be present. In some instances rose spots are seen. If these tests are negative the illness is not due to typhoid infection, but to some other infectious agent, gastro-intestinal catarrh, ptomaine poisoning or overwhelming foul odors, or the fever is symptomatic of some unrecognized local infection or inflammatory process.

Acute Exanthemata.—Scarlet fever, measles, and the variolous diseases may during the period of onset give rise to the suspicion that enteric fever is developing. The character of the temperature range, the coryza in measles, the angina of scarlatina, the intense headache and backache of variola, together with the initial rashes when present and the appearance of the definite eruption in a relatively short time, settle any question as to the essential nature of the infection. A doubt only can arise in regard to the eruption of measles. In several instances I have seen, especially in children, a typhoid eruption so copious as to suggest the exanthem of measles. In measles the rash shows itself upon the fourth day after a slight fall of temperature, usually first on the face and later over the trunk and limbs, presents a crescentic arrangement, and is preceded by marked catarrhal symptoms.

Febrile Enteritis and Gastro-enteritis.—As a rule, gastric and intestinal catarrhs run their course without fever. Febrile cases do, however, occur. It is to the infrequent cases of this kind that such terms as gastric fever and mucous fever owe their existence. Most of the cases so designated by practitioners are cases of enteric fever, and these terms are, fortunately, falling into disuse. The prominence of dyspeptic symptoms, colicky pains, the irregular course of the fever, the absence of disparity between the pulse-frequency and the elevation of temperature weigh heavily against the diagnosis of enteric fever, and a negative Widal reaction up to the time of complete defervescence would be conclusively against such a diagnosis.

Appendicitis.—The gastro-intestinal symptoms and especially the pain and tenderness may if attended by a rise in temperature simulate enteric fever. I have known of several instances in which a patient suffering from the latter disease has been admitted to a hospital at night and at once operated upon, with the recognition upon the following day of the true nature of the disease. This mistake ought not to occur. The sudden onset, the localization of the pain, the high degree of circumscribed tenderness, the absence of fever, or its irregularity when present, and the lack of the cardinal symptoms upon which the diagnosis of enteric fever rests should put the practitioner upon his guard. It is, however, to be borne in mind that after the middle of the second week of enteric fever perforation of the appendix has been observed.

Right tubo-ovarian disease with fever may also simulate enteric fever. The presence of a tender mass upon the right side with fixation of the uterus and leucocytosis are of positive diagnostic significance.

Meningotyphoid—cerebrospinal fever—takes first place among the diseases which simulate enteric fever in its more irregular forms. In fact the resemblance between the cerebrospinal form of enteric fever in the first week and cerebrospinal fever is so great that a differential diagnosis is in a majority of the cases altogether impossible. The onset is sudden with intense headache, photophobia, delirium, painful rigidity of the back of the neck, and sometimes vomiting. Kernig's sign may be present. Examination of the fluid obtained by lumbar puncture may show the meningococcus. The appearance of rose spots and abdominal symptoms at the end of the first week and mitigation of the nervous symptoms are characteristic of enteric fever. Herpes is common in cerebrospinal fever.

Pneumonia.—Pneumotyphus must be very rare. I have seen a very limited number of cases. The sudden onset with chill, high fever, pain in the side, cough, and the signs of consolidation are very misleading. The later course is that of enteric fever. The difficulties are increased in the irregular cases of enteric fever in aged persons. Such cases have been regarded as pneumonia until at the autopsy the intestinal lesions of enteric fever have been found. The recognition of intercurrent croupous pneumonia at the height of the disease is a comparatively easy matter provided that systematic routine examination by the methods of physical diagnosis are made.

There are cases of central pneumonia, occurring independently of enteric fever, which simulate it very closely. These cases run their course for days with no other symptoms than those of fever and perhaps a trifling cough. Pain, rusty sputum, and the signs of consolidation are not present. The diagnosis rests upon the abrupt onset with chill and high temperature, difficulty in breathing, the early appearance of herpes, and a slight degree of jaundice. Leucocytosis is of diagnostic value. It is to be remembered that this sign may be absent in the gravest cases of pneumonia and present in enteric fever complicated by inflammatory or purulent processes. The absence of the temperature curve of enteric fever, of relative slowness of the pulse, of considerable enlargement of the spleen, and of rose spots is important. The occurrence of rusty sputum, even when scanty, localized bronchophony, a tympanitic percussion sound, and crepitant râles establish the diagnosis of a central pneumonia, which is fully confirmed when defervescence takes place by crisis and free sweating.

Nephrotyphus.—The cases which begin with the clinical phenomena of an acute nephritis present great diagnostic difficulties during the first week and the practitioner who brings to the study of his cases in a systematic manner the ordinary clinical methods—early and thorough examination of the urine, for example—may in this particular group of cases encounter uncertainties that his less careful brother may avoid. There are headache, vertigo, mental dulness, disinclination for effort, loss of appetite, and fever. Epistaxis occurs in both conditions. The illness looks like enteric fever; the urine is that of an acute inflammation of the kidneys. It is scanty, high-colored, of high specific gravity—1.024 to 1.030—and contains much albumin, together with hyalogranular and epithelial tube-casts, cylindroids, and red blood-corpuscles. Rose spots, splenic tumor, a more or less characteristic temperature range, and the duration of the attack render the diagnosis clear. The nephritis does not tend to become chronic.

Sepsis.—I employ this term here to cover the conditions included under septicæmia, pyæmia, and septicopyæmia. In general the differential diagnosis between these conditions and enteric fever is not attended with difficulty. Where there is trauma or obvious bone disease or demonstrable suppuration no question arises. There are, however, forms of sepsis, especially those of cryptogenetic origin, having in common with enteric fever high temperature, splenic tumor, and nervous symptoms in which the diagnosis is very uncertain. Among the symptoms which favor the diagnosis of sepsis are the following: irregular fever with marked remissions and intermissions early in the illness; chills followed by profuse perspiration; endocarditis of the septic or malignant form; septic arthritis involving a single joint or many; tenderness upon pressure over the bones —sternum, clavicles, tibiæ—and retinal hemorrhage. The pulse also is very frequent and arrhythmic. In many of the cases there are marked meningeal symptoms and cutaneous lesions, herpes, urticaria, erythema, and petechiæ are common. We do not overlook the fact that sepsis frequently occurs in the course of enteric fever and that there is a recognized form of typhoid septicæmia—facts which are of importance in the diagnosis of individual cases.

(b) DISEASES WHICH RESEMBLE ENTERIC FEVER IN THE FASTIGIUM.—Malaria, typhus and relapsing fevers, internal anthrax, miliary and tuberculous peritonitis, certain forms of sepsis, and malignant endocarditis may be grouped in this category.

Malarial fever may as a rule be readily differentiated from enteric fever. The regularly intermitting forms present no difficulties. In the estivo-autumnal form the diagnosis may be uncertain for several days. The appearance after a time of the parasite in the blood settles all doubt as to the nature of the disease. Meanwhile the absence of chills, the continued fever with very moderate remissions, together with weakness, diarrhœa, and a palpable spleen, suggest enteric fever. The malarial and the typhoid infection may be present in the same patient at the same time. With the estivo-autumnal variety this association is not uncommon, as was shown in soldiers returning from Cuba and Porto Rico during the Spanish-American War. With the tertian and quartan parasites the association is rare and these organisms are very seldom present in the blood of individuals suffering from enteric fever. A hybrid disease such as is indicated by the term typhomalarial fever—a separate nosological entity—does not exist.

Typhus fever may be differentiated from enteric fever by the eruption, which in the latter is far and away more sparse and appears several days later in the course of the attack. Cases in which the rash appears early and is so copious as to suggest typhus fever do occur, but they are extremely rare. It does not, save in the rarest cases of hemorrhagic enteric fever, become petechial, as is the rule in typhus. The abrupt rise and critical fall of temperature in typhus are very significant, as is also the high pulse-frequency. Dicrotism is often present in both diseases. The difference in the duration of the two diseases, typhus lasting usually from ten to fourteen days, is to be noted. The Widal reaction is almost invariably wholly absent in typhus. Blood cultures may become necessary in a doubtful case.

Relapsing fever may be readily differentiated from enteric fever by its abrupt onset with chill and very high temperature, jaundice, pain and tenderness in the epigastric zone, critical defervescence, period of complete apyrexia, and relapse. The presence of the spirochæta of Obermeier in the blood is absolutely conclusive. In typhus and relapsing fevers the prevalence of an epidemic is to be taken into consideration in the differential diagnosis. Too great importance must not, however, be attached to epidemic influence. When an epidemic exists it does not necessarily follow that a person taken ill has contracted the prevalent disease.

Internal anthrax presents the symptoms of a severe infectious disease with intestinal symptoms. Fever, diarrhœa, and splenic enlargement occur. There are symptoms, however, which scarcely belong to enteric fever, as repeated vomiting, colic, bloody diarrhœa, hæmaturia, dyspnœa, cyanosis, and submucous extravasations of blood in the mouth. An examination of the blood reveals the presence of anthrax bacilli. If B. anthracis is not present, inoculation experiments must be practised.

Acute miliary tuberculosis is occasionally mistaken for enteric fever. This error in diagnosis arises from the fact that the former disease frequently begins rapidly in persons apparently in good health, with fever, enlargement of the spleen, and nervous symptoms, and without demonstrable signs of organic lesions upon physical examination. The presence in rare cases of a scanty eruption of rose-colored maculopapules not to be distinguished from the rash of enteric fever adds greatly to the uncertainties of the diagnosis. Their recurrence in crops is in favor of the latter affection. The Widal test should be tried and, in the case of a negative result, repeated at intervals of some days. In such cases there is usually little sputum and neither that which is expectorated nor the urine contains tubercle bacilli. Careful examination of the chest will often elicit suggestive signs, as vesiculotympanitic resonance at an apex and a few scattered small mucous or coarse crepitant râles of high pitch. Cyanosis and dyspnœa are prominent symptoms. The pulse, in the absence of an associated meningitis in which it is often slow, is frequent, feeble, and arrhythmic, showing in particular remarkable variations in frequency in the course of brief intervals of time. The splenic enlargement is less marked than in enteric fever; but there are a few cases of the latter disease in which the spleen is but little enlarged and many in which during the fastigium the enlargement cannot be demonstrated on account of the meteorism. The diazo reaction is sometimes absent in enteric fever and often present in tuberculosis. This last is therefore of secondary importance in the differential diagnosis between these two diseases. A complicating meningitis may occur in either affection, but is much more common in tuberculosis. If an ophthalmoscopic examination, which should be repeated from time to time, reveals the presence of tubercles in the choroid, the diagnosis is established. The atypical course of the temperature in tuberculosis, especially its extreme irregularity, the occurrence of remissions, and its occasional morning exacerbations and evening remissions—inverse type—are of great diagnostic value.

Tuberculous peritonitis may, in certain of its forms, present a misleading resemblance to enteric fever. The attack begins gradually with abdominal tenderness, meteorism, and diarrhœa. There are moderate fever of subcontinuous or remittent type and rapid wasting. The diagnostic criteria are those already mentioned under acute miliary tuberculosis. Ascites, a doughy distention of the abdomen, the presence of enlarged mesenteric glands or a sausage-shaped omental tumor are confirmatory data in tuberculous disease.

Sepsis may present the same difficulties in diagnosis from enteric fever in this period as in the first week. The points of differentiation are the same and have already been set forth in sufficient detail. The long-continued symptomatic fever of deep-seated suppuration, often obscure, may in the absence of chills and sweating closely simulate enteric fever. This is especially true of the deep abscesses which occasionally occur in visceral and bone tuberculosis.

Malignant endocarditis is not rarely mistaken for enteric fever. Changing murmurs, embolism, and the presence of leucocytosis are of great diagnostic aid. The Widal reaction and blood cultures when positive as regards B. typhosus dispel any doubts as to the presence of enteric fever.

(c) DISEASES WHICH RESEMBLE ENTERIC FEVER IN ITS LATER COURSE.—Septic conditions, various complications, and intercurrent relapse greatly modify the period of decline. In fact the terminal course of an ordinarily well-characterized, uncomplicated attack of enteric fever is often as typical as the onset. The falling temperature with its remittent and intermittent curve, the cleaning tongue and urgent hunger, the clearing mind and natural sleep, all coming on toward the close of an illness of three or four weeks' duration, would almost justify a diagnosis in the absence of a history of the previous course of the attack. Yet this favorable course is often greatly modified by the above conditions.

Sepsis of obscure origin—cryptogenetic—sometimes *sepsis due to obvious causes*, as purulent effusion, abscess formation, or caries, may so dominate the clinical picture as to raise a doubt in regard to the true nature of the primary attack. In default of a satisfactory anamnesis the methods of the laboratory, especially blood cultures, the examination of the urine for B. typhosus, and the Widal test are in many cases essential to a positive diagnosis. Complications, as pleurisy with effusion, bronchopneumonia, pulmonary abscess or gangrene, malignant endocarditis, cystitis and pyelitis, and various nervous diseases may assume such a degree of prominence as to dwarf the significance of the early symptom-complex and raise the question as to whether or not the previous symptoms have been those of enteric fever or simply earlier manifestations of the present disease. Here also the results of laboratory research render positive assistance.

Intercurrent relapse frequently prolongs the attack to six or seven weeks. Fresh crops of rose spots, the character of the temperature range, which may after having been strongly remittent again become subcontinuous, the persistent enlargement of the spleen, and the other symptoms of a specific rather than a septic infection afford the criteria for a diagnosis. If these symptoms are ill defined or there be secondary infection or marked and grave complications, the diagnosis of intercurrent relapse remains uncertain.

Uræmia in its chronic forms may suggest enteric fever at the later periods of its course by a rapid and feeble pulse, dry and fissured tongue, stupor, wandering delirium, subsultus, and continued fever of mild type.

The urinary findings, the condition of the arteries, a negative Widal reaction, and the previous history are usually sufficient for the differential diagnosis.

4. **Diagnosis by Exclusion.**—Enteric fever is by far the most common of the febrile infectious diseases; with the exception of intestinal symptoms, enlargement of the spleen, trifling bronchitis, and a relatively slow pulse, there are no constant evidences of visceral disease; the age at which the disease is most common and the immunity which is established by the attack are facts available in a doubtful case for the diagnosis by exclusion.

5. **A Provisional Diagnosis.**—There are cases in which a positive diagnosis cannot be made when the patient is seen for the first time. Delay may be required for the accumulation of the necessary data in the progress of the attack to a point at which characteristic symptoms appear. A provisional diagnosis becomes, under these circumstances, imperative. Pending the decision a due regard of the welfare of the patient and the community demands the exercise of all the measures of treatment and all the precautions against the spread of the disease that we would employ if a positive diagnosis were made.

THE DIAGNOSIS OF INTESTINAL PERFORATION.—The direct diagnosis of this accident rests upon the association of the symptoms to which it gives rise. There are cases, however, in which several of the more characteristic symptoms are not present, and every clinical phenomenon of intestinal perforation may show itself in the course of enteric fever in perforative lesions of other organs, as the appendix, gall-bladder, or peptic ulcer of the stomach or duodenum; while acute abdominal symptoms with or without collapse, followed by the signs of local or general peritonitis, may be the manifestations of intussusception, volvulus, strangulation of a Meckel's diverticulum, softened splenic infarct, hepatic abscess or pseudo-abscess of the mesenteric glands. Prompt recourse to surgical measures in any of these conditions may be the only means of saving life. In the face of the urgent symptoms of some grave intra-abdominal accident it is better in selected cases not to lose time in the attempt to make a diagnosis of the lesion by ordinary measures but at once to open the abdomen and ascertain the actual condition and if possible correct it. This diagnostic procedure is justified by the fact that enteric fever patients bear anæsthesia and operation well.

Prognosis. — The mortality varies greatly in different outbreaks, the range being from five to seventeen per cent. It is slightly higher in hospital than in private practice. After the first year, the prognosis is in general less favorable as the age of the individual increases. The mortality is greater after puberty; after forty it rises rapidly and in aged persons enteric fever is a very fatal disease. In respect of prognosis the infantile type—high evening temperatures with marked morning remissions throughout—is much more favorable than the adult type of the disease—high or moderate evening exacerbations with very slight morning remissions. Fat persons do not bear enteric fever well. Their powers of resistance to infections in general are less than in the spare and muscular; there is an increased tendency to parenchymatous degenerations of the viscera; the evidences of myocardial changes are early noted, and the nursing and treatment are less satisfactory. Those given to the abuse of alcohol also bear the disease badly. Women in general show a higher mortality than men, and pregnant and lying-in women attacked by enteric fever are in great danger. When the disease develops in persons suffering from chronic disease, especially affections of the heart, chronic bronchitis, emphysema, goitre, and pulmonary tuberculosis, the prognosis is less favorable than in those in previous good health. When the tuberculous patient survives the attack, the lung trouble usually manifests itself with increased intensity. There are, however, exceptions to this rule. I have seen several cases in which an apparent arrest took place after convalescence. An antecedent chronic nephritis adds to the gravity of the case. Diabetes mellitus is also unfavorable. The temperature is not usually high and sugar disappears from the urine, but the resisting powers of the patient are much impaired.

The intensity of the infection as shown by high temperature maxima, the limited range of the remissions, prolongation of the fever, and the prominence of nutritional disorders and nervous symptoms is of prognostic importance. The outlook is relatively much more favorable in the cases in which the fever is moderate and the associated symptoms of mild degree—typhus levissimus—and in those in which, while the fever is high and the morbid phenomena severe, the course is short—abortive cases. In general an abrupt rise of temperature occurs in the latter group of cases and is therefore not without value as indicating a short attack. Even in these cases a severe complication, a relapse, or the gravest accidents—hemorrhage, perforation—may occur. These events are, however, much more common in cases otherwise severe.

The maintenance of the power of the heart as indicated by the pulse is very important in prognosis. So long as the frequency remains low as compared with the fever and the volume is fair, the outlook is relatively favorable; but an increased pulse-frequency, associated as it almost invariably is in this disease with loss of power, is of grave prognostic significance. This is particularly the case when there are developed at the same time such evidences of circulatory failure as pulmonary hypostasis, faint cyanosis, coolness of the extremities, pulmonary œdema, and symptoms of collapse. The prognosis becomes progressively more ominous as the frequency of the pulse increases. In children and neurotic individuals, especially women, a frequent pulse is less significant, particularly when it again becomes slower.

As a general rule the gravity of the case is proportionate to the intensity of the nervous symptoms. Continuing delirium, stupor, coma, and particularly coma vigil, are of grave import; so also are meningeal symptoms, apoplectiform seizures, and local and general convulsions. Less alarming are the psychic derangements which occur toward the close of the attack and during convalescence—postfebrile insanity.

The prognosis is rendered unfavorable by the development of complications and sequels. These are, as has been already stated, more numerous than in any other infectious febrile disease and not infrequently determine the outcome of the case.

Intestinal hemorrhage, when slight and occurring early in the attack, is not necessarily unfavorable. Large bleedings occurring early are distinctly so, for four reasons: they indicate grave local lesions and usually also intense infection; they debilitate the patient and lower his powers of resistance; they are often repeated after a longer or shorter interval and in some instances are followed by perforation, and finally they constitute a distinct contraindication to the treatment by systematic cold bathing.

Intestinal perforation is almost always followed by the fatal issue. The patient succumbs in the course of a few days to the consecutive peritonitis. In rare instances a longer period may elapse and a very few cases of spontaneous recovery have been noted. A favorable prognosis cannot be made and the only chance for the patient lies in prompt surgical intervention. Peritonitis without perforation constitutes a complication of most gloomy import. It is probable that a proportion of the cases described as perforation with recovery have been instances of this kind.

Finally the prognosis is much influenced by the general management of the individual case and treatment. Skilful nursing, a careful dietary, the avoidance of drugging, and systematic cold bathing have reduced the death-rate to about seven and one-half per cent. The earlier the treatment is instituted the better the result.

PARATYPHOID FEVERS.

Definition.—A group of infectious febrile diseases, caused by organisms intermediate between Bacillus typhosus and Bacillus coli and presenting the clinical phenomena of enteric fever.

Etiology. — There are a number of organisms in this intermediate series, including the Bacillus enteritidis, and several varieties causing diseases in animals. Buxton has suggested the following classification: "PARACOLONS.—Those which do not cause typhoidal symptoms in man. A group containing many different members but culturally alike. PARATYPHOIDS.—Those which cause typhoidal symptoms. (A) A distinct species culturally unlike the paracolons. (B) A distinct species culturally resembling the paracolons."

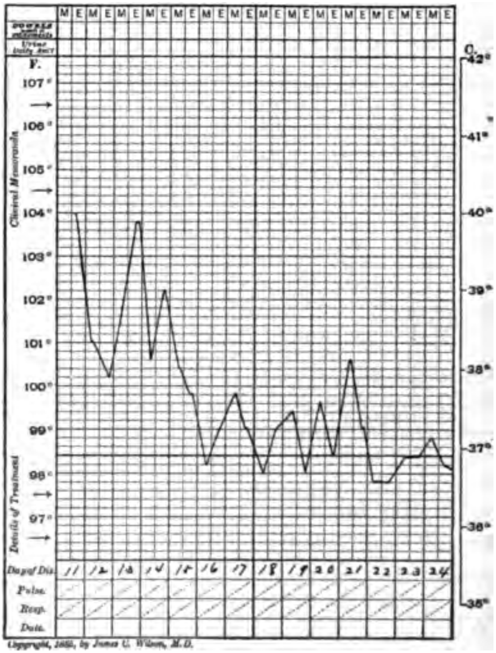


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FIG. 213.—Paratyphoid fever.—Jefferson Hospital.

Cases of paratyphoid have been reported from all parts of the world in which systematic laboratory work in bacteriology is carried on. It has occurred in series of enteric fever cases, in house epidemics, and under circumstances which render it probable that it is sometimes a water-borne disease.

Symptoms.—The features of reported cases are variable.

1. Many of the cases cannot be distinguished from enteric fever except by the failure of the blood-serum to agglutinate B. typhosus and its power to agglutinate the organisms of this group. Hemorrhage, crural phlebitis, and relapse occur. 2. Others present the clinical features of septic infections and resemble the so-called typhoid septicæmia or enteric fever with intercurrent or terminal sepsis. The diagnosis is reached by exclusion rather than by the ordinary clinical features of enteric fever. 3. Finally the organisms have been found in abscesses in cases in which no history of enteric fever has been obtained.

The first group of cases, those which are clinically indistinguishable from enteric fever, are almost always mild and terminate in recovery. The anatomical lesions are therefore as yet undescribed. The last group is without interest in this respect. Cases of the second group may end fatally. Wells and Scott (1904) studied a fatal case of their own in connection with four fatal cases collected from the literature, one of which occurred in my service in the Pennsylvania Hospital and was studied by Longcope. The most constant lesion was enlargement of the spleen. The intestinal conditions were variable. In two of the cases the intestines were normal. Ulcers were present in the others, but they resembled those of dysentery rather than of enteric fever. In all the cases the solitary follicles, the Peyer's patches, and the mesenteric glands were unaffected. The other changes present were those of a septicæmia.

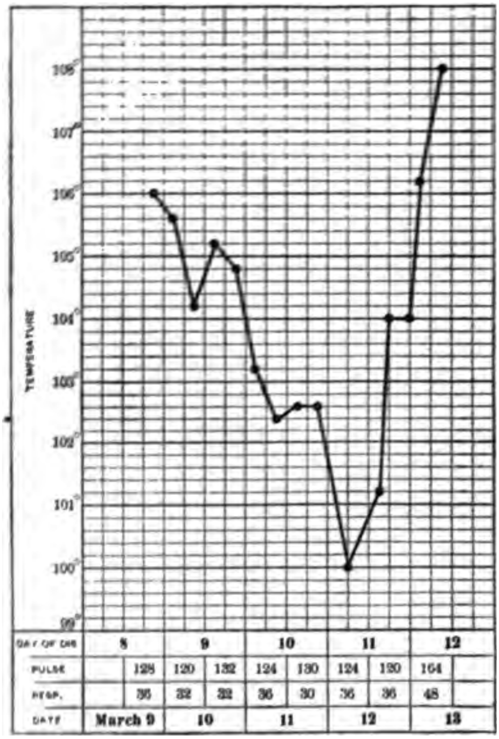


FIG. 214.—Fatal paracolon infection.—Pennsylvania Hospital.

Diagnosis.—The direct diagnosis of paratyphoid rests upon the failure of the blood-serum to agglutinate B. typhosus and its power to agglutinate B. paratyphosus or B. paracoli. It is in the highest degree probable that reported cases of mild enteric fever and cases of the so-called septicæmic variety have been instances of paratyphoid. Whether or not a separate disease should be recognized merely upon the agglutinating properties of the blood-serum is open to question. That conditions not to be clinically differentiated from enteric fever may be caused by a number of allied pathogenic organisms has been fully established.

II. TYPHUS FEVER.

Typhus Exanthematicus; Jail, Camp, or Ship Fever.

Definition.—An acute, infectious, epidemic disease characterized by sudden onset, intense fever, a peculiar rash, at first macular, later petechial, great prostration, marked nervous symptoms, a defervescence usually critical, and an average duration of fourteen days. There are no characteristic anatomical lesions.

Typhus—τῦφος smoke—used by Hippocrates to define a confused state of the mind with a tendency to stupor, expresses a prominent condition of the disease. Typhus is now a rare disease. Its practical disappearance dates from the period when enteric fever became prevalent. Sporadic cases and small local epidemics of typhus occasionally occur in great cities, particularly in Great Britain and Ireland and in Russia.

Etiology.—PREDISPOSING INFLUENCES.—Typhus is a disease of cold and temperate climates. Its prevalence is but little influenced by the season of the year or by meteorological conditions. Overcrowding, domestic and personal filthiness, insufficient food, and intemperance are important predisposing influences to typhus. It is a disease of the poor and under-fed of large cities. Age and sex are without influence. Occupation is without influence except as it involves actual exposure to the contagion, as in the case of hospital attendants, physicians, clergymen, and those who care for the dead.

THE EXCITING CAUSE.—The infecting agent has not yet been demonstrated. Typhus fever is readily transmissible from the sick to the well by actual contact, by means of the atmosphere to short distances, and by fomites. The claim of Nicolle that the common louse—Pediculus vestimenti—may transmit typhus to monkeys awaits confirmation. The attack confers an immunity which in most instances is permanent.

Symptoms.—The period of incubation is about twelve days. It may be less. Prodromes, as lassitude, vertigo, headache, loss of appetite, and restlessness at night, occasionally occur.

STAGE OF INVASION.—The onset is abrupt and marked by a chill or chilliness, followed by fever. In children the onset may be attended by vomiting or convulsions. The skin is hot, the face flushed, the eyes injected, headache is constant and severe. There is a feeling of dulness and confusion, with vertigo and tinnitus aurium. The patient complains of pain in the back and soreness of the limbs and joints. Early muscular weakness, an extreme sense of prostration, confusion of mind and failure of memory occur. Delirium is an early symptom; it may be mild and wandering or active and noisy. The tongue, at first large, pale, and coated with a thick fur, presently becomes brown and dry. Appetite is lost; there is thirst; the secretion of saliva is diminished; taste is perverted and a stale, unpleasant odor loads the breath. Constipation is the rule. The pulse is full but compressible. It soon grows feeble and varies in frequency from 120–130. Dicrotism is uncommon. The temperature rises rapidly. By the third or fourth day it may reach 103°–105° F. (39.5°–40.5° C.), and continues at this range with moderate morning remissions until the crisis. Hyperpyrexia, 107°–109° F. (42°–42.7° C.), not infrequently precedes death.

Stage of Eruption.—On the fourth or fifth day, rarely later, the eruption appears. It consists of numerous roseola-like spots of irregular outline and from one to three lines across, scattered singly or arranged in close-set groups like the rash of measles. These spots are at first of a dirty rose color, slightly raised above the surface of the surrounding skin, and disappear upon pressure. In the course of a day or two they become darker and are then no longer elevated but appear as faint, dirty brown stains without defined margins. A little later petechiæ show themselves at the centre of many of these spots. The spots fade during the first half of the second week and disappear with or without desquamation toward its close. In many of the cases petechiæ appear about the time the typical rash begins to fade. A faintly reddish, lightly defined mottling or marbling of the skin between the spots or groups of spots also occurs. This mottling has been described from its appearing to lie beneath the surface as the subcuticular eruption of typhus. The appearance of the rash varies greatly, the differences being determined by the general abundance of the two eruptions, by the relative preponderance of one or the other, and by the extent of the petechiæ. The spots and mottling together constitute the "mulberry rash" of Jenner.

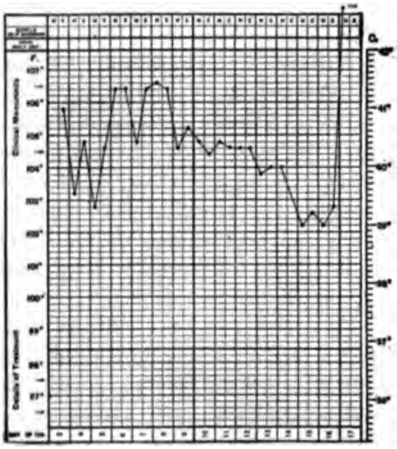


Fig. 215.—Typhus fever—fatal on 16th day of attack.

The eruption usually appears first on the sides of the chest or abdomen and spreads in a brief time over the body and limbs. It rarely occurs upon the neck or face. It may first appear upon the back of the hands. The roseola-like rash may be absent altogether, the faint subcuticular mottling alone being present. The entire absence of eruption is very rare. Sudamina are not common. The odor surrounding the patient has been regarded as characteristic.

Stage of Nervous Prostration.—During the second week the depression becomes profound. Headache passes into delirium; drowsiness and stupor are marked and in severe cases there is a tendency to coma. The delirium continues. Coma vigil, subsultus tendinum, and picking at the bedclothes may occur. The conjunctivæ are deeply injected; the pupils contracted; deafness is common. The flushing of the face gives place to a dusky pallor and emaciation progresses. There is cough and râles are heard in all parts of the chest. Hypostatic congestion occurs. The heart's action is faint; the pulse small, weak, often difficult to count—ranging from 112-140 or more. A systolic murmur in the mitral area is not infrequent. The area of splenic dulness is increased. The state of the bowels now varies from constipation to irregular, scanty dejections or moderate diarrhœa. The urine is decreased in amount, high colored, and frequently albuminous. In severe cases the discharges are passed involuntarily or there is retention of urine. Areas of the skin subjected to pressure show a tendency to slough. The surface now becomes cooler and is often moist.

Death in the malignant cases occurs in the course of a few days—*typhus siderans;* more commonly between the tenth and seventeenth days. The mode of death is by coma or by asphyxia in consequence of sudden pulmonary engorgement or by failure of the heart, the pulse becoming imperceptible, the surface cold, livid, and bathed in sweat. In the milder cases and especially in children the rash is slight, petechiæ are absent, and defervescence takes place at the end of the first or the beginning of the second week. In the average cases defervescence takes place about the fourteenth day by crisis, the temperature falling in a single night or in the course of twenty-four or forty-eight hours to the normal or even below it. Convalescence is rapid, relapse extremely rare.

Complications and Sequels.—Laryngitis, bronchitis, and bronchopneumonia are common; gangrene of the lung may occur. Independently of scurvy, which has been a frequent concomitant in typhus epidemics, bleeding from the nose, gums, bowels, urinary passages, and the vagina has been noted, as well as the spitting and vomiting of blood. In certain epidemics gangrene of the extremities, the nose, and the genitalia, and cancrum oris have occurred. Septic parotitis and arthritis and subcutaneous abscesses occur. Various palsies are met with.

Diagnosis.—The DIRECT DIAGNOSIS of typhus during epidemics is usually a simple matter. In early or isolated cases the nature of the disease must remain in doubt until the appearance of the eruption. The abrupt onset, initial chill, and sudden rise of temperature are important. The critical defervescence about the fourteenth day is characteristic.

DIFFERENTIAL DIAGNOSIS.—ENTERIC FEVER (see p. 634). RELAPSING FEVER has often prevailed in connection with typhus. The stage of complete apyrexia, the clear mind, epigastric pain and tenderness, absence of eruption, low death-rate, and the spirochætæ of Obermeier serve to differentiate this disease from typhus. CEREBROSPINAL FEVER may at the onset resemble typhus. Associated headache, vomiting, and painful rigidity of the muscles of the back of the neck, Kernig's sign, and in fatal cases characteristic lesions are of diagnostic importance. The presence of the Diplococcus intracellularis meningitidis in the fluid withdrawn by lumbar puncture will determine the question. PLAGUE.—Nausea and vomiting, pallor, and the early appearance of glandular swellings are characteristic. The duration of the plague is much shorter than that of typhus and the mortality greater. MALARIA.—The malignant malarial fevers of tropical and subtropical climates occasionally present strong resemblances to typhus but they are endemic, not contagious, unattended by specific eruptions, show greater enlargement of the spleen and in the blood the malarial parasite. MEASLES and typhus in children are attended by a somewhat similar eruption about the fourth day of the attack. In measles catarrhal phenomena are prominent during the stage of invasion; the eruption, which first shows itself upon the face, is brighter in its tints and rarely petechial. ALCOHOLISM.—Certain cases are attended by trembling delirium like that occasionally seen in typhus. Shivering, headache, pains in the limbs, fever, and eruption are absent.

Prognosis and Mortality.—The mortality ranges from 10 to 20 per cent. It is much influenced by age; not exceeding 4 per cent. under ten years and rising above 50 per cent. after sixty. Individual peculiarities unfavorably affecting the prognosis are intemperate habits, disease of the kidneys, gout, obesity, and mental depression.

TABARDILLO: *Mexican Typhus.*—This infectious disease is probably identical with European typhus. The period of incubation appears to be longer; the onset is less abrupt and the defervescence more commonly by lysis than by crisis. The eruption is less often petechial. The duration of the febrile movement is about the same and the intense headache and backache, prostration, vertigo, gastro-intestinal symptoms, apathy and delirium correspond to the typhus of Europe. McCampbell, who has carefully studied the subject, believes tabardillo to be a variety of typhus, the departure from type being due to the influence of temperature and other climatic conditions. This observer found a marked leukocytosis in all the cases examined, exceeding 30,000, in three cases terminating fatally. The erythrocytes were normal or slightly increased; the hæmoglobin ranged from 85 to 95 per cent. and the coagulativity of the blood was much decreased. The post-mortem findings are not characteristic. The actual pathogenic agent remains unknown. It cannot be grown in culture by the ordinary methods, is probably not filterable and is apparently capable of transmission to the monkey—Macacus Rhesus—in which it causes a febrile infection. The experiments of McCampbell and those of Ricketts and Wilder, especially the immunity tests of the last two observers, are highly suggestive of the transmission of tabardillo by the louse.

BRILL'S DISEASE.—Brill (1898) reported a series of cases of acute infection closely resembling enteric fever but lacking the characteristic phenomena of that disease. He subsequently extended the series to 221 cases and gave a detailed description of it, highly suggestive of typhus. Lewis has reported cases observed in Philadelphia, where I have also seen a number. The cases are usually admitted as enteric fever. They are characterized by intense headache, apathy and prostration, an abundant erythematous, papular eruption and a subcontinuous pyrexia terminating in crisis or rapid lysis in two weeks or less. In rare instances two or more cases have occurred in the same dwelling. It has not occurred immediately or remotely in other persons when cases have been treated in the general wards. The case mortality is low. The identity of the disease described by Brill with typhus fever has been definitely determined. (Anderson, Goldberger.)

III. RELAPSING FEVER.

Febris Recurrens; Spirillum Fever.

Definition.—An acute, infectious, epidemic disease caused by the spirochæta of Obermeier, characterized by a febrile paroxysm of five to seven days terminating by crisis, an interval of complete apyrexia of about the same duration and one or more abrupt relapses. There are no characteristic anatomical lesions.

Relapsing fever has prevailed extensively in Europe and particularly in Ireland, usually in association with typhus fever. It has occurred in India and other tropical countries. Brief outbreaks have occurred in America.

Etiology.—PREDISPOSING INFLUENCES.—Destitution, filth and overcrowding play an important part. Climate and season have no direct influence upon the development or propagation of relapsing fever. Age, sex, and occupation are without influence except that attendants upon the sick are apt to contract the disease. In the great Irish outbreaks a large portion of the hospital cases were wandering musicians, pedlers, beggars, and tramps.

THE EXCITING CAUSE.—Obermeier in 1873 demonstrated in the blood an organism which is the specific cause of relapsing fever. This micro-organism is a slender spirillum or spirochæta varying in length from 16 to 40μ, twisted spirally in from ten to twenty turns. In fresh blood it is very active. Under a low power it shows itself by the commotion among the blood-corpuscles, caused by its rapid movements. Culture experiments have not been satisfactory. These micro-organisms are present in the blood only during the febrile paroxysm. About the time of the crisis they disappear and are not found during the apyrexia. At this period minute, highly refractive bodies stated to be spores are seen in the blood. Upon the occurrence of relapse active spirochætæ are again found in the blood. Relapsing fever may be produced in man by inoculation with the blood of a patient, and several instances are recorded where infection has followed wounding of the hands at autopsies. Koch, Van Dyke, Carter, and others have produced the disease in monkeys by inoculation. Tictin produced an attack in a healthy monkey by inoculation of blood sucked by a bed-bug from an infected monkey. This observation explains many facts relating to the spread of the disease. In monkeys killed ten hours after the crisis the parasites are found in the phagocytes in the spleen. They are not present in the secretions or excretions. They have been found in the blood of the fœtus. Immunity from subsequent attacks does not occur. Second and third attacks in the same individual in the course of a few months have been observed.

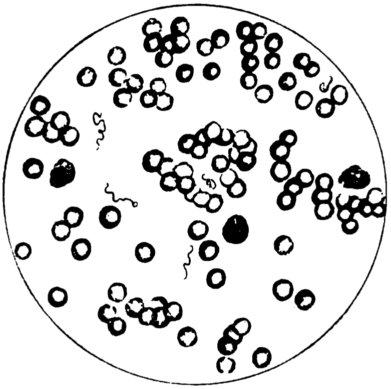


FIG. 216.—Spirillum of relapsing fever in blood.

Symptoms.—Incubation varies from five to seven days. It is exceptionally only twenty-four hours. The Primary Paroxysm.—Prodromes are as a rule absent. The onset is marked by chills or chilliness, rapid rise of temperature, headache, and pain in the back and limbs. Sweating is common. Appetite is lost and nausea and vomiting are sometimes persistent. The tongue is usually moist, covered with a thick white or yellowish-white fur. It is apt to continue in this condition throughout the paroxysm. Rarely it becomes dry or shows a dry brownish streak in the middle. The bowels are constipated. In many cases jaundice occurs. There is no characteristic eruption. Sudamina and facial herpes occur. As early as the second day there is distress in the epigastric zone. The liver and spleen are now found to be enlarged, the latter reaching some distance below the ribs. There is marked tenderness in the splenic and hepatic areas. General muscular pain and soreness are prominent symptoms. These pains are especially severe in the calves of the legs and are aggravated by movement and pressure. The mind is usually clear, delirium rare. Sleeplessness is a distressing symptom. Epistaxis is not common. The fever is intense, 104°–107° F. (40°–41.7° C.), and subcontinuous in type. Pulse is frequent, 110–130, of moderate fulness and tension, often quick, and sometimes dicrotic. Between the third and tenth days, commonly on the fifth or seventh, crisis occurs. The defervescence takes place in the course of a few hours and is frequently attended by profuse sweating or diarrhœa. The temperature may fall several degrees below the normal. In rare instances the crisis is attended by transient violent delirium. In feeble persons collapse may occur. The urine not infrequently contains albumin. The Intermission.—The crisis nearly always sets in during the night. The patient in the course of a few hours experiences remarkable relief. The pains, headache, and gastric symptoms promptly disappear with the fever. There is first a feeling of weakness but strength augments from day to day. This period usually lasts about a week. In some instances it does not exceed four or five days. In a limited number of cases the relapse does not occur. The Relapse.—Between the twelfth and twentieth days from the beginning of the attack, but commonly on or about the fourteenth day, the patient, with the same suddenness as before, again falls ill. The relapse sets in usually at night. The symptoms are a repetition of those of the primary paroxysm but less severe and the relapse is of shorter duration, usually about three days, sometimes not more than twenty-four hours. Occasionally a second relapse, less frequently a third, occurs and in very rare instances a fourth has been noted. Convalescence.—The close of the attack, especially after repeated relapses, finds the patient much prostrated. There is marked emaciation, and the convalescence is tardy, weeks often elapsing before the health is restored.

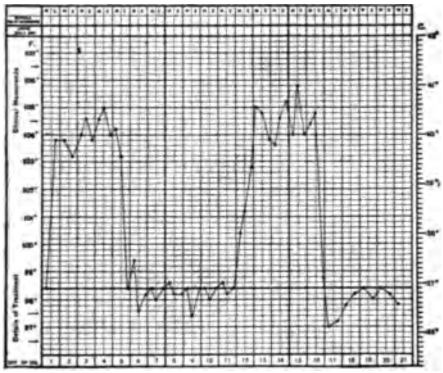


Fig. 217.—Relapsing fever.

Complications and Sequels.—Mild bronchitis is not uncommon. Pneumonia has been frequent in some of the epidemics. Chronic valvular disease and myocarditis render the patient liable to sudden death from syncope. The sudden swelling of the spleen may cause rupture of its capsule. Nephritis is a rare complication. Hæmatemesis and hæmaturia have been noted. Parotid bubo has been a prominent complication in some epidemics. Pregnant women almost invariably abort or miscarry during the course of relapsing fever. This accident exceptionally occurs in the first paroxysm, commonly in the second. The fœtus even at the approach of term perishes and the life of the mother is often lost. Forms of ophthalmia have been common sequels in some of the epidemics. Palsies may occur as the result of peripheral neuritis.

Diagnosis.—THE DIRECT DIAGNOSIS rests upon the abrupt onset, high temperature, enlargement of the liver and spleen, the critical defervescence at the end of five to seven days, and the occurrence of relapse. Spirochætæ in the blood render the diagnosis positive. Lowenthal used the serum test upon active spirilla with positive results in 30 out of 39 tests. In 14 cases he was able by this method to make a diagnosis after the disease was past, thus determining the true nature of a previous illness of doubtful character.

Prognosis.—The prognosis is, as a rule, favorable. The mortality in different epidemics varies between two and four per cent. Death may occur from exhaustion, at the relapse, after repeated relapses, or from collapse at the time of crisis.

IV. THE VARIOLOUS DISEASES.

Variola—Smallpox.

Definition. — An acute, infectious, endemic and epidemic disease, highly contagious, characterized by fever of typical course and a general eruption which passes through the progressive stages of macule, papule, vesicle, pustule, and crust.

Etiology.—PREDISPOSING INFLUENCES.—Smallpox may be regarded as the prototype of contagious diseases. It is endemic and in the absence of vaccination occasionally epidemic in every climate and among all races. Outbreaks are more common in the great centres of population, but when the disease is transported to countries in which it has never or not recently prevailed, as in Iceland or in North America among the aborigines, it has raged as a veritable scourge. The negro races are peculiarly susceptible and suffer more severely than whites.

AGE confers no immunity. The fœtus in utero may develop the disease if the pregnant mother has contracted it. Miscarriage as in other grave infections is liable to occur and the fœtus may be born with the signs of the disease or the child at term may develop it within the period of incubation. In rare instances the fœtus may bear the scars. Sometimes the child in a smallpox hospital is born without signs of the disease, and may, if at once vaccinated, escape. Such children are very delicate. Welch has seen a case at the age of eighty-three. SEX is without influence. The menstrual period and pregnancy are supposed to render individuals especially liable to contract the disease. Questions relating to vaccination and revaccination and exposure render generalizations in regard to these conditions useless. Previous disease has no influence. Neither acute nor chronic affections confer immunity, save in so far as patients suffering from acute infections such as scarlet or enteric fever, measles, or influenza are less exposed to the contagion in limited epidemics than persons going about.

The Exciting Cause.—Bodies resembling protozoa in the lesions were first described by Guarniere—*Cytoryctes variolæ*. Later Councilman and his associates demonstrated a protozoon with a cytoplasmic stage and a double cycle and small structureless bodies in the lower layer of the epithelial cells. Various observers have confirmed these findings. These organisms bear a definite relation to the lesions and the hypothesis that they are the cause of the disease is tenable.

The infecting principle is thrown off in the expired air and in the exhalations from the skin, in the secretions and excretions, and in the crusts of the unruptured and ruptured pocks formed during desiccation. The disease is transmissible during the whole course of the attack from the initial stage, before the appearance of the exanthem, until the dried crusts have entirely separated and the person and clothing of the patient have been disinfected. It may be communicated by approach, contact, by a third person himself immune, and by any articles serving as fomites. The dried scales and pus and the discharges from the nose and mouth floating in the air as dust play the chief rôle in the dissemination of the virus, and it is by this means that transmission through the atmosphere, in the absence of any communication, has taken place at distances of one hundred metres or more. It is inoculable by means of the lymph of the vesicles, pus, crusts, and the blood—the contents of the vesicles being most virulent at the time when turbidity appears, the blood during the early stages of the attack. The corpses of those dead of variola communicate the disease to susceptible persons not only in the performance of autopsies or dissections but also in their ordinary disposal for burial. The danger is greatest in the immediate proximity of cases, but under certain conditions it extends to remote distances. In this connection the part played by flies and other insects is not to be overlooked. The poison is not only virulent, it is also in the highest degree tenacious and persistent. Infected clothing that has been packed away may after several years give rise to the disease and thus become the unsuspected cause of outbreaks in localities long free from the disease. Cases have been traced to baled rags brought from distant countries as an article of commerce. It clings to articles of furniture, carpets, and rooms, and is liable in times of epidemics to infect cabs and other public conveyances.

The usual mode of access is by way of the inspired air. The susceptibility to the disease is in the absence of vaccination almost universal. Natural immunity has, however, been observed, and very rare instances are now encountered in which, vaccination having been unsuccessful, even when repeated, the individual has failed to contract variola upon exposure. Temporary immunity in unvaccinated persons has also in rare cases been observed. An acquired immunity results from the attack

In most instances it is permanent. Second attacks are exceedingly rare and third attacks almost unknown. Louis XV, of France, who had smallpox at fourteen, died of a second attack at the age of sixty-four. The immunity acquired by vaccination is of variable duration, the limit of which varies between five and ten years.

Variola is transmissible to monkeys by inoculation, and among the domestic animals, the cow and horse, a local reaction takes place. The variolous disease of sheep is analogous to but not identical with smallpox in the human body.

Symptoms.—Cases of smallpox present wide variations in intensity and clinical manifestations, from a malady trifling in itself to an overwhelming illness terminating in death as early as the third or fourth day. The differences mainly but not exclusively appear at the time of the eruption, the symptoms of the stage of invasion being much more constant. For purposes of description the following scheme is convenient:

A. Variola vera—Smallpox.
    (a) V. discreta—Discrete smallpox.
    (b) V. confluens—Confluent smallpox.
    (c) V. hæmorrhagica—Hemorrhagic smallpox.
        i. Purpura variolosa.
        ii. V. pustulosa hæmorrhagica.
B. Variola modificata—Modified smallpox.
    (a) Varioloid.
    (b) Variola sine eruptione.

The period of incubation varies from five to fifteen days. In the majority of cases it is twelve or thirteen days. The incubation is apt to be shorter in the malignant forms of the disease. At the time when inoculation was practised the local reaction and constitutional symptoms frequently appeared toward the end of the third or during the fourth day. Prodromes are as a rule absent.

The course of the attack may be divided into the stage of invasion, the stage of eruption, and the stage of desiccation and decrustation.

1. **Invasion.**—The initial symptoms are acute, usually intense, exceptionally mild. There is no constant relation between the severity of this stage and the gravity of the subsequent course of the attack. The mildest varioloid may begin with violent symptoms. On the other hand, symptoms of slight intensity at the onset are not often followed by confluence or grave hemorrhagic conditions. The attack usually begins with a chill which may be repeated several times during the first twenty-four hours. In young children a general convulsion may take the place of the chill. Severe headache usually frontal, dizziness, pain in the back, and vomiting occur. The temperature rises in the course of some hours to 103°–104° F. (39.5°–40° C.) and frequently reaches maxima of 105°–106° F. (40.5°–41.1° C.). Its type during the stage of invasion, namely, until the signs of eruption begin to appear, is subcontinuous, with slight morning remissions. The respiration and pulse are accelerated, the former not infrequently reaching 30–36 per minute, the latter 120–140. The pulse may be full and bounding; in grave and malignant cases it is often

feeble and soon becomes irregular and intermittent. The skin is hot and dry, the cheeks reddened, the conjunctivæ injected. The tongue is at first slightly swollen, indented by the teeth, and covered with a thick, moist, yellowish-white fur. Pharyngitis appears early and there is pain upon swallowing. The breath is foul. Thirst, loss of appetite, and nausea sometimes leading up to repeated vomiting, accompany the fever. The nervous symptoms of the onset persist throughout this stage. Headache, dizziness, and pains in the back and limbs become even more severe. Insomnia alternates with light slumber and delirium, sometimes wandering, sometimes furious. The lumbosacral pain is excruciating and during an epidemic, when associated with high fever, headache, and vomiting, is of diagnostic importance. In severe cases occasionally there is marked precordial oppression. Physical examination of the heart and lungs yields negative results. Exceptionally a few scattered râles are heard. The area of liver dulness is not increased. The spleen is usually palpable; it may remain normal in cases of varioloid and in hemorrhagic cases. Constipation is the rule. The urine is scanty and high colored. Febrile or toxic albuminuria is common. Hæmaturia is a frequent attendant condition in *purpura variolosa*. The blood shows no characteristic changes. It does not tend to form rouleaux. There is rapid disintegration of red cells ; during the fever they are normal or increased, but upon the occurrence of defervescence the number of red cells diminishes suddenly. Regeneration takes place slowly. In hemorrhagic cases the anæmia comes on quickly and is proportionate to the amount of blood extravasation. There is no leucocytosis in the mildest cases such as occur in vaccinated persons nor in the initial stages of graver cases. Leucocytosis does not appear in the absence of complications until suppuration takes place, and is due to infection by pus organisms and not to the poison of variola itself. Menstruation is excessive and if the onset of smallpox occurs toward its close the flow is increased and prolonged.

During this stage the so-called initial or accidental rashes occur. They are more common in some epidemics than others, but are encountered in from ten to fifteen per cent. of all cases. Two varieties may be recognized which differ in form, distribution, and in prognostic importance. The more common, *roseola variolosa*, is macular, suggesting the eruption of measles, though it does not present the characteristic papules nor their grouping in crescents. This rash usually appears upon the second day and disappears within twenty-four hours, never persisting after the appearance of the pocks. It comes out, as a rule, first upon the face, next upon the body, and finally in abundance upon the extremities. It is fully developed in the course of some hours and then fades somewhat more slowly. It is more common in mild than in severe cases. The second form, *erythema variolosa*, is much less common. It appears early, usually upon the first day, and may in some cases antedate the fever and other constitutional phenomena. It consists of a vivid dark crimson efflorescence, throughout which are scattered numerous purpuric spots of varying size,—*hemorrhagic erythema*. The distribution of this rash is remarkable. The regions involved constitute the "triangles of Simon," of which the first, the more common, has its base line across the abdomen, its lateral boundaries along the inner portions of the thighs, and its apex at the knees; the second, of which there are usually two, occupy the lateral thoracic region, the axillæ, a portion of the inner surface of the arm, and extend forward upon the chest. This rash lasts until after the true exanthem appears. It gradually fades, the purpuric spots more slowly than the surrounding erythema. Within the limits of these triangles the variolous exanthem frequently comes out less abundantly than elsewhere upon the surface of the body. Some observers, as Hebra, noted this form of the initial rash more commonly in females. It is of unfavorable prognostic omen. The duration of the stage of invasion is three days. In rare cases the eruption may first appear toward the end of the second day; more rarely still, not until toward the close of the fourth day.

2. **The Stage of Eruption.**—From the time of the appearance of the exanthem the divergence of the clinical varieties begins. The essential difference between *variola vera* or true smallpox and *variola modificata* or varioloid consists in the fact that in the former suppuration takes place in the fully developed pocks, with well-marked secondary fever, while in the latter most of the pocks undergo involution from the vesicular stage without further constitutional disturbance.

A. Variola Vera.

(a) **The Discrete Form.**—About the end of the third or the beginning of the fourth day the eruption appears. It shows itself first upon the face and scalp, particularly at the edge of the hair, and in some cases upon the wrists. It spreads downward over the trunk and extremities. By the close of the third day of the eruption and sixth of the attack it is fully developed and the surface is more or less thickly covered with pocks, which are more abundant and advanced upon the face, where they first appeared, than elsewhere. Here and there are to be seen scattered individual pocks that appear later than those which surround them. The exanthem is often conspicuously copious in local areas which are submitted to habitual pressure by the clothing, as the collar, corset, or garters, or recently irritated by some application, as iodine or a sinapism. It is usually less abundant in the hypogastric region and inner surfaces of the arms and thighs than elsewhere and upon the lower than the upper extremities.

As the eruption comes out the temperature falls, the constitutional symptoms subside, and the patient feels so much better that he regards himself as convalescent.

The evolution of the pock is as follows: The macule consists of a red spot, disappearing upon pressure and varying in size from the head of an ordinary toilet pin to a split pea. The color and distribution of the rash at this period suggest measles and the differential diagnosis, especially in adults, may be difficult. Within twenty-four hours a distinct hard papule appears which feels like a shot embedded in the skin. This rapidly becomes acuminate and there develops at the summit a minute vesicle with clear contents, which gradually extends to the size of the papule and becomes tense from the increase of contained lymph. The greater number of the fully developed vesicles present a well-marked and highly characteristic central depression—*primary umbilication.* At the centre of this depression may be found in many but not in all of the pocks a hair follicle or the duct of a sebaceous gland. Finally the clear, opalescent contents become cloudy, then opaque and yellow, the vesicle is converted into a pustule—*stage of maturation*—and with this change the umbilication disappears and the fully developed pock becomes hemispherical. The pustule is surrounded by a distinct areola several millimetres in width and the skin is slightly swollen. By the third day the pock has reached its full development and enters upon the stage of involution or desiccation. Resorption of the contents rapidly takes place, the roof of the pustule sinks in,—*secondary umbilication,*—hard, yellowish-brown or blackish crusts form, which gradually separate, leaving abruptly defined, shallow scars, with glistening red bases, which in the course of time become white and finally disappear. If the cutis has been implicated permanent scars—pittings—are formed. The resulting disfigurement in ordinary discrete smallpox is commonly slight.

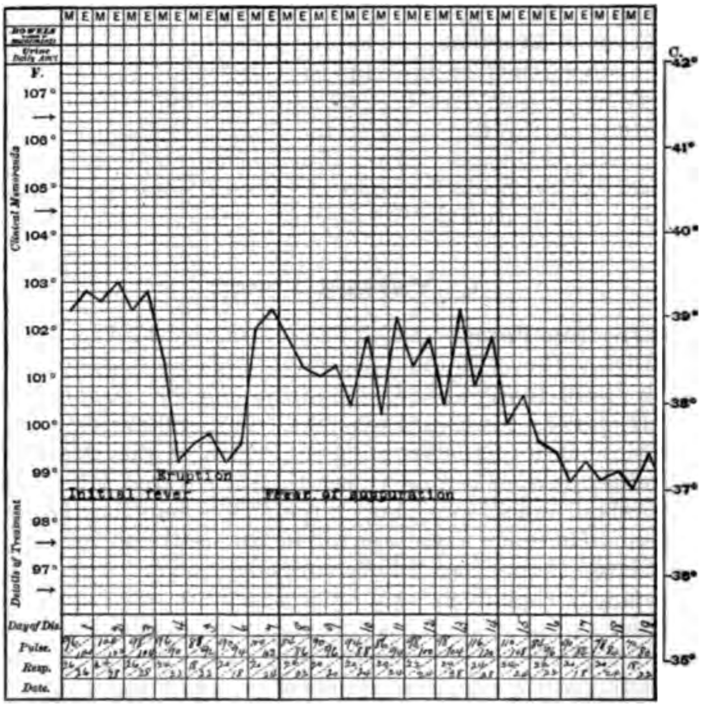


FIG. 218.—Discrete smallpox. Moderate fever of suppuration; recovery.—Royer.

The time occupied by the successive stages is approximately as follows: macular one day; papular one day; vesicular three days; pustular three days; desiccation five to ten days. Puncture of the vesicles with a fine needle is followed by the escape of a portion only of its lymph, which is enclosed in several spaces limited by septa. Upon the palms and soles the pocks are as a rule scanty and owing to the thickness of the epidermis they remain for some time deeply embedded. Early in the second week of the attack with the maturation of the pocks secondary fever and more or less severe constitutional symptoms occur. The skin is swollen, tense, and sore, especially upon the face, the eyelids are tumid, and the countenance greatly disfigured. There is marked leucocytosis. The secondary fever rises rapidly to about the range of that of the initial stadium and falls by lysis which in many cases is rapid so that defervescence is complete and the patient enters upon convalescence in thirty-six or forty-eight hours, namely, about the eleventh or twelfth day of the attack. The fever may, however, last several days. Delirium is common in severe cases and suicidal tendencies may show themselves. General septicæmia may develop.

The mucous membranes exposed to the air are involved usually at the same time with the skin; not rarely earlier. The nasal chambers, the buccal and pharyngeal surfaces, the palate, and the larynx and trachea are the seat of a more or less abundant eruption. The tongue is less frequently attacked, though now and then pocks may be observed upon the border and its under surface. The anal, preputial, and vulvar regions are later affected. The pocks upon mucous membranes are at first analogous to those upon the skin. Under the influence of heat and moisture in the vesicular stage their roofs undergo maceration and discrete superficial ulcers are formed. The mucous lesions are associated with catarrhal processes and add greatly to the sufferings of the patient. Among the symptoms to which they give rise are, in the mouth, pain and difficulty in swallowing, hoarseness and aphonia, and excessive secretion, and about the meatus urinarius in both sexes, distressing pain in micturition. These symptoms are greatly aggravated in the stage of suppuration. Among persons not protected by vaccination the discrete form has fortunately in all times been the most common.

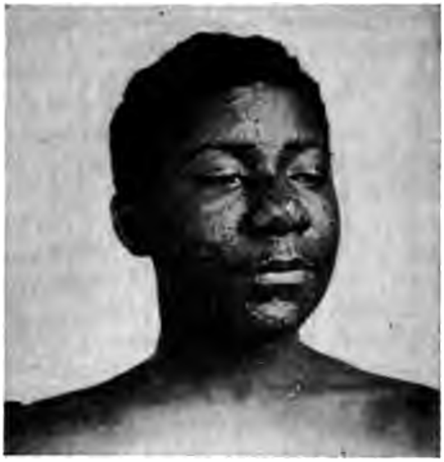


FIG. 219.—Discrete smallpox.—Royer.

(b) **The Confluent Form.**—The pocks are closely set and run together, especially upon the face, hands, wrists, and feet. This grave form of the disease is encountered among those children and adults alike who have not been protected by vaccination and revaccination. It has not been especially common in particular epidemics nor is it transmitted from person to person. On the other hand the mildest case of varioloid may give rise to an infection resulting in *variola confluens*, while the latter may cause in a partially protected person *variola discreta* or *varioloid.* Personal predisposition must therefore enter largely into its causation.

The invasion symptoms usually are very severe. The eruption appears some twelve or eighteen hours earlier than is common in the discrete form—in some cases by the end of the second or the beginning of the third day. The earlier its appearance the greater the danger of confluence. Its efflorescence is rapid so that by the end of the second day, the fourth or fifth of the attack, it has invaded the entire body from the head to the feet. The remarkable remission of fever and amelioration of the general symptoms seen upon the appearance of the eruption in the discrete form seldom occur. As a rule the improvement in this respect is only partial, fever persisting throughout the attack and becoming intense as suppuration takes place. The skin is swollen and hyperæmic; the individual pocks in the papular stage are small and crowded upon the skin; they are rapidly converted into vesicles, which, increasing in size, soon become pustules. During this process confluence takes place and extensive superficial abscesses are formed. The face in severe cases presents the appearance of a thick yellowish mask. Upon the arms and legs the pocks are numerous and limited patches of confluence are sometimes seen, especially upon parts subjected to pressure, as the buttocks, while they remain discrete upon the trunk. The swelling of the hands and feet increases and these parts are the seat of most distressing tension, pain, and tenderness. The eruption is abundant upon the mucous membranes of the nose, mouth, pharynx, and larynx. Confluence may give rise to extensive superficial ulceration. Deep abscess formation may occur in the tonsils or in the retropharyngeal

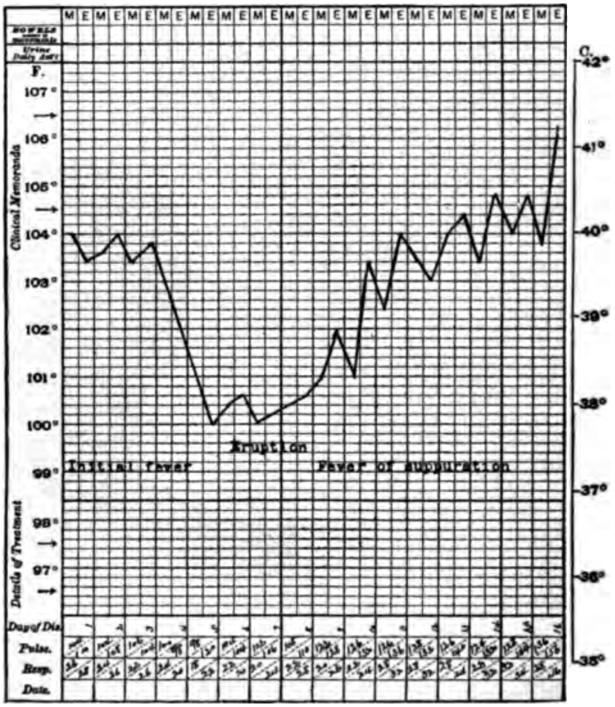


FIG. 220.—Confluent smallpox. Incomplete defervescence upon appearance of rash; severe secondary fever; death on the 16th day.—Royer.

tissues and necrosis of the laryngeal cartilages which may be associated with œdema of the glottis. The cervical lymph-glands are enlarged and tender. At this period purulent metastases in distant organs frequently develop. Toward the end of the first or at the beginning of the second week the fever rises to 104° F. (40° C.) or higher, the pulse to 120. Delirium, very often maniacal, sets in; the patient is often with difficulty restrained from leaving his bed, and with the signs of a profound toxæmia, progressively feebler and more frequent pulse, subsultus, involuntary discharges, or preagonistic hyperpyrexia, death closes the scene. When recovery occurs, the cutaneous and constitutional symptoms undergo gradual improvement, and irregular fever may even in the absence of complications prolong the convalescence into the fourth week.

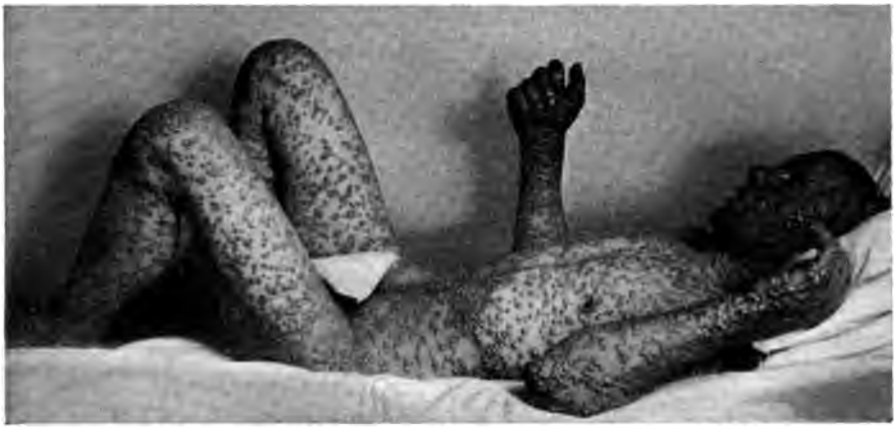


FIG. 221.—Confluent smallpox.—Royer.

3. Stage of Desiccation and Decrustation.—The areola fades; the pustule sinks and becomes flattened; its edge is sharply defined against the surrounding skin and separation gradually takes place, the entire process occupying in V. discreta two weeks or more and in V. confluens a longer time. As a rule the desiccation begins in the face and scalp, where crusts may be seen, while upon the extremities the exanthem is still in the pustular stage. In some instances the desiccation takes place on all parts of the skin at the same time. Many of the pustules break and the exuding contents dry in the form of broad thin crusts. The process of desiccation is attended by intense itching. The pocks upon the palms and soles are limited in number and form hard circumscribed nodules in the thick epi-

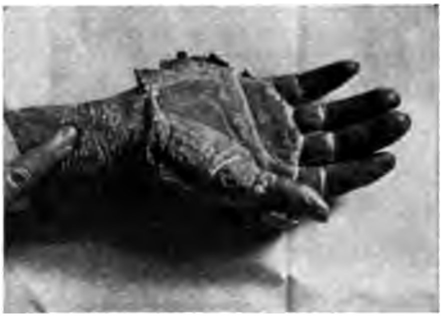


FIG. 222.—Exfoliation of the palmar epidermis containing embedded pocks occurring in the later stage of severe smallpox.—After Welch and Schamberg.

dermis, which in the course of three or four weeks undergo separation and may be picked out. The hair usually falls out and in some cases the nails are lost. In the confluent form the thick epidermis of the hands and feet is sometimes cast off entire. The crusts upon separation sometimes reform and the ulcerative lesions of the skin heal slowly by granulation. Upon the face the resulting scars are much more disfiguring than the pitting of V. discreta. They are extensive, of irregular outline, and intersected by lines and bands which gradually undergo contraction, causing ectropion of the eyelids and lips and interfering with the muscles of expression.

The appearance of the patient in the stage of maturation, particularly in V. confluens, is horrible. The swollen face, thickly covered with pustules and blebs, some of which are broken and exude a sticky pus, or with a hideous mask of necrotic skin, the tumid and closed eyelids, the distorted nose and lips, the disfigured ears, the foul secretions, and the stench which surrounds the wretched being create an impression not to be forgotten and merit the popular adjective *loathsome* applied to the disease.

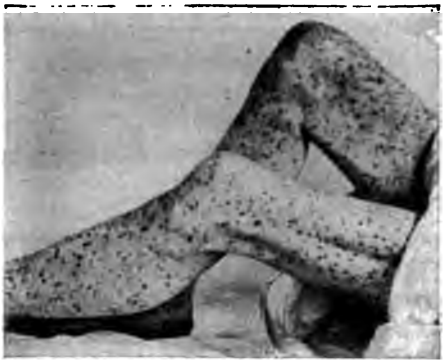


Fig. 223.—Hemorrhagic smallpox (Ross V. Patterson).

(c) **Hemorrhagic Forms.**—i. **Purpura Variolosa.**—This is the most malignant form of variola. It is fortunately comparatively rare and in some epidemics no cases have been observed. It may occur at any period of life, is less common among children than grown persons, and affects as a rule young and vigorous adults. The influence of vaccination and especially of repeated revaccination is of the greatest importance in preventing this clinical manifestation of the variolous infection. The incubation is short—five to eight days. Prodromes, especially lumbosacral pains, are not uncommon. The invasion is attended with profound constitutional disturbance. Fever may be moderate, but there is great prostration; the pulse is small and frequent, the respiration accelerated, and the patient experiences a feeling of overwhelming illness. Headache and backache are severe and precordial and epigastric distress are often associated with vomiting and purging. The mind remains clear. Commonly upon the second day, sometimes earlier, a diffuse, scarlatiniform rash makes its appearance upon the lower part of the body and the extremities and shortly thereafter upon the face. Purpuric spots of varying size rapidly appear. Ecchymoses invade and frequently entirely cover the face. The conjunctivæ, eyelids, and loose tissues adjacent are distended with a sanguinolent œdema and in a short time the greater part of the surface of the body is involved in a livid, purplish-red discoloration. Mucous hemorrhages are common, epistaxis, bleeding from the gums, and hæmaturia being the usual forms; hæmoptysis, hæmatemesis, and melæna less frequent. Metrorrhagia is common and pregnant women abort. In rare instances death may take place without the occurrence of free hemorrhage. Very often there is no trace of the exanthem. If life be prolonged a few scattered blood-tinged papules may be discovered upon the forehead and wrists. This form of variola terminates in death within a week and very often as early as the fourth or fifth day.

ii. **Variola Pustulosa Hæmorrhagica.**—This form is much more common than the preceding and usually occurs in feeble and cachectic persons and drunkards who are not protected by vaccination. The attack develops as an ordinary severe case of variola, which becomes hemorrhagic in the vesicular or pustular stage. Exceptionally bleeding takes place into some of the pocks while yet in the papular stage. The bleeding is in many instances restricted to the eruption upon the lower part of the body and the lower extremities. Blood extravasation may also involve the skin beyond the pocks and free hemorrhages from mucous surfaces may lead up to the fatal issue. The earlier in the course of the attack the hemorrhages appear the graver the outlook. Death commonly results at the end of the first or in the beginning of the second week. Recovery may occur in cases in which hemorrhage into the pocks does not take place until the stage of suppuration. A distinction must be made between this form of variola and cases of V. discreta in which, owing to mechanical violence, pressure, or other accidents, blood is extravasated into a few vesicles or pustules. To the latter group must be referred cases of hemorrhage into the pustules of the legs in patients who have gotten out of bed during their delirium. Osler describes a series of six cases in which hemorrhage into the vesicles was followed by "a rapid abortion of the rash and speedy recovery."

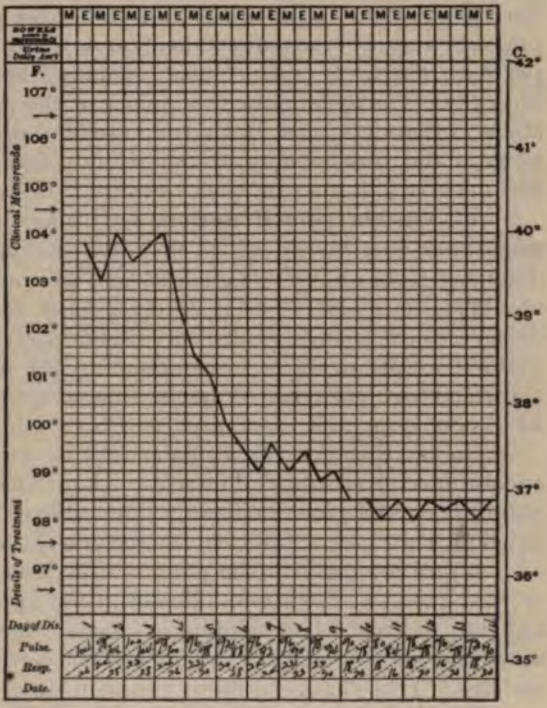


FIG. 224.—Severe varioloid.—Royer.

B. Variola Modificata.

(a) **Varioloid.**—This term is applied to the modified form of smallpox which occurs in persons who possess a partial immunity as the result of vaccination and revaccination. It is characterized anatomically by the fact that the typical exanthem causes as a rule only superficial lesions upon the skin and therefore rarely leaves permanent scars; clinically by the absence of the stage of suppuration and the secondary fever and a shorter and more favorable course. The period of incubation is the same. The onset is abrupt and in many cases the initial symptoms are severe. Generally the symptoms of the stage of invasion are relatively mild. Headache and backache may, however, be intense. If an initial rash appears it is the macular variety—*roseola variolosa*. The papules appear toward the close of the third or in the beginning of the fourth day, first upon the face and then elsewhere, spreading from above downward. They vary in number from ten or twelve upon the face and hands to a copious eruption distributed over the entire surface. Within forty-eight hours their appearance is complete, although during the latter part of this period a few fresh papules may be recognized here and there among those which first appeared. The fever ends at once by crisis and the general symptoms rapidly improve. The development of many of the pocks is arrested in the papular stage; others abort early in the vesicular stage and in some few the contents of the vesicles may become cloudy and slightly opaque. Desiccation sets in early and goes on with rapidity. Convalescence is usually complete.

(b) **Variola sine Eruptione.**—During outbreaks of variola cases are sometimes observed in which no trace of eruption can be discovered. The diagnosis rests upon etiological as well as upon clinical grounds. A history of exposure, sudden onset, fever, intense lumbosacral pains, and critical defervescence upon the third day justify a provisional diagnosis of smallpox without eruption. The transmission of the disease to others would render the diagnosis positive. Variola sine eruptione occurs in young persons who have been well vaccinated and invariably runs a favorable course.

The modification of variola caused by artificial inoculation is no longer encountered in western countries. About the eighth day local reaction was manifest at the point of inoculation. Fever and constitutional symptoms developed and were followed by a typical exanthem, not usually copious.

A rare anomaly in the pock is described under the name of horn-pox or wart-pox—*V. verrucosa*. The eruption appears upon the third or fourth day but instead of developing as usual the papules undergo desiccation upon the fifth or sixth day and are converted into dense warty or horny nodules. This change is more common upon the face than elsewhere.

Complications and Sequels.—The complications are not numerous and mostly develop during the stage of suppuration. They consist mainly of extensions of the suppurative inflammatory process in the skin or mucous membranes or of metastatic infections. It follows that they are more common and severe in proportion to the extent and intensity of the suppuration—in V. confluens than in V. discreta, and infrequent in varioloid. Bed-sores and acute gangrene are frequent in severe cases. Erysipelas is not uncommon. Phlegmonous inflammation of the skin may occur. Furunculosis and acne are often troublesome during convalescence. Superficial erosions in the larynx may in healing give rise to adhesions which result in permanent hoarseness; the cartilages may be involved and acute œdema of the larynx may cause sudden death. Lesions of the larynx play an important part in the causation of bronchitis and bronchopneumonia which is perhaps the most common of the complications. Croupous pneumonia is infrequent; pulmonary abscess may occur. Purulent pleurisy has been common in some epidemics. Cardiac complications are infrequent. Myocardial changes are observed. An apex systolic murmur may occur. Pericarditis is rare. Simple endocarditis is not common in smallpox. Malignant endocarditis has been in some instances found post mortem. Venous thrombosis may occur during the later course of the attack. In the digestive tract parotitis and inflammatory affections of the other salivary glands occasionally occur. Their frequency varies in different epidemics. Pseudomembranous angina is common in severe cases and especially in the hemorrhagic forms. The vomiting of the initial stage is not apt to persist. Diarrhœa is frequent, especially in children. Dysenteric symptoms are frequently mentioned by the older authorities but are rare at present. Complications in the urinary tract are infrequent. Toxic albuminuria is common; nephritis rare. Hæmaturia is usual in the malignant cases. Inflammation of the ovaries and of the testicles—orchitis variolosa—may occur. Pregnant women usually abort. Pyæmic arthritis and acute necrosis of bones or cartilages may, as in the other severe acute infections, develop during the convalescence. Diabetes mellitus is a rare sequel.

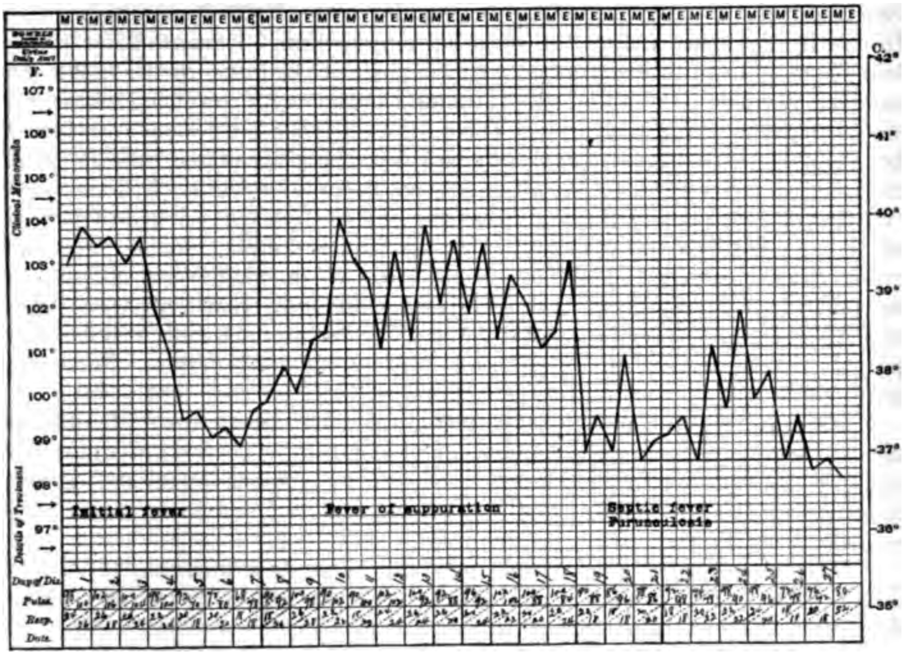


FIG. 225.—Confluent smallpox; furunculosis as a sequel.—Royer.

The nervous system is especially prone to react to the variolous infections. In children general convulsions are common at the outset and may be repeated. In adults and especially in alcoholics maniacal delirium may terminate in coma. Post-febrile insanity sometimes occurs. It is less common than after influenza and enteric fever and commonly ends in recovery. Epilepsy is a very rare sequel. Purulent meningitis and encephalitis are likewise rare complications. Hemiplegia has been observed in a few instances. Transient aphasia has been noted. Paraplegia may occur at any time during the attack. It begins abruptly, is usually subacute, and may involve the sphincters. In some instances the symptoms have been those of acute ascending paralysis and the termination rapidly fatal. The palsies of the lower extremities and the monoplegias observed occasionally are manifestations of an infectious neuritis. Ataxic symptoms may occur. Paralysis of the soft palate analogous to that which follows diphtheria sometimes occurs. Paralysis of individual muscles or muscle-groups, as the deltoid, and circumscribed areas of cutaneous anæsthesia have been described.

Otitis media purulenta is a frequent complication. It results from the extension of the catarrhal inflammation by way of the Eustachian tube. It usually develops during the stage of suppuration. Exceptionally purulent disease of the mastoid arises with necrosis of the bone, or the labyrinth may be involved. Serious affections of the eye with resulting blindness were common in former times. They are relatively infrequent at present because of the greater attention now given to the early ocular lesions. The conjunctivitis incident to the disease may become chronic. Diffuse keratitis may occur and result in ulceration and perforation with destruction of the eyeball. Iritis and choroiditis are less common. In the purpuric cases retinal hemorrhages may occur. Pocks develop very commonly upon .the outer surface of the eyelids and result in scar formation with ectropion and its attendant evils. Much less commonly they involve the palpebral conjunctiva and very rarely the ocular conjunctiva. Synechiæ may result.

External and internal nasal deformities sometimes result from the ulcerative processes and subsequent scar formation. Necrosis of the cartilaginous septum with perforation, occlusion of a nasal chamber, partial adhesion of the soft palate, and loss of the sense of smell are among the sequels of smallpox.

Diagnosis.—A correct diagnosis where there is a question of variola is one of the most critical and important of the duties of the physician. A mistake may be the cause of an extended and disastrous epidemic.

The DIRECT DIAGNOSIS of typical variola is unattended by difficulty after the appearance of the eruption. In the atypical and modified forms the difficulties are frequently insurmountable. During the prevalence of an epidemic every case of sudden illness is suspected. The sudden occurrence of headache, rigors, intense backache, epigastric pain, nausea and vomiting, and high temperature is important. The initial rashes may be misleading. The measly rash—*roseola variolosa*—bears in many instances some resemblance to measles, for which the disease may be mistaken. More commonly, however, during an epidemic measles is mistaken for variola. The scarlatinal rash—*erythema variolosa*—lacks as a rule the vividness of the eruption of scarlet fever and differs from it in distribution, occupying the abdominocrural or pectoral triangles of Simon. In fact the occurrence of this rash in connection with the foregoing symptoms renders the diagnosis of smallpox almost positive. A history of exposure and the absence of vaccination scars are of diagnostic importance. The development toward the close of the third or at the beginning of the fourth day of an eruption of red macules upon the face, scalp, and wrists, rapidly becoming papular and feeling like shot embedded in the skin, coincidently with a marked remission in the febrile movement, renders the diagnosis positive. In any doubtful case, especially in the negro, the buccal and faucial mucous membrane should be carefully examined.

In malignant hemorrhagic smallpox death may occur before the characteristic rash develops. If the patient survive to the end of the third or fourth day a few shrunken, shotty papules may be felt about the roots of the hair and upon the wrists.

DIFFERENTIAL DIAGNOSIS.—Smallpox during its initial stage presents some points of superficial resemblance to the following diseases: 1. SCARLET FEVER.—There is erythematous sore throat, more or less painful. The rash is brighter and more intense than the erythematous initial rash of smallpox, in which petechiæ are very commonly present. It appears first upon the chest and throat and is rapidly diffused. 2. MEASLES.—The stage of invasion is accompanied by marked catarrhal symptoms. The rash appears about the fourth day but is distinctly maculopapular, lacking the shot-like firmness of the variolous papule and showing a crescentic arrangement. There is no remission in the febrile movement upon the development of the rash. 3. TYPHUS.—The eruption is rare upon the face and when present in this region is comparatively faint. The macules are not hard or elevated. There is no fall of temperature upon the appearance of the eruption.

During the vesicular and pustular stage smallpox may be mistaken for: 4. VARICELLA.—Errors in diagnosis between these two diseases are very common. In varicella initial symptoms are usually absent. The eruption is always discrete and appears in successive crops. It is usually not observed until it has reached the vesicular stage. The vesicles are irregularly oval or circular, tensely distended with a transparent fluid. They are clear, bright, and pearly. Primary umbilication does not occur, but in rare cases, as the contents of the vesicles undergo resorption, the roof of the vesicle falls in, giving rise to the condition known as secondary umbilication. 5. PUSTULAR SYPHILIDES.—Individual pustules occasionally bear a close resemblance to the variolous pock. The polymorphous character of syphilitic eruptions, their symmetrical distribution, their persistence, the presence of mucous patches, the history of the case, and the nature of the febrile movement when present serve to render the differential diagnosis a relatively easy matter. 6. PUSTULAR GLANDERS is attended with malaise, pyrexia, and pain in the limbs. There is an eruption of indurated red papules upon the summit of which pustules develop. There is a fetid nasal discharge. The invasion symptoms and the course of the disease are totally unlike smallpox. 7. CEREBROSPINAL FEVER.—The sudden onset, intense symptoms, and petechial rash may lead to errors of diagnosis. Painful rigidity of the back of the neck and spastic contraction of the limbs may be present in both these conditions. Papules should be carefully sought for at the roots of the hair and upon the wrists. Photophobia, pupillary inequalities, strabismus, and very irregular fever suggest meningitis rather than smallpox. Lumbar puncture is important. 8. DRUG EXANTHEMS.—Iodide of potassium, the bromides, and the local use of croton oil may produce rashes suggesting smallpox, but the resemblance is extremely superficial and the true nature of these affections becomes apparent upon examination.

Prognosis and Mortality.—In persons not protected by vaccination smallpox is a very fatal disease. In the older epidemics the mortality ranged from 40 to 60 per cent. and smallpox was dreaded alike from its loathsomeness, its high mortality, and its serious sequels. At the present time the mortality of smallpox is greatly influenced by:

1. Vaccination and Revaccination.—During the nineteenth century smallpox epidemics diminished in the most remarkable manner in extent and frequency and showed a correspondingly lowered mortality. Nevertheless among the unvaccinated the gravity of the disease and the death-rate are practically unchanged. MacCombie's statistics show in 3940 unvaccinated cases of all ages 1758 deaths—44.6 per cent. Among 17,756 vaccinated cases 1441 deaths, a mortality of 8.1 per cent. Welch, Municipal Hospital, Philadelphia, reported in 2831 cases of variola 1534 deaths, a mortality of 54.18 per cent., and in 2169 cases of varioloid 28 deaths, a mortality of 1.29 per cent. In Sheffield in the outbreak of 1887–8, of 4703 cases 474, or 10 per cent., terminated fatally. Of 552 patients not vaccinated 274, or 49.6 per cent., died; while of 4151 vaccinated patients 200, or 4.8 per cent., died.

The character of the vaccination is of great importance as affecting the prognosis. The relative value of multiple or repeated vaccinations is shown by McCombie's analysis of 11,724 cases. This author regards an area of ¼ to ½ square inch of well-foveated surface as indicating efficient vaccination. He found the mortality among cases with one good mark to be 6.4 per cent.; among those with one indifferent mark, 16.7 per cent. Among those with two good marks, 3.7 per cent.; among those with two indifferent marks, 11.2 per cent. Among those with three good marks 3.7 per cent.; among those with three indifferent marks 7.4 per cent. With four or more good marks 2.7 per cent.; with four or more indifferent marks 4.8 per cent., and concludes that the protection against a fatal attack is three or four times greater among patients with efficient than those with indifferently successful vaccination. W. M. Welch analyzed 5000 cases with reference to the character of the cicatrices, whether produced in primary vaccination or in revaccination, with the following result: The mortality among persons having good scars was 8 per cent.; fair scars 14 per cent.; those with poor scars 27 per cent.; average mortality in persons showing the cicatrices of vaccination 16 per cent.; mortality among unvaccinated persons 58 per cent.

Death from smallpox is rare in persons whose primary vaccination was efficient and in whom revaccination has been successful. These well-established facts are of the greatest practical importance.

2. The Virulence of the Attack.—Smallpox modified by efficient vaccination and revaccination is a comparatively trifling disease with a death-rate but little exceeding 1 per cent. of all cases. Ordinary discrete smallpox is a grave affection with a greatly increased mortality. Confluent smallpox is even more grave, the majority of the cases terminating in death, and those recovering frequently suffering from serious, often

irremediable, sequels. Finally, the hemorrhagic form—*purpura variolosa*—is invariably fatal. Petechial rashes and hemorrhagic phenomena are especially unfavorable. A rise of temperature directly after the appearance of the eruption is a bad sign. Continuing delirium, persistent high temperature, and convulsions are of grave prognostic omen.

3. THE PATIENT'S SURROUNDINGS.—Unfavorable hygienic conditions, overcrowding, poverty, and want greatly increase the mortality.

4. THE OCCURRENCE OF COMPLICATIONS.—The complications of variola affect the prognosis unfavorably. Certain epidemics have been attended with an unusual death-rate in consequence of the frequent occurrence of ordinary grave complications. The laryngeal and pulmonary complications are especially ominous.

5. AGE.—Among the conditions unfavorably affecting the death-rate in individual cases age is of great importance. In young children the disease is peculiarly fatal. Unvaccinated infants in the first year mostly die, and the mortality is high up to the tenth year. From ten to twenty years of age there is a slight decrease in the death-rate, which after the thirtieth year again rises.

6. PREVIOUS ILLNESS.—Previous severe illness and alcoholism render the prognosis unfavorable.

7. PREGNANCY.—Pregnant women are especially liable to the disease in the confluent and hemorrhagic forms. Abortion usually occurs and is apt to be followed by septic infection. To this rule there are fortunately occasional exceptions. The fœtus may show a well-developed eruption and quickly die or it may develop the eruption shortly after birth. In cases in which abortion does not occur the child may undergo the disease in utero and be after birth immune alike to vaccination and variola.

Vaccinia, Cowpox, Kinepox.

Definition.—An eruptive disease of the cow, communicable only by inoculation and causing, when transmitted to the human being, local reaction in the form of a pock and constitutional disturbances which are followed by a more or less lasting immunity against smallpox.

Vaccination.

Definition.—The artificial inoculation of vaccine virus for the purpose of producing immunity against smallpox.

Arm to arm vaccination was formerly very generally practiced in order to perpetuate the lymph and secure its greatest purity. The use of crusts came into vogue at a later period. Bovine vaccine lymph has now come into general use, and has the advantage over the arm to arm method of avoiding the opening of the vesicle and thus affording the opportunity for accidental infection and of wholly eliminating the danger of syphilis and other infections. When it is necessary to use human lymph it should be taken upon the eighth day from a typical unbroken vesicle in a perfectly healthy child at least three months old. The vesicle must be pricked at several points, care being taken not to draw blood. The lymph may be in a preserved dry state upon sterilized bone points or slips. When required for use the dry lymph is moistened by a few drops of warm sterilized water. It may also be preserved in capillary glass tubes, each containing the quantity required for one vaccination, sufficiently long to admit of sealing in the flame of a spirit lamp, thin enough to enable them to be instantaneously sealed, and strong enough to be handled and transported.

Glycerinated Lymph.—The thorough incorporation of four parts of a sterilized 50 per cent. solution of chemically pure glycerin in water with one part of lymph or vesicle pulp, and the storing of this mixture in sealed capillary glass tubes, protected from light for some weeks, is followed by the destruction not only of the ordinary saprophytic bacteria found in the lymph, but also of tubercle bacilli and the streptococcus of erysipelas. Lymph thus treated is fully as efficient as ordinary lymph.

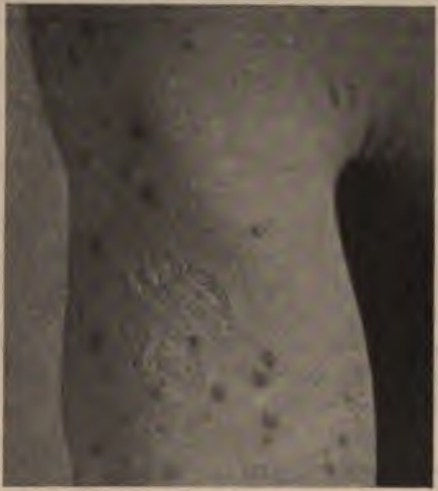


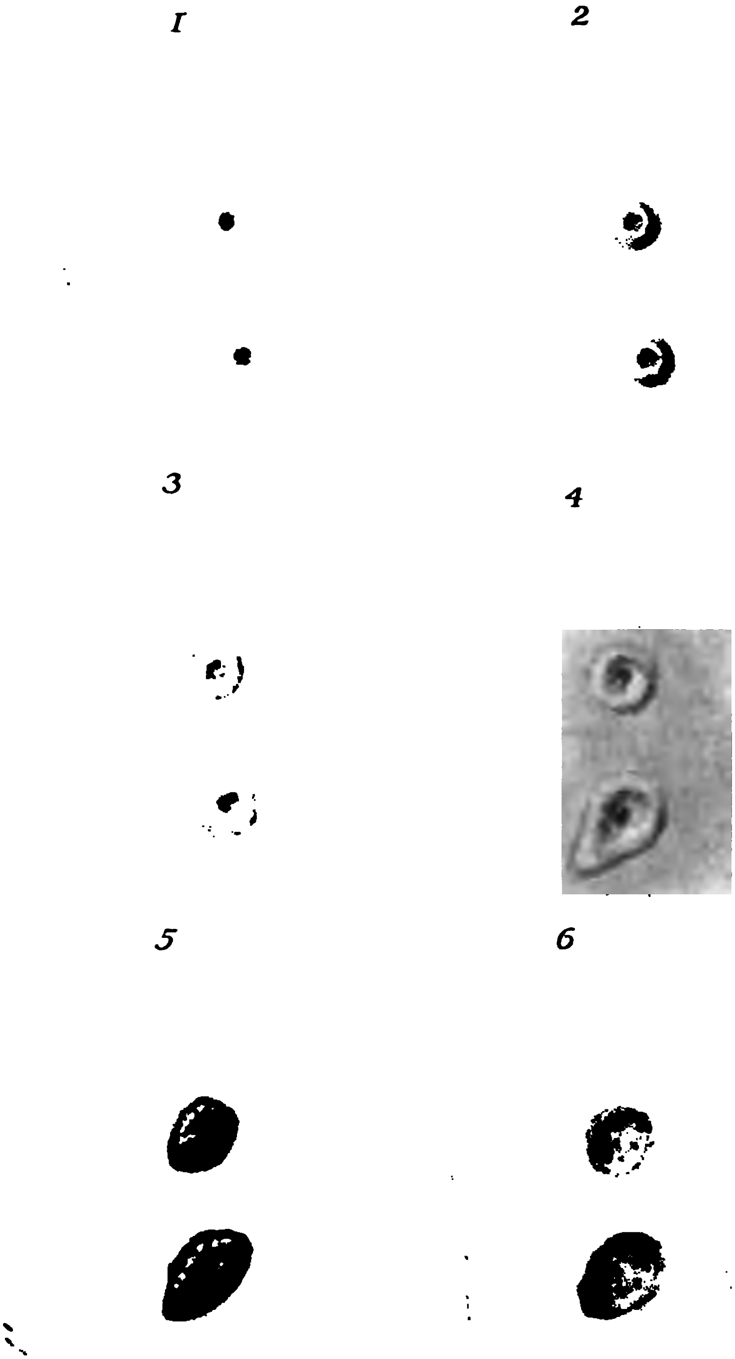
FIG. 226.—Scars from an infantile vaccination.—After Welch and Schamberg.

Vaccinia produced by humanized lymph has a somewhat more rapid evolution than that caused by bovine virus and is attended with milder constitutional symptoms.

THE TECHNIC.—The outer surface of the arm near the insertion of the deltoid is usually selected. In infants the left arm is preferable. In females the outside of the leg just below the knee is sometimes chosen. The surface must be washed, dried with a soft towel, and the lymph inserted by puncture, multiple superficial crossed incisions, or after the removal of the epidermis by scraping. The spots are to be rendered moist by the exuding serum but care should be taken not to draw blood. For this purpose a thoroughly sterilized old-fashioned thumb lancet or an ordinary flat-headed surgical needle should be employed. The insertion should be performed at two points about an inch apart and the diameter of the abraded or scarified area should be about one centimetre. The clothing should not be replaced until the serum has thoroughly dried. A thin layer of sterilized gauze should be lightly applied and held in place by means of adhesive plaster, not encircling the limb. This should be occasionally renewed. The pock should be kept dry and clean, and may be lightly dusted with starch or toilet powder. The newborn should be vaccinated only during the prevalence of smallpox. Children are commonly vaccinated in the course of the third month. In case of failure the operation must be repeated. Persons exposed to the contagion of smallpox should be immediately revaccinated. The immunity conferred diminishes with time. Revaccination should be performed at the seventh year of age, again at puberty, and from time to time as epidemics occur.

Typical Vaccination.—The period of incubation varies from three to five days. At the end of this time local reaction shows itself in the form

PLATE XII.



TYPICAL VACCINATION.

1. Papules. 2. Vesicles with clear contents. 3. Vesicles with opaque contents. 4. Fully developed pocks with erythematous areola. 5. Crusts. 6. Appearance immediately after separation of crusts.

of one or more reddish papules at the point of inoculation. These in the course of five days develop into compound vesicles, the contents of which are at first clear but later become opaque. By the eighth day the vesicle is fully developed and is round or oval with prominent and well-defined edges and a depressed or umbilicated centre. About the tenth day an erythematous areola usually appears and the contents of the pock become purulent. The surrounding skin is swollen, indurated, and tender. Scabbing now begins at the centre of the pock and rapidly extends toward its borders. The areola fades about the end of the second week, and the pock is converted into a thick brownish crust which gradually becomes dry and hard and separates between the twentieth and twenty-fifth days after the vaccination, leaving a scar of a dusky red color which gradually becomes white and pitted or foveated. The corresponding superficial lymphatic glands, namely, the axillary or inguinal, as the case may be, during the evolution of the pock become slightly enlarged and tender.

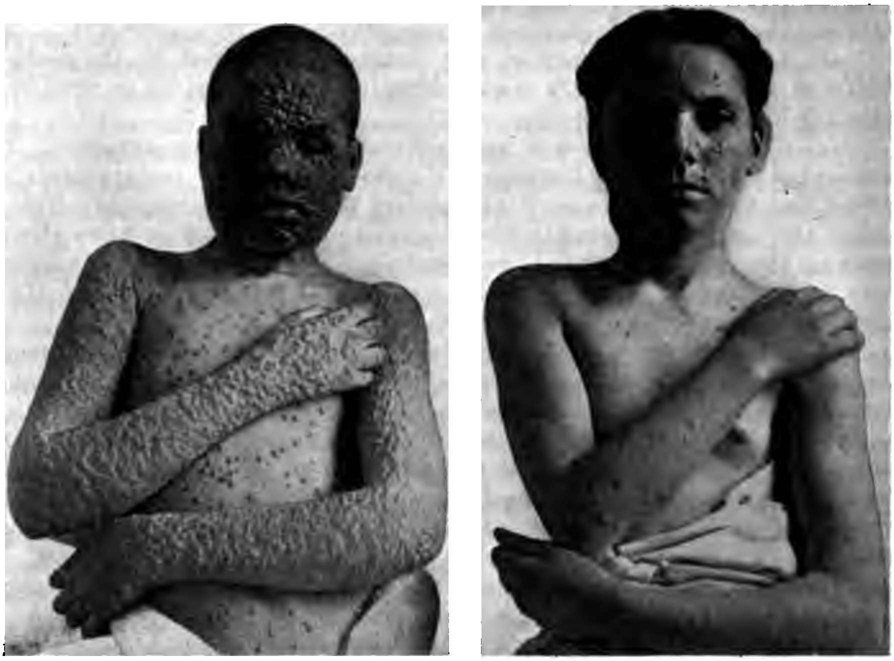


FIG. 227.—Two sisters suffering from smallpox. The one on the right was successfully vaccinated in infancy; she contracted a mild varioloid and recovered without any scarring. The other, unvaccinated, developed a severe smallpox, and recovered, though considerably pitted.—After Welch and Schamberg.

The constitutional reaction is commonly slight. It shows itself by moderate fever, restlessness at night, loss of appetite, and irritability. These phenomena usually appear upon the third or fourth day and continue until the early part of the second week. Erythema, roseola, or urticaria may develop at any time during the course of the vaccine disease. These eruptions are usually transient. Leucocytosis shows itself about the third day coincidently with the appearance of the local eruption, and again about the time the pock reaches maturity. The resulting immunity against vaccinia like that against variola varies in duration in different individuals. In rare instances it is permanent, but as a rule successful revaccination may be performed in the course of some years. The pock of revaccination, however, lacks in most instances the typical development of the primary vaccine lesion. The constitutional reaction in revaccination is sometimes severe. If no characteristic lesion follows the attempt at revaccination, the operation should be repeated once or twice at short intervals.

Atypical Vaccinia in Man.—1. VARIATIONS IN THE NUMBER OF POCKS.

(a) *Supernumerary pocks* occasionally develop in the vicinity of the original vaccine lesion.

(b) *Confluent pocks* may in rare instances be formed by the coalescence of the supernumerary pocks either among themselves or with the original lesions.

(c) *Generalized vaccinia or vaccinal eruptive fever* is less common. It consists of a vaccine rash developing in various parts of the body, especially about the wrists or on the back. Secondary pocks usually begin to develop about the eighth or tenth day after vaccination and are often more abundant on the vaccinated limb than elsewhere. The pocks appear in successive groups so that they may be seen in all stages of development. The disease sometimes lasts several weeks.

(d) *Vaccinal Eruptions Generalized by Autoinoculation.*—Supernumerary pocks may be produced by scratching with the nails after they have been in contact with the ruptured vaccine pock. They may occur in any part of the body and vary from one or two to many. The number is sometimes very great. They have been observed upon the cheek, lips, tongue, buttocks, breast, and the genital organs. On the mucous surface of the vulva the resulting ulceration may give rise to the suspicion of venereal disease.

(e) *Local vaccinal eruptions* may arise at the seat of previously existing cutaneous lesions, as impetigo, eczema, acne, or psoriasis.

2. VARIATIONS IN THE SIZE OF THE POCKS.

(a) *Two or more of the primary vesicles* caused by vaccination may coalesce to form one large pock.

(b) *The size of the pock* may be increased by coalescence of supernumerary pocks in the immediate neighborhood.

3. VARIATIONS IN THE CONTENTS OF THE POCK.—In cachectic individuals the contents of the vesicle, instead of being clear and limpid at the end of the first week, may be watery, hemorrhagic, or purulent.

4. VARIATIONS IN THE EVOLUTION OF THE POCK.

(a) *Acceleration.*—The pock develops more rapidly in summer than in winter. Its evolution is apparently hastened by idiosyncrasies on the part of individuals and by the character of the lymph employed.

(b) *Retardation.*—Cases occur in revaccination in which vesicles apparently aborted become active a week or more after the original insertion.

(c) *Abortion.*—The non-development of the pock is determined by the immunity of the patient, the quality of the lymph, and the skill of the vaccinator. In revaccination a bright red papillary lesion, "raspberry excrescence," sometimes develops about a week after the insertion of the lymph. Vesicles do not form and the papules remain hard and dense for several weeks. There is no areola, and healing ultimately takes place without the formation of a scar.

5. Variations in the Involution of the Pock.—These anomalies are determined by vaccinal injuries. Secondary infection may take place at the time of the operation or subsequently if the pock is injured. It may be due to the use of contaminated lymph or infected instruments or may arise at a later period from other causes. Vaccination is not wholly free from the danger of accident. Severe inflammation, suppuration, deep-seated ulceration, or gangrene may occur in mismanaged cases. Erysipelas is an occasional complication. Cellulitis, abscess formation, and septicæmia may occur. These accidents are, however, not peculiar to vaccination. They may arise in any lesion of the skin in default of proper antiseptic measures.

6. Variations in Healing and the Formation of Scar.—The lesion of the skin caused by vaccination is usually fully healed by the end of the third week. It may, however, remain open for some weeks. The scar sometimes manifests hypertrophy or puckering or runs into keloid.

7. Transmission of Chronic Specific Disease by Vaccination.

A. *Vaccination and Syphilis.—Vaccinosyphilis.*—Syphilis has been transmitted by vaccination, but the number of well-authenticated cases is limited. The general use of bovine lymph has rendered invaccinated syphilis a remote possibility. It may, however, occur in consequence of the use of lymph taken from an individual suffering from syphilis or from contamination of the instrument or wound or it may be due to infection from the vaccinator. The sequence of events is as follows:

If the subject be susceptible to vaccination the pocks may show no departure from the normal course, but in some instances they abort. If they be irritated, the vaccinal sore may become inflamed, suppuration may occur, and the ulcers may scab over and again break out. Whether the vaccination runs a typical or an atypical course, a chancre with indurated base eventually forms at the point of inoculation, and the signs of general infection at a later period.

Acland makes the following deductions from a well-known case of autovaccination with vaccine virus from a syphilitic child; they are in entire accordance with general observation.

(a) That vaccination can be successfully performed with lymph taken from a source tainted with syphilis without necessarily communicating that disease.

(b) That if syphilis be communicated in the process of vaccination it does not follow that all the points of insertion will become infected.

(c) That the evolution of syphilis, as regards the primary and secondary stages, is not necessarily disturbed; that it is neither accelerated nor retarded by simultaneous vaccination.

(d) That no care in the selection of lymph obviates the risk of vaccinating from an obviously tainted source.

(e) That when syphilis is communicated by vaccination, the first appearance of the disease is at the seat of puncture.

B. *Vaccination and Tubercle.*—The communication of pulmonary tuberculosis as a result of vaccination is of exceeding rarity. It may in fact be doubted whether it has ever occurred. In well-regulated vaccine laboratories the animals used are previously submitted to the tuberculin test. It has been suggested that they should, after the collection of the vaccine material, be slaughtered and submitted to examination for tuberculous lesions. In case of their presence, the vaccine material must be rejected.

In some few instances lupus has been observed at the seat of vaccination.

C. *Vaccination and Leprosy.*—The alleged cases of transmission of leprosy by vaccination are open to serious doubt.

D. *Vaccination and Cancer.*—There are no authentic cases on record in which cancer has resulted from vaccination or developed in the vaccination scar.

E. *Vaccination and Tetanus.*—The vaccine lesion, like other wounds of the skin, renders the patient liable, under certain circumstances and the absence of proper precautions, to tetanus infection. A limited number of instances of fatal tetanus after vaccination are reported.

Acland has arranged the dates at which various eruptions or complications may be looked for after vaccination, as follows:

1. During the first three days: erythema; urticaria; vesicular and bullous eruptions; invaccinated erysipelas.
2. After the third day and until the pock reaches maturity: urticaria; lichen urticatus; erythema multiforme; accidental erysipelas.
3. About the end of the first week, and generally after the maturation of the pocks: generalized vaccinia—(a) by autoinoculation, (b) by general infection; impetigo; accidental erysipelas; vaccinal ulceration; glandular abscess; septic infections; gangrene.
4. After the involution of the pocks: invaccinated diseases, for example, syphilis.

V. VARICELLA.

Chicken-pox.

Definition.—An acute infectious, endemic and epidemic disease of childhood characterized by mild constitutional symptoms and a vesicular exanthem which develops in irregular, successive crops.

Chicken-pox was formerly confused with smallpox and until recently there have been those who regarded it as a greatly modified and very mild variety of smallpox. It is now generally looked upon as an entirely distinct disease. One of these diseases never gives rise to the other; the attack of one does not confer immunity against the other, and it is no rare event for a person who has recently suffered from one to contract the other. Vaccination confers no immunity against varicella, and children who have recently suffered from varicella react to vaccination in the ordinary manner.

Etiology. — PREDISPOSING INFLUENCES. — Varicella is a wide-spread disease, endemic and frequently epidemic in the great centres of population, usually in the autumn or early spring. As in the case of other readily transmissible infections, sporadic cases occur and frequently become the centre of house epidemics or extended outbreaks. The liability is general, and scarcely any individual who has not had the disease escapes when it makes its appearance in a school or other public institution. It is a disease of childhood, the majority of cases occurring before the eighth year and few after the tenth. It is comparatively infrequent during the first year and, though rare, occasional cases occur after puberty. Sex is wholly without influence as a predisposing factor.

THE EXCITING CAUSE is not known. The disease is highly contagious and usually communicated in the ordinary intercourse of children in the family, the school, or the playground. Direct contact is not necessary, the infection being communicable at some little distance by the air and to greater distances by persons who pass from the sick to those who are susceptible, and finally by means of fomites. Inoculation experiments have shown that it is present in the contents of the vesicles. Outbreaks of varicella are sometimes associated with measles, whooping-cough, scarlet fever, or variola.

Symptoms.—The period of incubation is usually thirteen or fourteen days Prodromes as a rule do not occur. In a majority of the cases the eruption is the first sign. It first appears usually upon the face and spreads into the hairy scalp and progressively over the trunk and extremities. It sometimes comes out first upon the back and shoulders and very often at the same time upon the wrists and forearms. The pocks are more numerous upon the trunk and upon the upper than the lower portions. The rash consists of small red, scattered flat papules, circular or ovoid in shape, which rapidly develop into vesicles. They usually come out in irregular crops, fresh spots continuing to appear among the older, so that by the fourth or fifth day they are seen in all stages of evolution and involution. Some few of the papules do not develop into vesicles at all but undergo complete resolution in the course of several hours. Nearly all of them develop into vesicles which are fully formed within twenty-four hours. Not infrequently the papular stage is so brief that the fully developed vesicle appears upon skin that shortly before seemed entirely normal. The vesicles are usually at first hemispherical and appear to be superficially situated in the skin. Their contents are limpid, so that they sometimes present the appearance of a drop of clear or faintly yellowish fluid resting upon the surface. In the course of a few hours they become milky and then seropurulent and in a further brief period desiccation takes place with the formation of flat, yellowish-brown, firmly adherent crusts, which separate in about a week, leaving in the majority of instances no scar. The vesicles are readily ruptured by scratching and other injury. Under these circumstances, and when the lesion approaches more nearly to that of variola and involves the deeper structures of the skin, pitting may result, especially upon the face. It is probable that spontaneous rupture of the vesicles does not occur. The pock in varicella commonly has little or no areola, but in the severer cases marked infiltration and hyperæmia of the surrounding skin may be observed. The diameter of the vesicle varies from 1 to 15 or 20 millimetres; their number from ten to hundreds. They are in most cases discrete, but when very numerous confluence may often be discovered upon careful search. Primary umbilication does not occur, but as desiccation takes place a depression in the centre of the crust—secondary umbilication—is sometimes seen. The eruption occurs upon the mucous membrane of the mouth. It is rare upon the conjunctivæ and upon the labia and prepuce. In these situations, under the influence of warmth and moisture the roof of the vesicle is rapidly destroyed and the lesion converted into a circumscribed superficial ulcer. The peripheral lymph-glands are not infrequently slightly swollen and tender. In the lighter forms there is little or no elevation of temperature during the whole course of the disease; in the more severe cases fever, if not previously present, develops with the rash, to the abundance of which it bears, however, no constant relation. It commonly subsides in two or three days and very seldom lasts a week. It does not conform to type.

The duration of the attack is variable. Three or four weeks may elapse before the separation of the last crusts. Relapses do not occur. The immunity acquired is in most instances permanent. In rare cases subsequent attacks have been observed.

The anomalies of the disease relate to the rash. In rare instances some of the vesicles contain blood, with ecchymoses and bleeding from the mucous surfaces—*Varicella hæmorrhagica;* still more rarely they develop into bullæ like those of pemphigus or ecthyma—*V. bullosa;* in cachectic children some of the skin lesions may become extensively ulcerated or even gangrenous, and death occur as the result of exhaustion—*V. gangrenosa vel escharotica.*

Diagnosis.—DIRECT DIAGNOSIS.—Varicella in cases seen from the beginning is easily recognized. The mildness of the initial symptoms, the persistence of fever if present upon the appearance of the eruption, the character of the individual pock, which is essentially vesicular, its rapid evolution, the absence of primary umbilication, the appearance of the lesions in irregular crops, so that papules, vesicles, and crusts are seen at the same time in the same region, are of diagnostic importance. Varicella is usually endemic in cities; variola occasionally epidemic.

DIFFERENTIAL DIAGNOSIS.—This important matter principally relates to the discrimination of varicella from smallpox, and the chief points are indicated in the foregoing paragraph. In smallpox the onset is abrupt; the fever high; headache and backache intense. There are cases in which, at the period of desiccation, the differential diagnosis cannot be made.

The resemblance of urticaria, pemphigus, and other diseases of the skin to varicella is remote.

Prognosis.—Varicella is a benign affection. Convalescence is in the majority of cases uneventful and complete. In rare instances death has resulted from nephritis, sepsis, or laryngitis.

VI. SCARLET FEVER.

Scarlatina.

Definition.—An acute, infectious disease, occurring sporadically and in circumscribed epidemics, and characterized by erythematous angina, a diffuse uniform exanthem followed by desquamation, and a tendency to nephritis.

Etiology.—PREDISPOSING INFLUENCES.—No region can claim immunity from scarlet fever. It occurs in every climate and attacks all races. It may prevail at any season, but, owing to the mode of life and the closer intercourse of school children in autumn and winter, epidemics are more extensive and severe at these seasons of the year. Among personal conditions predisposing to this disease age plays an important part. Children at the breast are rarely attacked. More than half the cases occur before the fifth year and 90 per cent. before the tenth. Adults occasionally contract the disease. After puberty the liability rapidly diminishes. Sex is without influence.

EXCITING CAUSE.—The specific pathogenic germ is not yet known. Streptococci have been demonstrated in the skin and the blood during life and in the viscera after death, and the disease has been regarded as a streptococcus infection. These micro-organisms are, however, present under widely different conditions. Mallory found between the epithelial cells of the epidermis a protozoon which assumed rosette forms like the malarial parasite. Inoculation experiments upon human beings have demonstrated the presence of the infecting agent in the blood, the tears, the secretions of the nose, larynx, and bronchi, the urine, the desquamating skin, and in the contents of miliary vesicles. It is probably eliminated in the discharges from the bowels. The disease caused by artificial inoculation is usually of severe form. The infecting principle is virulent and tenacious. It is transmitted directly from the sick to the well and indirectly by means of fomites. These may be the clothing of the patient, the bedding and furniture of his room, toys, books, letters or other articles with which he has been in contact or which have been exposed to an atmosphere bearing the fine dust made up of his dried secretions or the particles of his desquamating skin. The vessels he has used and remnants of food may also convey the disease. Persons, themselves insusceptible, may be the carriers of the contagion to others at a distance. Physicians and nurses are especially exposed to this risk and the experience of the medical profession in this respect is peculiarly sad. The only way to escape catastrophe is to realize its danger. Household pets, as cats, dogs, and birds, may under certain circumstances carry the disease. Several epidemics have been traced to milk supply. It is probable, as shown by Dornblüth, that the milk has been merely the means of conveyance. The cream is especially liable to cause the disease, and the risk is avoided by boiling. The poison is not borne to any great distance in the air. Its virulence is greatly diminished by oxygen, sunlight, and diffusion. On the other hand, it shows under opposite conditions a vital persistence greater than that of any other disease. Articles of clothing folded and packed away have given rise to the disease after months and even after years. The time at which the patient becomes a source of contagion has been the subject of controversy. It is not only probable but it is also safe to assume that transmission may occur at any time, from the beginning of the attack until desquamation is completed, or, in the case of a pathological discharge, as in otitis media, for an indefinite period. The infecting principle gains access by way of the inspired air. Occasionally the attack of scarlet fever is complicated by another specific infection, as erysipelas, measles, pertussis, varicella, enteric fever, or diphtheria. The Klebs-Löffler bacillus may, however, be absent in throat affections of the most severe character, even those in which pseudomembrane is conspicuous. It is now thought that the febrile and desquamative diseases to which certain of the domestic animals, as the cat, dog, and horse, are subject—forms of "distemper"—are not, as was formerly supposed, identical with scarlet fever in the human being.

The individual predisposition to scarlet fever is much less general than to measles, variola, and many other contagious diseases. Many persons upon exposure escape. Individuals sometimes escape upon close exposure and contract the disease years later. Certain families show an immunity transmitted from generation to generation; on the other hand many families suffer from a fatal liability. The immunity after puberty is not wholly due to previous attacks. The attack confers an immunity which commonly is permanent. Second attacks are very rare. I have seen three attacks separated by intervals of several years in the same person. Certain individuals who possess an immunity acquired by the attack in early life suffer from slight sore throat when exposed to the infection.

The disease frequently occurs sporadically under circumstances in which it is impossible to trace its transmission. Any such case may become the focus of an epidemic. General epidemics are made up of series of local or circumscribed outbreaks. They last longer than epidemics of measles, sometimes several months, and show remarkable remissions and exacerbations. Epidemics of scarlet fever differ greatly in the severity of the primary disease and in the prominence of severe complications.

Symptoms.—The period of incubation varies from one to seven days; in the majority of cases the disease develops on the third or fourth day after exposure. A short incubation is commonly followed by a severe attack.

(a) STAGE OF INVASION.—The onset is usually abrupt, without prodromes. It very often comes on at night. The intensity of the initial symptoms is frequently in direct relation to the severity of the subsequent course of the disease. A chill is rare; convulsions are common, especially in young children. Vomiting may be the first symptom. It is in many cases repeated. The temperature rises in the course of a few hours to 104°–106° F. (40°–41.1° C.), and the skin is extremely dry and hot. In very mild cases the temperature may not exceed 101°–102° F. (38.3°–38.9° C.). Older children complain of sore throat and upon inspection the mucous membrane of the soft palate, tonsils, and pharynx shows a uniform erythematous blush varying in intensity according to the severity of the case. The hard palate shows a finely stippled punctiform rash. Febrile phenomena are marked—a furred tongue, red at the tip and edges, refusal of food, thirst, scanty, high-colored urine, restlessness, somnolence, and delirium. Cough and other symptoms of implication of the respiratory tract are slight or altogether absent.

(b) STAGE OF ERUPTION.—The eruption appears during the latter part of the first or in the course of the second day, coming out first upon the neck, chest, especially in the infra-axillary regions, and spreading over the face and body to the extremities with such swiftness that in the course of from twenty-four to thirty-six hours the entire surface is covered. It consists at first of minute, close-set red points, coarser and more widely separated upon the legs than elsewhere. The intervening skin rapidly becomes reddened and slightly swollen. In severe cases œdema of the hands and fingers is often marked and a like condition of the eyelids occurs.

The fully developed rash upon the back, abdomen, and thighs is of an intense scarlet or violet-red color, difficult to describe or reproduce and quite unlike that of any other eruptive disease. It has been compared, but very inappropriately, to the color of the boiled lobster. Its distribution over the surface of the body is, in the great majority of cases, nearly uniform, with the common exception of a ring around the mouth which, remaining free, is in striking contrast with the rest of the face. Exceptionally the face and neck only are involved, or the body and extremities, and in some instances, especially in adults, the rash is irregularly patchy in areas varying in diameter from 5 to 20 cm., oval or circular, not abruptly marginate but shading off into the surrounding skin. The eruption is largely hyperæmic and the white streak produced by drawing the finger over the surface immediately disappears.

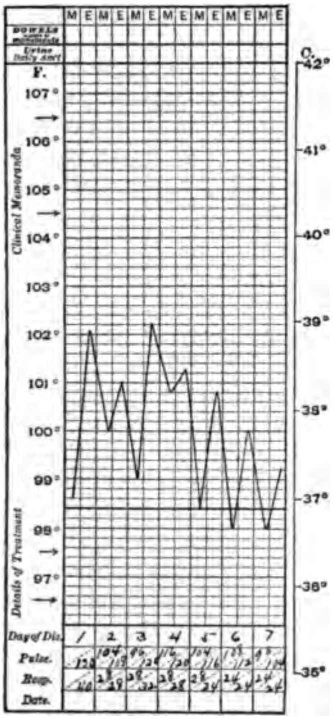


FIG. 228.—Scarlet fever.

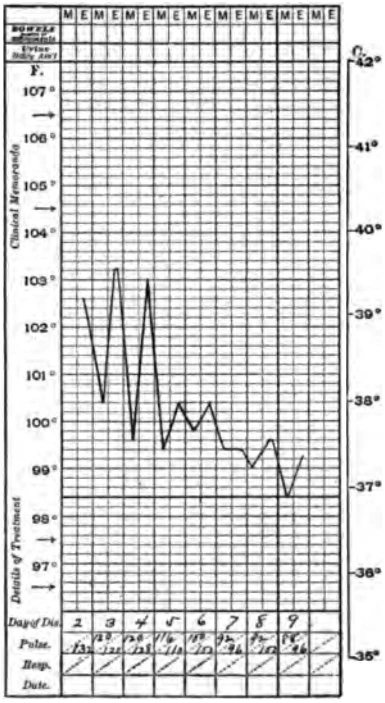


FIG. 229.—Scarlet fever.

Sudamina may appear and in a group of cases—scarlatina miliaria—the skin is extensively covered with minute yellow vesicles. In the more intense eruptions punctiform hemorrhages may appear and in malignant cases petechiæ and extensive subcutaneous hemorrhagic effusions.

Subjectively sensations of tension, burning, and pricking, together with more or less intense itching, attend the eruption and to these symptoms the restlessness and jactitation in many of the cases are largely due. Swelling of the peripheral lymph-nodes, especially in the region about the angles of the jaw, is common and in some instances they are very tender

The rash reaches its height upon the third or fourth day and gradually fades first where it first appeared. By the end of the first week it has in most instances wholly vanished.

The tongue is at first covered with a thick whitish fur through which project the tips of the swollen red papillæ—an appearance described as the strawberry tongue. In the course of a few days the coating separates, leaving the whole dorsum of the tongue red and rough, the so-called strawberry or raspberry tongue of writers. These terms are neither exact nor descriptive and ought to be abandoned. The bowels are, as a rule, constipated. Diarrhœa may be an early symptom and is sometimes severe. It may occur as the result of a simple catarrhal enteritis or of a dysenteric colitis with tenesmus and bloody stools. In grave cases meteorism may be present.

In a majority of the cases there is simply an erythematous angina with slight enlargement of the tonsils and palatine arches; in other cases these and the contiguous structures are intensely swollen and infiltrated and foci of suppuration may appear; finally, more or less extensive pseudomembrane may develop with intense inflammation, cervical adenitis, and inflammatory infiltration and œdema of the tissues of the neck. The ordinary inflammation is due to the scarlatina poison; the graver forms and many of the pseudomembranous inflammations to secondary infection by pus-producing micro-organisms, while true diphtheria occurs as a complication resulting from infection by the Klebs-Löffler bacillus. Membranous laryngitis is rare in scarlet fever and still more rare is the development of a membranous (croupous) exudate in the bronchi.

The temperature conforms much less closely to type than that of measles or variola. It is much modified by secondary infections. Its rise is abrupt, to 104°-106° F. (40°-41.1° C.); it remains with slight morning remissions about this level until the eruption is fully developed, about the third day. From this point in uncomplicated cases the temperature gradually falls coincidently with the fading of the rash, until toward the end of the first week the defervescence is complete. In mild cases the maximum temperature may not exceed 102° F. (38.9° C.), while in the gravest cases hyperpyrexia may occur—105.8°-109° F. (41°-42.8° C.). The temperature finally reaches subnormal ranges and the morning remissions continue in some cases to be subnormal for two or three weeks. It is important to continue thermometric observations for some time after the acute symptoms have ceased.

The pulse is rapid, 120-160; its tension notably increased. In uncomplicated cases the erythrocytes and hæmoglobin are but moderately decreased. Leucocytosis is marked from the onset. In postscarlatinal nephritis profound anæmia rapidly develops. The area of splenic dulness is increased and the lower margin of the organ may be palpable. The area of liver dulness is not enlarged. The urine during the period of invasion and in the early days of the eruption may show a trace of albumin with a few tube-casts—toxic or febrile albuminuria. This disappears with the defervescence. Systematic examination of the urine should be made at intervals of two or three days until desquamation has ceased and convalescence is fully established.

(c) STAGE OF DESQUAMATION.—Desquamation follows the disappearance of the rash. It may begin at once or not until after several days have elapsed. It first shows itself, as a rule, upon the neck or chest; sometimes upon the abdomen above the inguinal folds. It bears some relation to the intensity of the eruption, being in mild cases furfuraceous, in grave cases lamellar or membranous. The thicker layers of the skin upon the hands and feet may come off in extensive patches and children sometimes remove the epidermis of several fingers like the rag of a glove. The average duration of the desquamation is about two weeks, but the process is in many cases repeated and may continue for five or six weeks. The palmar and plantar surfaces are the last to cease shedding. Occasionally the hair falls out.

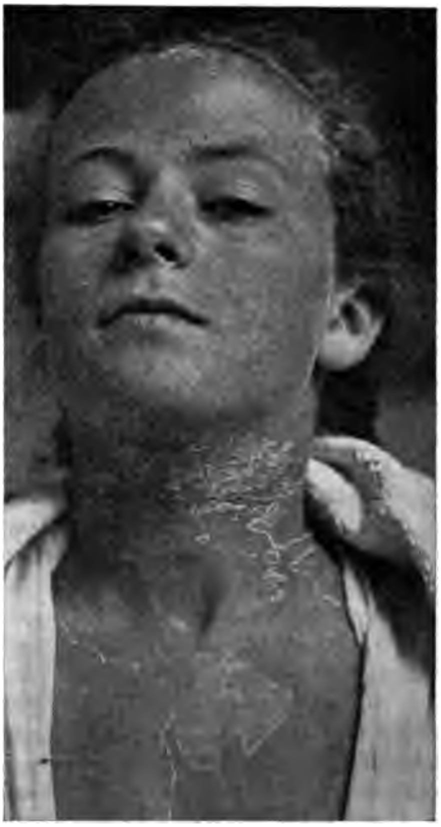


FIG. 230.—Desquamation upon face, neck, and chest after scarlet fever.—After Welch and Schamberg.

Varieties.—The cases differ greatly in severity and duration and may be arranged in the following groups:

1. THE ORDINARY FORM—SCARLATINA SIMPLEX.—This variety has been outlined in the foregoing general description. It presents varying grades of severity, but the symptom-complex is well defined and the acute process in uncomplicated cases comes to an end by the sixth or seventh day.

2. LARVAL OR UNDEVELOPED FORMS.—(a) *Scarlatina Afebrilis.*—The objective phenomena of illness are absent and the patient does not regard himself as sick. Fever is absent or slight—100° F. (37.8° C.), and lasts only a few hours. The rash also is faint, usually limited to the throat and chest, and transient. (b) *Scarlatina sine Eruptione.*—There may be sudden fever with sore throat and vomiting but the skin remains free from eruption. At most there is a transient faint erythematous blush such as is often observed in acute indifferent febrile attacks in young persons of blond complexion. (c) *Scarlatina sine Angina.*—Fever may be present and the eruption may be more or less well defined but the appearance of the throat remains normal throughout the sickness.

These incompletely developed cases often give rise to great difficulties in diagnosis. They occur with some frequency in certain epidemics and are to be recognized by the epidemic tendency, the severe complications which frequently develop, the occurrence of postscarlatinal nephritis, and the fact that they may become centres of infection. The afebrile forms are especially likely to spread the disease. It not infrequently happens that, in a family of children, one may apparently escape the attack and play about as usual, until, as the others recover, desquamation or sudden pallor, dropsy, and albuminuria make it evident that he also has suffered the infection but without the usual symptoms.

3. Malignant Forms—Scarlatina Maligna.—(a) *Scarlatina Siderans.*—These cases occur sporadically and during severe epidemics. The child is overwhelmed at once by the intensity of the infection. Blinding headache, vomiting, convulsions, hyperpyrexia, delirium passing rapidly into coma succeed one another in appalling succession. There is suppression of urine; the heart's action is progressively rapid and feeble and dyspnœa occurs. Death ensues within twenty-four or thirty-six hours. (b) *Scarlatina Hæmorrhagica.*—Petechiæ appear and develop in a brief time into vibices and extensive suggillations. Epistaxis and hæmaturia are common. All the evidences of a profound toxæmia occur and death takes place in the course of two or three days. Enfeebled and badly nourished infants and especially the inmates of public institutions are particularly liable to this form of scarlet fever, but it occasionally occurs in well-nourished children living under favorable circumstances.

4. The Anginose Form—Scarlatina Anginosa.—The throat symptoms early dominate the clinical picture. Intense inflammation of the tonsils and contiguous structures is attended with pain, dysphagia, and mechanical dyspnœa. Pseudomembrane develops and may extend over the soft palate and uvula into the nasopharynx and nasal chambers or downward to the larynx. The Eustachian tube is involved with infection of the middle ear. Local necrosis takes place and the fetor is almost unbearable. The glands of the neck are enlarged. General sepsis frequently results with fatal termination. If life be prolonged the separation of the sloughs may give rise to fatal hemorrhage from the carotid artery.

5. Protracted Form—Scarlatina Typhosa.—Severe and prolonged cases with delirium, profound prostration, sustained high temperature, and all the evidences of grave general infection. Gastro-intestinal disturbances and marked tympany may be present. The fever may continue for two or three weeks and terminate in lysis. This form has been described also as septic or adynamic scarlatina.

6. Surgical and Puerperal Scarlatina.—Some years ago the subject of scarlet fever after surgical operations and in the lying-in woman attracted much attention. Scarlatiniform rashes may occur under two conditions: First, the patient may, at the time of operation or confinement or thereafter, contract scarlet fever and present all the symptoms of the disease; in this case the surgeon or accoucheur must question himself as to his part in conveying the infecting principle; and second, the patient may develop an erythema as the result of sepsis or as a drug exanthem. The angina and peculiar appearance of the tongue are not present. The distribution of the rash is irregular and its course not that of the eruption of scarlet fever. Local erythema may result from carbolic acid or sublimate solutions of undue strength, and general rashes from certain drugs, as copaiba, oil of santol, and quinine. Desquamation may occur as the result of intense erythema due to any cause. These cases have become infrequent under the methods of modern surgery.

Complications and Sequels.—The complications of scarlet fever are numerous. They are mostly due to secondary streptococcus infection, the primary infection rendering the tissues vulnerable and lowering the powers of resistance of the organism. They are usually severe and have much to do with the gravity of the disease. Some epidemics are characterized by the prominence of certain complications. Almost any tissue of the body may become the seat of inflammation and abscess formation during an attack. The following are more commonly involved:

(a) THROAT.—Pseudomembranous and necrotic inflammation of the faucial structures occurs in the anginose form. It is described as scarlatinal diphtheria and may develop during the stage of invasion or upon the appearance of the exanthem. In grave cases there is extensive inflammatory œdema of the neck—"collar of brawn." General septicæmia is apt to supervene and in many epidemics the throat affection is the cause of high mortality. As a rule the throat affection is due to streptococcus infection; much less commonly there is a true complicating diphtheria.

(b) KIDNEYS.—Nephritis constitutes the most common and important complication. Toxic or febrile albuminuria occurring at the height of the fever is usually transient and without greater significance than the same condition in other diseases, as pneumonia. Much more important is the albuminuria which comes on with the fading of the rash or during the stage of desquamation. The anatomical condition in the quickly fatal cases is that of a glomerulonephritis of varying intensity. Clinically the following grades may be recognized:

1. *Mild Catarrhal Nephritis.*—The urine remains normal in amount. It contains a moderate amount of albumin and a few tube-casts. Cylindroids are present. There is no blood. Œdema is slight and is confined to the eyelids and the pretibial areas. The process may last a few weeks and terminate in complete recovery or it may be the starting point of a chronic nephritis. Sudden intensification of the symptoms may occur with anuria and fatal uræmia, œdema, or effusion into the serous sacs or the larynx.

2. *Graver cases* with increased albumin, casts, and blood. The urine is diminished and there is slight or moderate œdema of the face and lower extremities. Effusion into the serous sacs may occur. The symptoms are not urgent, but anæmia is pronounced and the nephritis tends to become chronic. Uræmia may suddenly develop. When apparent recovery takes place renal inadequacy is often established, and the albuminuria of adolescence or the nephritis following an acute illness or exposure to cold years afterwards may be traced to this condition. These are the cases that under well-directed treatment frequently end in recovery.

3. *Very severe cases* with rapidly developing intense anæmia, general anasarca, anuria, or the passage of small amounts of bloody or porter-colored urine which coagulates upon the application of heat and is loaded with blood and casts. Uræmic accidents, vomiting, facial spasm, convulsions, and coma promptly occur, and despite treatment a large proportion of the cases die.

In rare instances œdema may occur without albuminuria. Under these circumstances the dropsy may change its location, coming and going without apparent cause. It may be associated with effusion into the serous sacs, especially ascites, œdema of the glottis or sudden pulmonary œdema. This form of œdema may be the result of anæmia or cachexia.

(c) Joints.—Rarely during the acute attack, more commonly in the second or third week, there may develop inflammation of the joints, synovitis scarlatinosa, so-called scarlatinal rheumatism. The cases may be arranged in two groups: 1. Those in which a serous synovitis of more or less intense character is present, involving most frequently the small joints of the fingers, the wrists and knees, exceptionally the spinal articulations and terminating in complete resolution in the course of a few days. Several joints are usually affected—polyarthritis—but there is little or no rise of temperature and the joint affection lacks the fugacious character so marked in rheumatic fever. This form has been regarded as due to the scarlatinal poison. 2. Suppurative arthritis, usually implicating a single joint and appearing as a local manifestation of a general septic process. Streptococci have been demonstrated in the intra-articular exudate.

(d) Heart.—Benign endocarditis may occur alike when joint complications are present and in their absence. Malignant endocarditis is very rare. Pericarditis is much less frequent than endocarditis. If effusion takes place it may be purulent or, in grave cases, hemorrhagic. Myocarditis is not uncommon. Hypertrophy and dilatation are constant accompaniments of scarlatinal nephritis in children; they are less frequent in adults. It is of importance to perform auscultation of the heart and lungs as a matter of daily routine in every case of scarlet fever. There is no reason why rheumatic fever may not occur as a complication of scarlatina and account for the joint affection and cardiac lesions in certain cases.

(e) Respiratory Organs.—Bronchitis and inhalation pneumonia—bronchopneumonia—are present in cases attended with severe lesions of the upper air-passages. Hypostatic congestion is common in the gravest cases. Croupous pneumonia is rare. Pleurisy is relatively common. It may be plastic; more frequently it is serofibrinous or purulent; rarely hemorrhagic. It usually develops about the middle of the second week.

(f) Auditory Apparatus.—Infection of the middle ear by way of the Eustachian tube is very common. It occurs in almost every case of anginose scarlatina and is attended by serious dangers, both near and remote. In about 85 per cent. of the cases both ears are affected. Suppurative otitis media results with perforation of the tympanic membrane. The inflammation may extend to the labyrinth or to the mastoid cells. Extensive necrosis of parts of the temporal bone may result. Paralysis of the facial nerve is a rare sequel. Meningitis, brain abscess, and sinus thrombosis may occur. In the absence of those accidents spontaneous healing may take place in the course of a few weeks. More commonly the otitis becomes chronic and in many cases, despite treatment, gives rise to permanent impairment or loss of hearing. Burkhardt-Merian found among 4309 cases of acquired deaf-mutism 445, or 10.3 per cent., to be due to scarlet fever.

(g) Lymphatic Glands.—Implication of the lymphatic system occurs in all cases. In mild scarlet fever there is commonly some degree of enlargement of the superficial lymph-nodes, especially in the neck. It is, however, slight and undergoes resolution during convalescence. In more serious cases the glands may be enormously swollen with cellulitis of the surrounding tissues. Phlegmonous inflammation—angina Ludovici—may occur and lead to extensive necrosis with erosion of vessels and fatal hemorrhage. Retropharyngeal abscess is a very rare complication. The enlargement of the cervical glands is occasionally persistent.

(h) Among the RARER COMPLICATIONS and sequels are enterocolitis, local periostitis, noma, perforation of the soft palate, symmetrical gangrene, and various palsies. Protracted anæmia may occur and the growth and development of the child may be greatly retarded. Acute psychoses, mania, or melancholia sometimes develop during convalescence. These conditions are commonly of brief duration, coming to an end in the course of some hours or days. Exceptionally they last several weeks. Hereditary predisposition is present in many of the cases. Boys more frequently suffer than girls. The prognosis is uniformly favorable.

Diagnosis.—The DIRECT DIAGNOSIS rests upon the sudden onset with rapid rise of temperature, vomiting, and nervous disturbances, as convulsions or stupor, the erythematous angina often accompanied with pseudomembranous exudate, the peculiar tongue, the exanthem, the desquamation, and the swelling of the superficial lymph-nodes. The prevalence of an epidemic or a history of exposure is of importance in doubtful cases. There are cases in which for a time the diagnosis must remain uncertain.

DIFFERENTIAL DIAGNOSIS.—1. MEASLES.—Less abrupt onset, catarrhal symptoms, Koplik's sign, longer period of invasion, a distinctly maculopapular rash, coarse, measly, and thick-set, with an irregularly crescentic arrangement, the less intense sore throat, the absence of leucocytosis, and the fine desquamation are important criteria. The rash appears upon the third or fourth day or later, and first upon the face. It is duller in hue than that of scarlet fever. In rare cases of measles in which a uniform, vivid red rash covers the face and body, maculopapules will be found about the wrists or ankles.

2. RÖTHELN.—The eruption may closely resemble that of scarlet fever. The disease is usually slight; fever and constitutional disturbances are insignificant; angina is absent or trifling; the adenitis involves the postauricular glands to a greater extent than the anterior chains; and the prevalence of an epidemic is of diagnostic importance.

3. SEPTICÆMIA.—Not only so-called surgical and puerperal infection but other forms of septicæmia may present rashes almost identical with that of scarlet fever. Certain of these cases show also erythematous sore throat with a tongue thickly furred and red at the edges and tip, and if death does not occur desquamation may take place precisely as in scarlet fever. The differential diagnosis is difficult and may in any given case be impossible.

4. DIPHTHERIA.—False membrane may develop early and present the appearance of true diphtheria. Scarlet fever with intense pseudomembranous angina, diphtheria with an erythematous rash, and the coexistence of scarlet fever and diphtheria are to be considered. The early membranous sore throat of scarlet fever does not usually show the presence of the Bacillus diphtheriæ, which is, however, usually present in the later forms. The erythema of diphtheria is comparatively rare and when present

lacks the uniform distribution and the intense scarlet color of that of scarlet fever. In every suspected case a bacteriological examination must be forthwith made.

5. Other Acute Infections.—In rare instances rashes suggestive of scarlet fever occur in influenza, cerebrospinal fever, variola,—the initial rashes,—varicella, and enteric fever. The differential diagnosis must rest upon the symptom-complex in individual cases. As a rule little real difficulty arises and even that is dispelled in a brief time.

6. Acute Exfoliative Dermatitis. — Sudden onset with fever, scarlatiniform eruption rapidly becoming universal and fading after five or six days with membranous desquamation present a problem in diagnosis difficult of solution. The absence of angina and the tongue of scarlet fever, the occurrence of alopecia, the loss of the nails, a tendency to recur periodically, the occurrence in adults, and the sporadic character of this rare disease in which the case never becomes a focus of contagion usually render the diagnosis a simple matter. Cases first seen during the stage of erythema sometimes present great difficulties in diagnosis.

7. Erythema Simplex.—This dermatosis occurring in young children may suggest scarlet fever. The trifling constitutional disturbance, the transient eruption, the absence of angina, and in most cases absence of desquamation are of diagnostic value.

8. Drug Exanthems.—Local erythematous rashes may follow the application of mustard, solutions of carbolic acid, corrosive sublimate, or other irritants. Circumscribed or diffuse eruptions may follow the internal administration of belladonna, quinine, the iodides, more rarely chloral, sulphonal, antipyrin, turpentine, and the hypodermic injection of tuberculin. These rashes are not accompanied by fever, a fact which may be rendered unavailable in diagnosis by the presence of fever in the condition for which the drug is administered; nor by angina, save in the cases of belladonna, in which dryness and redness of the throat are conspicuous; and certain of them occasion symptoms which are distinctive—tinnitus in the case of quinine, coryza in iodine, and so forth. The difficulty in diagnosis is usually slight.

It may be said that the greater the experience of the practitioner in the acute exanthemata, the greater his hesitancy to make a positive diagnosis in doubtful cases. The only safe rule of practice is to regard every uncertain case as a possible source of infection and treat it accordingly until the diagnosis becomes clear.

Prognosis.—The mortality varies greatly in different epidemics. It is modified by the severity of the infection and the prevalence of grave complications and is lower in private practice than in hospitals. In some outbreaks the disease has been benign, the death-rate not exceeding 5 per cent.; in others, exceedingly severe, with a maximum mortality of 30 or even of 40 per cent. The prognosis in individual cases is influenced by the following conditions: 1. Age. The danger is much greater in infancy and early childhood than later. A large proportion of the deaths occur between the third and sixth years. The absolute mortality among adults is low but my observation leads me to believe that, in proportion to the number of cases, scarlet fever after puberty is very fatal. 2. Sex. It is said that the mortality is higher among males than females. 3. The previous health of the patient. Delicate, poorly nourished, and sickly children bear the disease badly. Like the other acute infections scarlet fever is severe and attended with a high death-rate in orphan asylums and similar institutions.

The danger is great in proportion to the intensity of the primary infection as shown by early high fever, stupor, delirium, restlessness, the evidences of general septic infection, and the prominence of local infective processes, such as membranous or suppurative angina, cervical cellulitis, laryngeal obstruction, bronchitis, bronchopneumonia, pleural effusion, heart complications, and nephritis. Hemorrhagic cases are usually fatal. A persistently high pulse-rate is of itself an unfavorable sign.

The prognosis must in all instances be guarded. Apparently benign cases may develop the gravest symptoms or fatal nephritis may occur during convalescence. No case, however favorable, can be regarded as out of danger until at least four weeks have elapsed from the onset of the attack.

VII. MEASLES.

Rubeola; Morbilli.

Definition.—An acute febrile infection occurring in epidemics, and characterized by initial coryza, bronchial catarrh, and a generalized maculopapular eruption.

Etiology.—PREDISPOSING INFLUENCES.—The susceptibility to measles appears to be almost universal. The disease prevails in every climate and attacks all races. Negroes suffer more severely than whites and are more liable to develop grave complications and sequels. *Season.*—Outbreaks take place at all times of the year but are more common in the winter and spring than in the warmer seasons. *Age* plays an important part. Measles is peculiarly a disease of childhood. Adults not protected by an attack in early life may contract it and frequently manifest the symptoms in an aggravated form. It is more common after puberty than scarlet fever. Congenital cases have been observed; it is rare during the first six months of life. The great majority of cases occur before the tenth year. The *sexes* are alike liable.

EXCITING CAUSE. —The specific pathogenic cause has not been demonstrated. Inoculation experiments upon human beings have shown the presence of the infecting principle in the blood, in the tears, in the secretions of the nasal, pharyngeal, and bronchial mucous membrane, and in the contents of vesicles occasionally present. Inoculation with the epithelial scales thrown off at the close of the disease has been unsuccessful. Transmission of the disease under ordinary circumstances takes place by the breath or the nasal or bronchial secretion. It may result directly from close approach or contact with the patient, or remotely through the conveyance of the poison by a third person or by fomites. Measles is very commonly disseminated in school or upon playgrounds by children who appear to be suffering from ordinary nasal or bronchial catarrh but who are in reality in the pre-eruptive stage. The infecting principle is intensely active but neither tenacious nor persistent as is that of scarlet fever. The disease is communicable during its whole course from the earliest appearance of coryza. The individual predisposition to measles is so general that upon exposure very few escape. The adult who suffers has, as a rule, not been exposed to the infection in childhood and is often an only child who has been educated at home. A congenital immunity seems to exist in rare instances. In the majority of cases an acquired immunity results from the attack. Second, or even third, attacks may occur at intervals of some years, but they are infrequent. I cannot agree with those who regard multiple recurrences as common, since my experience coincides with that of Jürgensen and Eichhorst in Germany, and Holt in this country, who hold that second attacks are rare. Measles in cities is in a certain sense endemic. Sporadic cases occur at intervals and constitute the starting-point of more or less extensive epidemics. When the susceptible individuals in the affected locality have had the disease the epidemic ceases. The poison is frequently carried to neighboring regions by persons in the period of incubation or in the pre-eruptive stage, who become centres of infection for new local epidemics. Extensive outbreaks occur at intervals of five or six years and at long intervals the disease becomes pandemic. Occasionally other epidemic diseases of children, especially whooping-cough and varicella, precede, accompany, or follow outbreaks of measles. Outbreaks are common in time of war among the younger recruits and conscripts, many of whom come from country districts in which the disease has not prevailed for long periods.

Symptoms.—The period of incubation is from seven to fourteen days, usually about ten. In artificially inoculated cases it is commonly less than ten. Prodromes are common. They consist of loss of appetite, restless sleep, fretfulness, and in many cases feverishness or light fever.

(a) STAGE OF INVASION.—The prodromal symptoms are intensified. There is chilliness which may be repeated, sometimes shivering, but convulsions and distinct chills are uncommon. The temperature rises, reaching 102°-104° F. (38.9°-40° C.) upon the first or second day. It then falls a degree or more to rise again upon the appearance of the exanthem. Nausea, vomiting, and headache are present. The tongue is furred. Coincidently with the appearance of these symptoms coryza develops and is often intense. The phenomena are those of an ordinary severe influenza. Irritation and smarting of the eyelids, lachrymation, photophobia, persistent sneezing, running at the nose, sore throat, discomfort in swallowing, hoarseness, and cough, at first of a brassy or croupy character, develop one upon the other in rapid succession and varying intensity. These initial catarrhal symptoms are characteristic and occur in the mildest cases in which chilliness, fever, and the associated signs of the reaction of the organism to general infection are not observed. Upon inspection the vessels of the conjunctivæ are injected, the eyelids swollen, the nasal mucosa tumid and reddened. The mucous membrane of the mouth and throat is erythematous, while upon the soft palate and roof of the mouth, and particularly upon the buccal mucous membrane, are to be seen pin-head or split-pea-sized, circumscribed, round or irregularly shaped reddish blotches slightly or scarcely at all raised above the surrounding surface, usually discrete, sometimes confluent. This eruption also shows itself in the larynx and is doubtless the cause of the laryngobronchial symptoms as it is of the coryza. It has been called the *enanthem* in contradistinction to the rash upon the skin, the *exanthem.* In a strong light there may be seen upon some of the spots upon the mucosa of the cheeks and lips minute bright whitish or bluish-white flecks described by Koplik. These flecks appear early and soon disappear, and since they have not been observed in other diseases are of value in the early diagnosis of measles. The duration of the stage of invasion, or, as it is sometimes called, the catarrhal stage, is commonly three or four days; exceptionally it is shorter or it may be as long as a week.

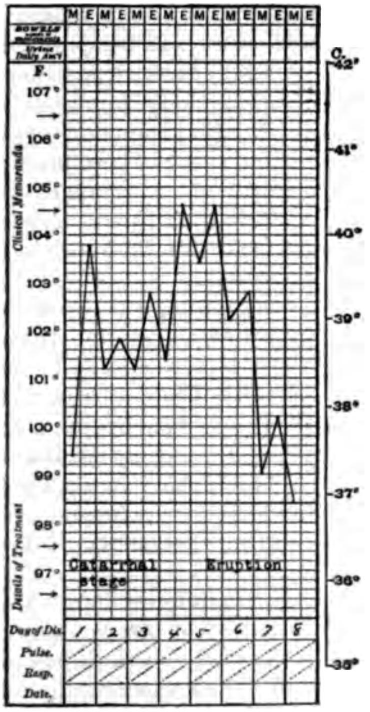


Fig. 231.—Measles.

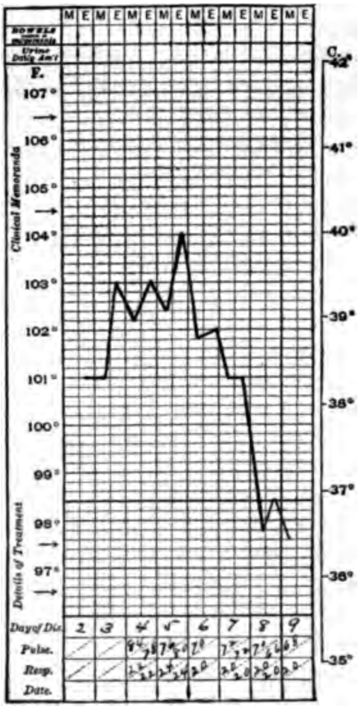


Fig. 232.—Frank uncomplicated measles in a woman aged 52.

(b) Stage of Eruption.—The fever rises and may reach 104°-106° F. (40°-41.1° C.), the pulse-rate may be 140 or higher. Delirium or stupor may be present in the severer cases. The patient complains of heat and burning of the skin, sore throat and general discomfort, and is restless and wakeful. Usually upon the second or third day of the eruption great and rapid amelioration of all these symptoms occurs and the fever which has remained high falls by crisis or by rapid lysis to normal or subnormal ranges. The eruption appears as small red or brownish-red soft flat papules which rapidly increase in size and in numbers. When fully developed the individual spots are irregularly circular or oval and differ greatly in size, the average diameter being that of a split pea. They are unevenly distributed but close set and very often confluent, especially upon the face, buttocks, hands and feet, where there is also some tumefaction. They are circumscribed and the intervening skin is normal or slightly hyperæmic. Not infrequently a crescentic arrangement may be made out elsewhere. The color momentarily fades upon pressure or upon tension of the skin. The eruption appears first upon the forehead, chin, and cheeks; it invades the hairy scalp and spreads rapidly to the neck, back, hands and arms, anterior surface of the trunk and lower extremities, commonly in the order named, invading the entire surface in the course of twenty-four or thirty-six hours. By the end of the second or during the third day the eruption is fully developed. In severe cases punctiform hemorrhages now appear in some of the papules, especially upon portions of the body exposed to pressure. This condition is not significant and must not be confounded with the true hemorrhagic variety of the disease. From this time the eruption rapidly fades, first where it first appeared, namely, upon the face, and in the course of a further period of two or three days disappears, leaving faint yellowish or dirty-brown areas of pigmentation which in turn gradually pass away. In the beginning of the stage of eruption and in many cases throughout its course the skin is moist and often bathed in free perspiration. At the height of the eruption the superficial lymph-nodes of the neck and elsewhere are often slightly swollen and tender.

(c) STAGE OF DESQUAMATION.—Following the fading of the rash desquamation takes place in the form of fine branny scales, so fine that the condition may be easily overlooked. This process begins on the face and involves the entire surface, occupying about a week. The catarrhal symptoms in uncomplicated cases gradually disappear, so that, by the end of the second week from the initial coryza, convalescence is fully established. Cough frequently persists. In the absence of inflammatory complications leucocytosis does not occur. Epistaxis is common at the height of the attack; diarrhœa is apt to occur at some time during its course. Relapses of measles are extremely rare.

During epidemics atypical cases occur. They are not common. Variations in the rash may consist: (1) in the development of distinct papules, hard to the touch but not extending deeply into the skin—*morbilli papulosi;* (2) a vesicular form, *m. vesiculari;* (3) cases in which the eruption does not appear, although the general symptoms and the coryza are present—*m. sine exanthema;* (4) cases in which the mucous membranes are not involved—*m. sine enanthema.* To these must be added variations in the constitutional manifestations. In rare cases there is no rise of temperature—*m. afebriles.* In the malignant forms the organism is unable to withstand the intensity of the infection and death takes place in the course of two or three days after sustained hyperpyrexia, profound adynamia, or hemorrhages into the skin and mucous membranes. The malignant forms are very rare in private practice; they occasionally occur in asylums and in the fierce epidemics of camps, and were common in the first outbreak among the natives of the Fiji Islands, where measles prevailed as a veritable scourge. Death may occur before the rash appears, or a few papules may show themselves upon the forehead and wrists. Hemorrhagic or black measles—*m. hæmorrhagica*—is characterized by convulsions, delirium and coma, petechiæ, bleeding from mucous surfaces, and profound constitutional depression.

Complications and Sequels.—Epidemics differ greatly as regards the frequency and severity of complications. In their absence measles is a comparatively benign malady, but they are sufficiently common to place it among the more serious diseases of childhood. Debilitated and badly nourished children living in unhygienic surroundings and those in asylums and institutions are especially liable. The ordinary complications are due to an extension or intensification of the catarrhal processes peculiar to the disease.

Otitis media is not very uncommon. It may result in perforation of the tympanic membrane and permanent impairment of hearing, or lead to sinus thrombosis, meningitis, or abscess of the brain. Purulent conjunctivitis may occur and in neglected cases infiltration and ulceration of the cornea. Catarrhal laryngitis is of frequent occurrence; the pseudomembranous form is rare and very dangerous; œdema of the glottis is very uncommon. Diphtheria is much less frequent in measles than in scarlet fever.

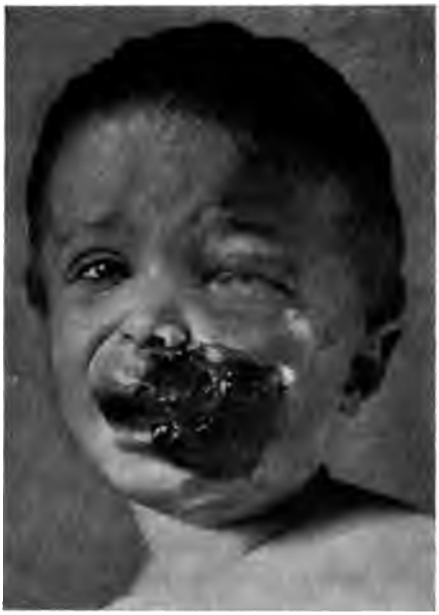


Fig. 233.—Cancrum oris complicating measles.—After Welch and Schamberg.

The catarrhal bronchitis so prominent in the disease is, in itself, without serious significance, and in favorable cases terminates in resolution with the convalescence. Its tendency to extend to the finer tubes and give rise to lobular collapse and bronchopneumonia constitutes the gravest danger. If the involvement of the vesicular structure is limited there is increase of fever with acceleration of the pulse, harassing cough, and disturbance of respiration. The sickness is prolonged but terminates favorably. When the lesions are extensive the symptoms become urgent and a large proportion of the cases die. It is to this complication that the high death-rate of measles under unfavorable circumstances is due. Croupous pneumonia is much less common. Pleural effusion is rare. Acute enterocolitis is a frequent and serious complication in some epidemics. Toxic albuminuria occasionally occurs as in other febrile infections, and the diazo reaction is present. There is little tendency to nephritis. Arthritis is very rare. In young and debilitated children gangrenous stomatitis and in girls gangrene of the pudenda occur during convalescence with greater frequency than in any other infectious disease.

Among the common sequels are chronic local inflammations, conjunctivitis, otitis, nasal catarrh, laryngitis, and bronchitis. The intestinal catarrh arising as a complication may lead to chronic colitis. Tuberculosis is a very common sequel. The lesions of measles are such as to render the patient peculiarly liable to this infection, but the rapidity with which tuberculous bronchopneumonia and acute miliary tuberculosis develop in many instances renders it probable that a latent tuberculous process has been roused into activity during the attack. Pulmonary tuberculosis is a common remote sequel of measles in the adult, and miliary tuberculosis and tuberculous meningitis may frequently be traced to measles in earlier life. In some cases enlarged caseating bronchial glands may be the starting-point of the general infection. Among the rarer sequels are tuberculosis of the cervical lymph-nodes and of the bones and joints. Palsies occur as the result of peripheral neuritis but are much more rare than in diphtheria or scarlet fever.

Diagnosis.—The DIRECT DIAGNOSIS of well-developed measles after the appearance of the eruption is generally unattended with difficulty. During an epidemic coryza persistent sneezing and fever are suspicious. The appearance of the eruption on the second or third day upon the mucous membrane of the mouth and throat, and Koplik's sign are of positive diagnostic value.

The DIFFERENTIAL DIAGNOSIS concerns: 1. RÖTHELN (see p. 689). 2. VARIOLA (see p. 661). 3. TYPHUS FEVER (see p. 644). 4. SCARLET FEVER (see p. 679).

5. SYPHILITIC ROSEOLA usually occurs in the adult. The eruption if carefully examined is seen to be polymorphous, the enlargement of the superficial lymphatic glands is greater, and the signs of syphilis are to be found in the mouth and throat and upon the genitalia.

6. DRUG EXANTHEMS.—Exceptionally the administration of salicylates, antipyrin, quinine, turpentine, or copaiba is followed by a rash suggesting rather than resembling that of measles. These rashes are not accompanied by fever or throat symptoms, nor have they the uniform appearance and distribution of the measles exanthem (see p. 683).

In the negro the difficulties in doubtful cases are increased; but the mode of onset, the coryza and bronchitis, and the peculiarities of the rash upon the mucosa of the mouth are of diagnostic importance. The soft flat papules may be distinguished in the darkest skin.

The diagnosis in certain cases must for a time remain doubtful, especially when the disease appears sporadically or prevails during epidemics of rötheln, scarlatina, variola, or typhus.

Prognosis.—The character of the prevailing epidemic and previous condition of the individual greatly modify the prognosis. The death-rate during the first six months of life is relatively low; it reaches its maximum during the second year and rapidly falls after the fifth year. After the twentieth year it rises again. In private practice the mortality is about 3 per cent., in some epidemics not more than 1.8 per cent.; in hospital and asylum practice and in camps and barracks it may reach 30 per cent. The vital statistics of measles are misleading, because the people regard the disease as an insignificant malady and among the lower classes only the more serious cases come under medical observation. Uncomplicated measles is, in point of fact, a benign infection, but the tendency to pulmonary complications renders it one of the gravest diseases of childhood. It is estimated that about one-third of the cases in which bronchopneumonia develops terminate fatally.

VIII. RUBELLA.

Rötheln; German Measles; Epidemic Roseola.

Definition.—An acute epidemic infectious disease characterized by a diffuse maculopapular eruption and swelling of the superficial lymphatic glands.

Rubella has some points of resemblance to scarlet fever and to measles and was at one time regarded as a hybrid of the two. It is now known to be an independent substantive affection.

Etiology.—The infecting principle has not yet been discovered. The disease is readily transmissible and usually prevails in extensive epidemics. Outbreaks occur in series, followed by long intervals during which the disease does not recur. In the absence of an acquired immunity persons at any age are susceptible. Rubella does not protect against the infection of scarlet fever or of measles, nor do these diseases protect those who have passed through them against rubella.

Symptoms.—The period of incubation varies from ten to twenty days. Prodromes are usually absent.

STAGE OF INVASION.—The symptoms are generally mild. They consist of the ordinary manifestations of the reaction of the organism to an infection of little intensity, and the coryza, laryngitis, and pharyngitis which usually precede the exanthemata. The duration of this stage is not constant, varying from a few hours to two or three days. The symptoms may be so slight as to be altogether overlooked and the rash may then be the first indication of illness. The elevation of temperature is trifling, usually about 100° F. (37.8° C.), and rarely exceeding 102° F. (38.9° C.), and transient. In asylums and foundling institutions rubella sometimes prevails as a serious malady.

STAGE OF ERUPTION.—The rash commonly appears upon the first day; it may be as late as the third. It shows itself first upon the face and neck, and spreads in the course of twenty-four hours over the body and extremities. It consists of round or oval reddish spots about the diameter of a split pea, mostly discrete, sometimes confluent, and surrounded by areas of hyperæmic skin. In some of the cases extensive tracts of the skin are intensely hyperæmic so that the rash resembles that of scarlet fever rather than measles. The crescentic arrangement of the papules, seen in measles, cannot usually be made out. The eruption frequently fades irregularly in patches some hours after it comes out, so that certain areas of the surface are covered and not the entire body at the same time. In the course of two or three days the rash gradually disappears with fine furfuraceous desquamation, leaving a faint pigmentation which persists for a short time. Slight itching commonly accompanies the rash. The superficial lymphatic glands, especially those of the neck, are slightly enlarged. They undergo resolution in a short time after the fading of the eruption. The suboccipital and lateral chains are commonly involved to a greater extent than the anterolateral. The enlargement of the lymph-nodes in some instances precedes the appearance of the eruption.

Relapses are rare; complications infrequent. Albuminuria, bronchitis, colitis, and pneumonia have been noted. Herpes may occur. There are no special sequels. The immunity acquired by the attack is not always permanent. Second and third attacks may occur.

Diagnosis.—The early cases may present great difficulty in diagnosis. When an epidemic is prevalent the recognition of rubella is easy. The DIRECT DIAGNOSIS rests upon the trifling nature of the disease, the short initial period, the character of the eruption, its patchy distribution, the early enlargement of the glands, and the absence of severe throat symptoms and coryza.

DIFFERENTIAL DIAGNOSIS OF SCARLET FEVER, MEASLES, AND RUBELLA.

	Scarlet Fever.	Measles.	Rubella.
Contagion	Highly contagious	Highly contagious	Variable in epidemics.
Transmissibility	By direct contact, approach, and fomites	By direct contact, fomites, and through the air	Direct contact and fomites; not through the air.
Period of Incubation	Average 2 to 7 days	Average 9 to 14 days	Variable; average 1 to 3 weeks.
Prodromes	Absent or very brief—onset commonly sudden	Commonly 3 to 4 days. Drowsiness and catarrhal symptoms	Slight and of short duration.
Koplik Spots	Not present	Present in about 90 per cent. of cases	Not present.
Vomiting	Common at onset	Infrequent	Rare.
Temperature	High- 103°–105° F.—lasting about a week	High, lasting about a week, average 102°–104° F.	Slight elevation, seldom more than 101°–102° F.—subsides in 1 to 3 days.
Catarrhal Symptoms	Commonly absent	Prominent throughout	Slight.
Tongue	Whitish fur, enlarged papillæ; later dry and red	Tongue coated	Slightly coated, not characteristic.
Throat	More or less intense erythematous angina	Moderate redness of mucous membranes	Punctiform red spots over uvula, palate, and pharynx.
Lymph-nodes	Glands at angle of the jaw enlarged	Cervical, postauricular and submaxillary nodes enlarged	General enlargement, especially of postcervical chains.
Pulse	High frequency—120–140	Corresponding to elevations in temperature	Varies with fever.
Urine	Early toxic albuminuria in severe cases. Later signs of nephritis	Albuminuria rare	Albuminuria very rare and slight.
Eruption	First appears on neck and chest, spreads slowly over entire body. Fully developed about the fourth day. Small punctate efflorescence or diffuse blush disappearing on pressure. Lasts about a week. Intense scarlet color; usually absent around mouth	First appears on face, spreads gradually over entire body. Fully developed by the second or third day. Consists of small papules arranged in crescentic groups; these are confluent in places; fades in 4 or 5 days; deep red, dusky or purplish	First appears on face, spreading to neck and breast, then to arms, legs and feet. Fades in parts first involved while spreading to others. Two varieties — morbilliform, small, slightly elevated papules, discrete, sometimes confluent; scarlatiniform. Duration 2 to 4 days or less. Color rose red but variable
Desquamation	Coarse, bran-like; lamellar	Branny	Fine, branny.
Convalescence	Tardy; complications frequent, especially nephritis, otitis media, etc.	Slow; tendency to complications as bronchopneumonia and other chest diseases, especially tuberculosis	Rapid without complications.

Differential Diagnosis.—Rubella is most frequently mistaken for mild measles or scarlatina. From measles it is distinguished by the want of prominence of catarrhal phenomena, the slighter fever, the brighter hue of the eruption, the absence of the crescentic grouping of the papules, the fact that the adenitis involves to a greater degree the suboccipital and postauricular glands, and the absence of Koplik's sign; from scarlatina by its gradual onset, benign character, the absence of severe throat symptoms, the peculiarities of the rash, the character of the desquamation, the tongue, and the fact that there is no special tendency to nephritis.

Prognosis.—Rubella is a benign disease almost invariably terminating in recovery. In foundling hospitals and asylums it has sometimes assumed unusual severity, and fatal cases have occurred commonly as the result of an intercurrent pneumonia, colitis, or nephritis, rather than of the primary disease (see table on opposite page).

THE FOURTH DISEASE.

In 1900 Dukes described an infectious disease which he called "the fourth disease." This communication attracted considerable attention and the subject has been discussed by a number of clinicians. The incubation period is stated to be about the same as that of German measles, ten to twenty-one days. Prodromes were absent in most of the cases but malaise and a mild erythematous angina were occasionally observed at the time of the appearance of the rash. The evolution of the exanthem was rapid, the entire body being covered in the course of a few hours. Whether or not it was present upon the face is not stated. Its color was like that of scarlet fever but brighter. The superficial lymph-nodes were enlarged. The temperature was subfebrile, not often exceeding 101° F. (38.5° C.). Upon the subsidence of the eruption there was desquamation. Sequels were not observed and the attack did not confer immunity against scarlet fever or rubella. This affection has not been generally recognized as a clinical entity.

IX. WHOOPING-COUGH.

Pertussis; Tussis Convulsiva.

Definition.—An infectious endemic and epidemic disease characterized by hyperæsthesia and catarrh of the respiratory tract and a peculiar, spasmodic cough occurring in paroxysms which terminate in a prolonged inspiration attended by a shrill crowing sound or whoop.

Etiology.—Predisposing Influences.—Whooping-cough is a widely prevalent disease. The individual susceptibility, like that to measles, is almost universal. Very few persons unless rendered immune by a previous attack escape upon exposure. Nearly twice as many cases occur during the winter and spring as during the summer and autumn. It is peculiarly a disease of infancy and early childhood. More than one-half the cases occur during the first two years of life; very few cases after the second dentition. That the immunity after the seventh year is acquired rather than congenital is shown by the fact that in individuals not protected by a previous attack the disease may be contracted upon exposure at any period of life. Sucklings are not exempt. Sex is without influence in early life, but among adults women are more liable than men, a fact to be explained in part by increased exposure, in part by the more common neurotic constitution in women. Pregnancy appears to be attended with an especial liability. The previous condition of health is of great importance. Delicate children and those suffering from nasal or bronchial catarrh are especially liable to contract the disease upon slight exposure.

EXCITING CAUSE.—The specific infecting agent has not yet been demonstrated. Afanassiew, 1887, discovered in the secretions a short bacillus, cultures of which injected into the respiratory passages in animals produce catarrhal inflammation. Koplik has more recently found a bacillus resembling that of influenza but larger, which he regards as the cause of the disease. Spengler and others have described an organism to which the name Bacillus pertussis has been given.

Clinical experience makes it clear that the infective material is eliminated by way of the mucous discharges and perhaps by the expired air. It reaches the organism with the inspired air. Actual contact, close approach, or fomites, especially such articles as the handkerchief or towel. constitute the usual means of transmission from the sick to the well. Under certain circumstances a third person may readily transmit the infective material. The dried sputum circulating as dust in the atmosphere is probably also a source of infection. The disease is transmissible from the earliest appearance of the catarrhal symptoms, and since it cannot be recognized until the spasmodic stage, and since in suitable weather the patients are kept as much as possible in the open air, the patient alike in the nursery and school and out of doors is in constant danger of disseminating it. The attack confers an immunity which in most cases is lifelong. The occasional occurrence of cases in elderly persons, who have passed through an attack in childhood, living in the house with children suffering from the disease, shows, however, that the protection is not always permanent.

Whooping-cough is endemic in large cities when it takes the form of extended epidemics at irregular intervals of from two to four years. Its prevalence is sometimes so wide-spread as to merit the descriptive term pandemic. Outbreaks are not infrequently associated with epidemics of measles, scarlet fever, or varicella, and these diseases occasionally run their course coincidently with whooping-cough in the same individual.

Symptoms.—The period of incubation varies from seven to ten days. If, after exposure, two full weeks elapse without the development of catarrhal symptoms, the probability becomes very strong that infection has not taken place.

THE COURSE OF THE ATTACK.—(a) The *catarrhal stage* begins with the symptoms of an ordinary subacute bronchitis, which gradually increase in intensity. In the course of some days the cough tends to become paroxysmal, the spells being more frequent and severe during the night and after meals. Running at the nose, hoarseness, and a croupy ringing cough, the indications of a nasal and laryngeal catarrh of moderate severity, are associated symptoms. There is very often fever of moderate grade. The duration of this stage is about a week or ten days. Cases vary greatly in this respect, however, some children developing the whoop in the course of a day or two from the beginning of the catarrhal symptoms, others not until three or four weeks have elapsed.

(b) *Spasmodic or Paroxysmal Stage.*—The fever subsides. The catarrhal symptoms continue and may be intensified. The cough becomes distinctly paroxysmal and characteristic, the attacks ending in a long-drawn "whoop" from which the disease receives its name. The true nature of the disease is now first apparent. The patient experiences a sensation of tickling in the larynx or under the sternum. Little children run terrified to the nurse or mother and cling to her; older persons grasp some object, as the arms of a chair, for support. The fully developed paroxysm usually begins with a long-drawn inspiration which is immediately followed by a series of ten or fifteen short explosive coughs of increasing intensity and repeated so rapidly that breathing is ineffectual until at length a prolonged deep inspiration occurs, during which the whoop is produced. One or more new series of coughs terminating in the whoop may forthwith follow and the paroxysms may not come to an end until a mass of tough stringy mucus is raised. This is usually small but in little children it is often very abundant and must be removed from the mouth by the finger. It may be expelled in the act of vomiting. An abundance of thick mucus is at the same time discharged from the nose. The signs of mechanical disturbance of the venous circulation are conspicuous. The face and neck become congested and cyanotic, the veins of the face and the jugulars stand out prominently, there is protrusion of the eyeballs, sometimes marked injection of the conjunctivæ, and the lips are swollen and blue. As the attack comes to an end the face or the whole body may break out into a more or less profuse sweat. In severe paroxysms the sphincters may be relaxed. Headache and vertigo are common, and at the close of a severe attack the child sinks exhausted upon the mother's lap. The condition is most distressing, but in a little while the child usually recovers himself and goes about his play until another spell occurs. The single paroxysm lasts from fifteen to forty-five seconds, rarely longer; when two or more immediately succeed each other, the whole attack may be prolonged to two or three minutes. They are fortunately not all of the same intensity, frequent milder attacks occurring between those which are more severe. They may be brought on by taking food or drink, especially anything cold, by laughter or vexation, and in some cases by traction of or pressure upon the tongue. They are more frequent in a close room than in the open air and by night than during the day. The number of paroxysms varies from three or four to sixty to one hundred during twenty-four hours. Severe paroxysms after the taking of food almost always cause vomiting and the patients very often become much emaciated and reduced in strength. In many cases, however, as soon as the distressing symptoms are over the child will eat another meal, which should always, under these circumstances, be offered to it. The violence of the cough forces the tongue against the lower incisor teeth and very often causes laceration of the mucous membrane of the frænum, which is followed by superficial ulceration. A marked leucocytosis occurs. In very mild cases the paroxysms are not only less frequent but they are also less violent, and occasional cases occur in which the whoop is absent throughout the attack, the nature of which is apparent from the presence of other cases in the house, the spasmodic spells of coughing attended with vomiting and terminating with the expulsion of a mass of tenacious mucus, and the protracted course of the illness.

The cough is the result of an extended irritation involving the upper air-passages. It is probable that the gradual accumulation of mucus in the region of the bifurcation of the trachea plays an important rôle in its production. The mechanism of the whoop consists in the forcible indrawing of air through the spasmodically narrowed glottis. The disease has the characteristics of a neurosis affecting the respiratory tract.

Laryngoscopic examination frequently shows the mucous membrane of the larynx to be congested and swollen, especially in the interarytenoid space, and sometimes the seat of hemorrhagic patches or superficial erosions. Irritation of the mucous membrane between the arytenoids or of the posterior surface of the epiglottis with a sound, always causes the paroxysm. A similar condition of congestion and swelling has been observed in the trachea, in which a plug of mucus has been seen just before the paroxysm—Roosbach. The difficulty of such examinations is obvious.

Physical examination yields unimportant signs. The resonance is impaired during the paroxysm and increased at its close. Auscultation yields commonly an enfeebled vesicular murmur and a few bronchial râles, usually dry—sonorous or sibilant.

The average duration of the spasmodic stage is about one month. The symptoms progressively increase in intensity for a fortnight or longer, remain stationary for a time, and gradually subside. In mild cases this stage may not exceed a week or ten days, or the whoop may be wholly absent; in severe cases it may be prolonged, especially if the patient must be housed, as in the winter, for three or four months, with remissions and exacerbations.

(c) *Stage of Decline.*—The paroxysms diminish in severity and frequency; the expectoration becomes more abundant and less tenacious; and finally, as at the beginning, the symptoms are those of an ordinary catarrhal bronchitis, which varies in intensity and continues two or three weeks in favorable and much longer in unfavorable cases, especially during the winter months when convalescents must be kept housed.

The duration of the attack varies between two and four months. A majority of the cases in older children can scarcely be regarded as ill. They are out of bed and eat well, and in proper weather can pass some hours in the open air. They, however, lose flesh and become pale.

A cough habit is often developed during the attack, and for several months after full convalescence has been established, with every cold or nasobronchial catarrh, however trifling, a paroxysmal cough with whooping returns, and this is particularly the case with the children of neurotic parents. Under these circumstances the disease is not communicated. Relapses practically do not occur. Second attacks are by no means uncommon, but as has already been mentioned they usually occur after the lapse of years, and children and their parents or grandparents often suffer at the same time.

Complications and sequels are numerous and may be arranged in two categories, the mechanical and the infectious.

(a) The *mechanical complications and sequels* are caused by increased respiratory pressure during the paroxysm, or circulatory disturbances. Acute emphysema is common. It is as a rule transient. If it persists pseudohypertrophic emphysema results. Rupture of the tissue of the lung may give rise to interstitial emphysema, or the air may find its way along the peribronchial connective tissue to the anterior mediastinum or upwards and give rise to subcutaneous emphysema of the neck. Pneumothorax is less common. Dilatation of the right heart may occur in consequence of the interference with the pulmonary circulation during the paroxysms. It is possible that valvular disease may, in some instances, be due to the heart strain of severe whooping-cough. The pulse after the paroxysm is often feeble and irregular, and progressively so as the attack goes on. The vomiting is largely due to mechanical disturbance caused by cough. Sometimes the patient vomits freely during several paroxysms daily for periods of weeks, and as a result is greatly reduced in flesh and strength. Partly as the result of the violent succussion and partly from exhaustion, involuntary discharges of gas or fecal matter are of common occurrence in severe paroxysms. Prolapse of the bowel and hernia are common and must be ascribed to the same causes. Involuntary discharge of urine likewise occurs. Pregnant women frequently abort. Very common are lesions of the blood-vessels during the paroxysms, resulting in hemorrhages into the skin, particularly about the forehead and eyes, and into the mucous membranes, especially subconjunctival ecchymoses. Much less common are slight hemorrhages—not more than a few drops—from the ear in consequence of superficial lacerations of the tympanitic membrane. Epistaxis is very frequent, hæmoptysis rare. Hemorrhage from the bowel is very unusual, and when it occurs is due to the mechanical derangements which cause prolapse. Convulsions are not uncommon, especially in very young children, and have been ascribed to the engorgement of the cerebral vessels. Meningeal and cerebral hemorrhages occur, but these accidents are extremely infrequent. Hemiplegia and aphasia may result. Sudden death has occurred.

(b) The *infectious complications* include inflammatory enlargement of the bronchial glands, sufficient in some instances to give rise to dulness over the manubrium; bronchopneumonia, which is very common and the cause of death in the majority of the fatal cases; tuberculosis, which may take the form of a tuberculous bronchopneumonia, miliary tuberculosis, or an acute caseous consumption; croupous pneumonia, which is infrequent; pleurisy, still more rare; and nephritis, likewise very uncommon. Other inflammatory complications are seldom encountered. Transient albuminuria is not infrequent and glycosuria is occasionally observed. Many of the complications are essentially chronic and persist as sequels. The patients not infrequently show an especial predisposition to recurrences of bronchial catarrh. Emphysema and asthma are common sequels.

Diagnosis.—The DIRECT DIAGNOSIS of whooping-cough during the early part of the catarrhal stage is impracticable. In the course of a week the increasing severity of the symptoms and the tendency of the cough to become paroxysmal and worse at night, to cause vomiting, suffusion of the eyes, and flushing of the face, render the diagnosis during an epidemic, or with a history of exposure, a probable one. A like uncertainty arises in regard to very mild cases. A child may cough for several weeks without having a well-developed paroxysm. If there be no fever, only a few râles now and then upon auscultation, and ordinary treatment be without effect, the diagnosis by exclusion may be made. The occurrence of the whoop renders the diagnosis easy and certain. It is to be remembered that pressure upon or traction of the tongue, the act of swallowing, and emotional disturbances may cause a paroxysm—facts which the physician may use for diagnostic purposes. The diagnosis may also be difficult in early infancy, when the cough attending ordinary bronchitis sometimes assumes a paroxysmal character and is attended with a croupy or crowing sound that is suggestive of the whoop. The ulcer upon the frænum and subconjunctival or other hemorrhages are not apt to occur in mild cases and these only present diagnostic uncertainties.

Prognosis.—Uncomplicated whooping-cough tends to run a favorable course. The great tendency to complications places it, however, among the most serious of the diseases of childhood. It has been estimated that fully two-thirds of the deaths from this disease occur within the first year. After the fourth year the danger rapidly diminishes. Bronchopneumonia and enterocolitis are the most common causes of death. Convulsions very often occur in fatal cases in early infancy. Delicate and badly nourished children, those living under improper hygienic conditions, those who have rickets or who have been debilitated by a recent attack of measles, influenza, or other serious infection are apt to suffer severely. The aged bear whooping-cough badly. It is peculiarly fatal among negroes. The danger of early or remote tuberculosis lends especial importance to this disease. Death may occur during a paroxysm from intracranial hemorrhage or asphyxia, but such accidents are exceedingly uncommon. The prognosis is to some extent modified by the frequency as well as by the severity of the paroxysms. Cases run a more favorable course in summer than in winter. Reliable general statistical facts relating to the mortality are not available. Many of the milder cases never come under medical observation. In foundling asylums and children's hospitals the death-rate may exceed twenty-five per cent.

X. MUMPS.

Epidemic Parotitis.

Definition.—An acute infectious disease, prevailing in limited epidemics, and characterized by inflammation and enlargement of the salivary glands, especially the parotid.

Etiology. — PREDISPOSING INFLUENCES. — Mumps is a wide-spread disease and is usually endemic in large cities. Sporadic cases occur and become foci of circumscribed outbreaks which run a lingering course of months or, in some instances, of a year or more. The infecting principle is much less readily transmitted than that of many of the contagious diseases and the congenital immunity much more common. In general practice extensive epidemics are infrequent, but when the disease appears in reformatory institutions and schools a large proportion of the inmates usually contract it. The cases are more numerous in the spring and autumn than at other seasons. Mumps is peculiarly a disease of childhood and adolescence. It is not common in early infancy nor after the twentieth year. More boys than girls suffer in a ratio estimated as high as two to one.

EXCITING CAUSE.—The specific cause has not been demonstrated. The disease is directly transmitted by personal contact. Rare instances have been observed in which the contagion has been indirectly transmitted by a third person or by fomites, especially clothing. Two views may be entertained as to the mode of infection. The first is the one generally accepted, namely, that the pathogenic principle finds its way from the mouth to the glands along the course of the salivary ducts and, as the parotid is usually involved, through the duct of Stenson; second, that the infection is a general one, to which certain anatomical structures, as the salivary glands and, in particular, the parotid gland, especially react. The occasional occurrence of inflammation of the testes, and of the ovaries and mammæ in the female, and the definite incubation and typical course of the disease lend support to this view.

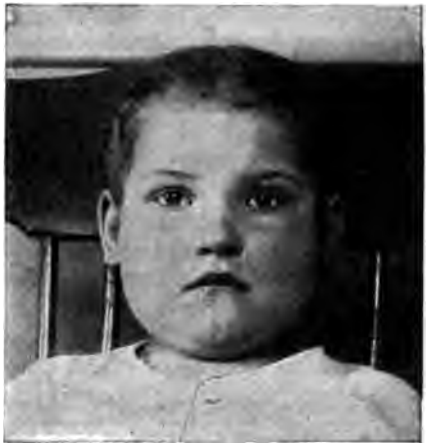


FIG. 234.—Mumps.—Cotton.

Symptoms.—The period of incubation varies from fourteen to twenty-one days. In rare instances it has appeared to be shorter. Prodromes are commonly absent. In mild cases the swelling and associated local symptoms constitute the earliest manifestations. In severer cases more or less pronounced constitutional disturbance, with shivering, vomiting, and moderate fever, 100°–101° F. (37.8°–38.3° C.), characterize the invasion, which is abrupt and precedes the local inflammation by about twenty-four hours. In severe cases the temperature may reach 103°–104° F. (39.5°–40° C.). A feeling of tension with soreness is felt just below one ear, more commonly the left. Upon examination slight swelling may be observed, which increases until, in the course of forty-eight hours, it reaches its maximum. The parotid is now greatly enlarged and the adjacent tissues of the neck and often of the side of the face tensely œdematous. The skin is glossy, hard to the touch, its folds are obliterated, and, commonly, by reason of interference with the circulation by pressure, white in color. It pits only slightly upon pressure. The swelling occupies the lateral region of the neck between the jaw and the mastoid process, extending upward to the zygoma and downward and forward toward the clavicle and the median line. Its extent varies with the intensity of the attack. The ear is pushed upward, and its lobule, which occupies the centre of the swelling, is sharply pushed outward. In almost all instances the other side is affected in a day or two, sometimes not for several days or until the inflammation upon the side first affected has subsided. Very often the swelling of the second gland is so slight that it can only be detected upon close scrutiny. The disfigurement is marked and when both sides are affected the patient may be scarcely recognizable. In some instances the other salivary glands are involved, and several cases have come under my observation in house epidemics in which the submaxillary glands have been inflamed while the parotids have remained unaffected. The sublingual glands and the lachrymal glands may also be involved. Movements of the jaw, the act of deglutition and, in severe cases, even phonation are attended with difficulty and pain. There is trouble in taking any form of nourishment, even liquids. The fetor is often extreme. Movements of the head are restricted and in order to relieve tension there is flexion of the neck toward the affected side. The salivary secretion is usually diminished, exceptionally increased. Its reaction may be acid. Slight deafness and earache often occur, and in rare instances otitis media. Permanent deafness, usually one-sided and complete, without otitis media, has been observed. This condition develops suddenly during the course of the disease or in convalescence, with nausea and vomiting, vertigo and a staggering gait. It is probably due to a lesion of the labyrinth. After from five to ten days the swelling gradually subsides, the stiffness and impairment of movement disappear, and normal conditions are restored. Local desquamation may occur. The glandular inflammation undergoes resolution without abscess formation. If suppuration occurs it is due to a mixed infection and must be regarded as a complication. The pus may be evacuated externally or may burrow in the tissues of the neck. Secondary pyothorax or pyopericardium may follow with or without general sepsis.

Orchitis occurs in about one-third of the cases after puberty. In infancy and childhood it is exceedingly rare. It usually affects one testicle only, occasionally both. Weight, swelling, and pain are the symptoms. The testicle may be enormously enlarged. Epididymitis is not common. There may be effusion into the tunica vaginalis, scrotal œdema, and a mild mucopurulent urethral discharge. Atrophy may result and, when both testicles are involved, loss of the procreative function. Even with some atrophy of both testicles functional power may be retained. In adolescents and young adults great anxiety upon this question arises during the attack. Mastitis may occur in boys. In females also, usually after the age of puberty, enlargement and tenderness of the breasts, pain and tenderness in the ovaries, hæmatoma of the labia, or a vulvovaginal discharge may occur. These phenomena are very uncommon. Thyroid enlargement is extremely rare.

Exceptionally the symptoms are very severe. High fever may be accompanied by vomiting, delirium, and sleeplessness. Great exhaustion may result. As a rule the patient is not seriously ill. The constitutional disturbance comes to an end within a week; the local symptoms more slowly.

Relapse is extremely rare. The attack confers an immunity which is practically permanent.

Complications and Sequels.—The frequent involvement of the generative organs and the fact that it sometimes precedes the parotitis or occurs without it compel the recognition of these local inflammations as incidental manifestations of the disease rather than complications. The frequency of actual complications varies in different epidemics. The fatal cases are frequently associated with meningeal symptoms. Hemiplegia, coma, and acute mania may occur. Among the rare complications are albuminuria, nephritis with uræmic accidents, polyarthritis, endocarditis, facial palsy from pressure, peripheral neuritis, and hemiplegia. The common sequels have been already indicated. The inflamed glands may not undergo resolution but remain enlarged and hard. Ptyalism or xerostomia may persist for some time. Parotid bubo is very rare in idiopathic mumps. Local gangrene may occur. Deafness may be permanent. Optic atrophy is among the rarest of sequels.

Diagnosis.—The DIRECT DIAGNOSIS of mumps is under ordinary circumstances easy. The location of the swelling in front of the ear and below it, and the abrupt displacement of the lobule outwards, together with the circumscribed outline at first corresponding to that of the parotid, are important anatomical considerations. Mumps is a primary affection and very rarely goes on to suppuration. The relative rapidity with which the swelling develops and subsides is characteristic of mumps.

DIFFERENTIAL DIAGNOSIS.—1. Parotid bubo. The glandular inflammation is secondary to some acute infection or sepsis and commonly proceeds to multiple or general abscess formation. This condition is very rare in childhood. 2. Acute cervical adenitis. The swelling is below the angle of the jaw. It does not at any time correspond to the outline of the parotid; nor has it the location of the submaxillary glands. It may be tuberculous or secondary to tonsillar or peritonsillar infection. Irregular contour, redness, the absence of involvement of the generative glands in all cases, and the absence of a definite, self-limited course are to be considered. 3. Hodgkin's disease is a chronic affection of the lymphatic glands. The salivary glands are not involved. 4. Abscess from disease of the jaw in dental caries gives rise to swelling localized in wholly different positions from that of the parotid and is not characterized by definite constitutional phenomena or transmissibility. 5. Gonorrhœal orchitis has a definite history and upon examination of the discharge the gonococcus is found. Doubts can only arise when the inflammation of the testes precedes or occurs coincidently with the parotitis.

Prognosis.—Mumps, in the vast majority of cases, is a mild disease and terminates in complete recovery in a short time.

XI. INFLUENZA.

Epidemic Catarrhal Fever; La Grippe.

Definition.—An acute, infectious, pandemic disease, caused by the bacillus of Pfeiffer. It is characterized by catarrh of the mucous membrane of the respiratory tract, less frequently of the digestive tract, by quickly on-coming debility and nervous symptoms. There is a tendency to complications, especially pneumonia. The general outbreaks are followed for some years by the local epidemic or endemic occurrence of the disease.

Etiology.—PREDISPOSING INFLUENCES.—When the disease invades a community, a large proportion of the population is attacked without distinction. Previous illness affords no protection. Aged and infirm persons and those of nervous temperament are peculiarly liable to suffer, but the robust possess no immunity. All races and dwellers in every clime are liable to the disease. Adults are attacked earlier than children and in some epidemics the latter have manifested a slight relative immunity. A limited number of persons appear to be immune and there are those who, having passed through a series of outbreaks in safety, finally acquire the disease. An attack of influenza confers no exemption from the disease in subsequent outbreaks, and, independently of relapses, which are frequent, individuals have been known to experience a second attack during the prevalence of the same epidemic. The disease bears no relation to known atmospheric conditions. It may prevail at any season of the year. It follows lines of travel and advances at about the ordinary rate of commercial intercourse. The duration of the outbreak in a community is from four to eight weeks, exceptionally a longer time. The epidemic of 1831 was continuously prevalent in Paris for nearly a year. The epidemics rapidly reach their height and usually subside almost as suddenly as they begin. In large cities influenza makes its appearance at the same time in several different localities and spreads from these as foci of infection throughout the community. In the great pandemics of influenza the other acute infectious diseases are less common than usual.

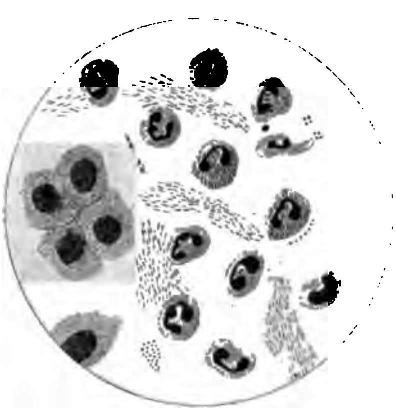


FIG. 235.—Bacillus influenzæ in sputum.

EXCITING CAUSE.—Pfeiffer—1892—isolated from the nasal and bronchial secretions of patients suffering from influenza a bacillus now recognized as the cause of the disease. It occurs in great numbers in the nasal secretions and is frequently seen in the sputum in almost pure culture. It persists in the secretions for some time after the symptoms have subsided.

The disease is readily transmissible by direct contact and fomites; also to short distances by the atmosphere. Influenza bacilli are destroyed by drying, and rapidly perish in water. They probably enter the body by means of the inspired air. Pfeiffer, from a study of the biological characters of the influenza bacillus, concludes that its development outside the human body—that is to say, in the ground or in water—is impossible; that its dissemination when dry can take place only to a limited extent, and that the contagium is, as a rule, transferred by the recent moist secretion from the nasal and bronchial mucous surfaces of influenza patients.

Symptoms.—Influenza presents the greatest variations as regards intensity, from a trifling indisposition to an illness of the gravest kind. In every epidemic the majority of the sufferers manifest the disease in a mild form, very many in a rudimentary form. The symptom-complex is extremely variable and greatly modified by complications and sequels. The period of incubation varies from a few hours to three days. Prodromal symptoms are rare. The onset is abrupt, marked by chilliness or a chill which may be repeated. There is fever, headache (usually intense), with pain back of the eyeballs, severe pain in the back, limbs, and joints, and a general feeling of muscular soreness with tenderness upon pressure. These symptoms are accompanied by mental and physical depression, with malaise and restlessness. The circulation is depressed, the spleen slightly enlarged. In a considerable proportion of the cases catarrhal phenomena do not occur and the attack consists of a fever-storm with its associated phenomena, together with rapidly developing asthenia, more or less profound. The cases may in general be grouped as mild and severe.

In mild cases the chill may be slight or absent altogether. Headache and muscle pains are moderate. There is a sense of weariness upon effort, disinclination for affairs, some difficulty in fixing the attention. Coryza, erythematous angina, and a tickling cough occur. The fever is usually slight, the temperature not rising above 102° F. (39° C.). Many of the patients suffering from influenza in this form are able to continue their ordinary avocations. No great intensification of the symptoms is necessary, however, to compel them to betake themselves to bed.

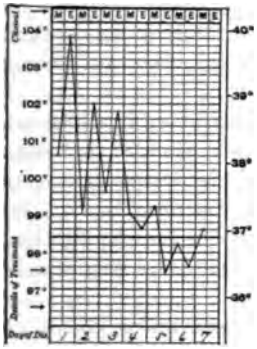


Fig. 236.—Influenza—remittent type.

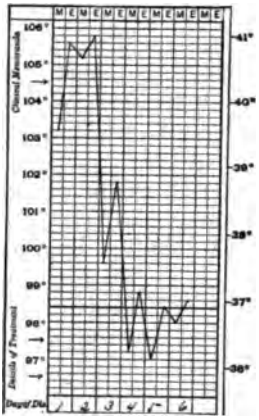


Fig. 237.—Influenza—interrupted crisis.

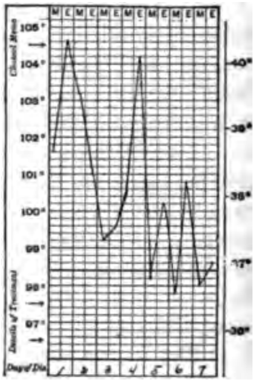


Fig. 238.—Influenza—intermittent type.

In the severer cases the chill is more marked or the shivering more prolonged. Fever is rapidly established, the acme being reached within twenty-four or thirty-six hours. The temperature may rise to 104° or 105° F. (40°–40.5° C.). Sensations of heat alternate with chilliness. There is pain in the orbits and at the root of the nose. Coryza is severe. Epistaxis is occasionally observed. The throat is sore; there are tickling sensations in the upper air-passages, hoarseness, and sometimes dyspnœa. The cough is paroxysmal, distressing, and at first unproductive. Chest pains and stitches in the side also occur. The pulse may be full and compressible; more commonly it is feeble, small, and irregular. It is as a rule only moderately increased in frequency. In some cases there is slight blueness of the lips and finger-tips. The patient is distressed by want of sleep. At the end of four or five days the febrile symptoms decline rapidly, less commonly gradually. The defervescence is often accompanied by copious sweat, spontaneous diarrhœa, increased flow of sedimentary urine, and considerable amelioration of the subjective symptoms. The catarrhal symptoms outlast the fever two or three days, but cough and expectoration may persist for some time. Cutaneous hyperæsthesia is often present and areas of burning pain in the skin occur. Neuralgias may develop during the attack and persist for a long time.

Symptoms referable to the nervous system may dominate the clinical picture. In other cases gastro-intestinal symptoms are conspicuous. The attack may develop abruptly with symptoms like those of cholera morbus. Finally, cases occur in which there is no marked localization of the infectious process. The patient suffers from fever and great depression and simultaneous implication of the respiratory, circulatory, nervous, and gastro-intestinal systems.

Herpes is common. Urticaria and purpura have been observed. The sense of smell is often lost and that of taste impaired or perverted. The hearing is blunted. Febrile albuminuria is not uncommon in the severe cases. A mild anæmia develops in grippe. Leucocytosis does not occur in uncomplicated cases.

Attempts have been made to arrange the cases of influenza in different groups, and in theory a thoracic, cardiac, gastro-intestinal, and nervous variety may be recognized. In practice, however, various described types merge into each other, and are so modified by individual peculiarities of the patient and by complications which arise in the course of the attack that there is but little advantage in referring particular cases to theoretical categories.

The duration of the milder forms of influenza is from one to three or four days. In well-developed cases without complications convalescence sets in between the fourth and seventh days. Severe cases with complications may be protracted for several weeks. Relapses occur in about 10 per cent. of the cases. If the fever continues beyond the seventh or eighth day it will usually be found upon careful examination to be due to some complication. The temperature curve of influenza not infrequently merges into that of a complicating bronchitis, bronchopneumonia, or croupous pneumonia.

Complications and Sequels.—Among the complications, intense bronchitis, implicating the large and small tubes and giving rise to a prolonged symptomatic fever, may occur. This bronchitis has no special peculiarities. The sputum may be abundant and thin, or may be of a greenish-yellow color and nummular. It is sometimes bloody. Bronchopneumonia is not uncommon, especially in children and aged persons. It may be due to the influenza bacillus or to mixed infection. It constitutes a serious complication and is a frequent cause of death. Influenza pneumonia may occur at any time during the course of the attack. Its symptoms are frequently obscure and its course irregular. Extensive involvement of the lung may take place without great rise of temperature. Croupous pneumonia is less common. Abscess or gangrene of the lung may follow the pneumonia of grippe. Pleural effusion is not an uncommon complication and empyema may occur. Pulmonary tuberculosis may develop after an attack of influenza, or, if already present, it is usually aggravated. Endocarditis and plastic or purulent pericarditis may occur in connection with pneumonia or independently of that complication.

Among complications relating to the nervous system meningitis, encephalitis, and brain abscess are to be mentioned. Peripheral neuritis not uncommonly develops during the course of the attack. Headache, insomnia, and neuralgia are common sequels. Forms of neurasthenia occur. Hysteria and chorea have been noted, and psychic disorders, as melancholia and the insanities of malnutrition.

Otitis media constitutes one of the more distressing complications and sequels of influenza. Rapid disorganization of the structures of the middle ear may give rise to permanent deafness. Persistent vertigo may follow influenza. Conjunctivitis is frequent and may be severe. Iritis and optic neuritis are rare sequels. I have seen severe and protracted xerostomia develop after defervescence. Cardiac symptoms are common and distressing. They consist of heart consciousness, precordial pain, breathlessness and faintness upon effort, and unsatisfactory sleep. The physical signs are those of an enfeebled and irregular heart. Arrhythmia, tachycardia, and bradycardia are common. These symptoms are to be ascribed in part to the disturbed nutrition of the heart muscle and in part to the derangements of the cardiac innervation. An attack of influenza has appeared in some instances to be the starting-point of pernicious anæmia. Less common complications and sequels are parotitis, nephritis, phlebitis, venous and arterial thrombosis.

Diagnosis.—DIRECT DIAGNOSIS.—During a pandemic it is unattended with difficulty. The progress of the outbreak, the number of individuals attacked nearly at the same time or in quick succession, the profound asthenia, and the prominence of the nervous symptoms serve to distinguish it from other epidemic diseases. Bacteriological diagnosis can be made by an examination of the bronchial sputum.

DIFFERENTIAL DIAGNOSIS.—*Non-specific Influenza.*—The differential diagnosis between influenza and non-specific catarrhal affections rests upon the pandemic or epidemic prevalence of the former, great prostration, and prominence of the nervous symptoms. The relation of these two diseases is analogous to that between *cholera Asiatica* and *nostras.* The diseases designated by the term *influenza* may be divided into: (1) pandemic *influenza vera,* caused by the bacillus of Pfeiffer; (2) endemic-epidemic *influenza vera,* recurring from time to time locally after the pandemics, caused by the same infecting agent; (3) endemic *influenza nostras*—pseudo-influenza, catarrhal fever—sometimes miscalled grippe—a disease *sui generis.* The infecting micro-organism is not known. *Enteric Fever.*—In the gastro-intestinal form the malaise, headache, dulness of hearing, mental depression, fever, epistaxis, a coated tongue, tender belly, and diarrhœa may suggest enteric fever. An attack of influenza in uncomplicated cases runs its course before the time at which splenic tumor and rose spots establish the diagnosis of enteric fever. The occurrence of influenza during the period of incubation of enteric fever may add to the difficulties of diagnosis. Bacteriological methods and especially the Widal test are necessary in doubtful cases. *Cerebrospinal fever* has prevailed during some epidemics of influenza. The occasional occurrence of cases of influenza marked by painful retraction of the muscles of the back of the neck and vomiting renders the differential diagnosis between these two affections difficult. Nor is the fact to be overlooked that meningitis occurs as a complication of influenza. *Dengue* closely resembles influenza. Each of these diseases occurs in abruptly developing pandemics affecting almost all the inhabitants of the regions invaded. They resemble each other in the frequency of relapse, liability to repeated attacks during the same outbreak, the fact that they are not self-protective, in the want of accord between the gravity of the symptoms and the low death-rate of uncomplicated cases, the suddenness of the attack, intensity of the pains, and the high degree of mental and physical depression. Influenza lacks, however, the cutaneous manifestations, the remission in the course of the fever, and the tendency to arthritis seen in dengue. It differs also in the liability to serious complications and in prevailing in all climates.

Prognosis.—Death is rare in uncomplicated cases except at the extremes of life. The very young bear influenza badly, the aged bear it worse. Previous disease is unfavorable. Individuals suffering from chronic bronchitis, emphysema, myocarditis, and nephritis offer diminished resistance. Exhausting diseases increase the danger of the attack. Cases attended by very severe symptoms usually recover unless the patient be very young or very old or the subject of some complicating malady. The prognosis in individual cases is greatly modified by the character of the prevailing epidemic. In some epidemics the death-rate has been low and the mortality from other diseases only slightly increased. More commonly the death-rate of endemic affections is much increased, and in some of the early epidemics influenza appears to have been attended by a high direct death-rate.

XII. DENGUE.

Definition.—A pandemic infectious disease of tropical and subtropical climates, characterized by a febrile paroxysm with recurrence, intense pains in the joints and muscles, and an early erythematous and a late polymorphous eruption.

The popular term *break-bone fever* denotes the atrocious character of the pain.

Etiology.—Predisposing Influences.—Dengue first excited general attention by its epidemic prevalence in the West India Islands in 1827. Benjamin Rush observed an outbreak in Philadelphia in 1780. Dengue is, in the strictest sense, a pandemic disease. No other disease, with the exception of influenza, prevails so widely and attacks so large a proportion of the population. Equally remarkable is its rapidity of diffusion. In Galveston in the epidemic of 1897, 20,000 persons were attacked in the course of two months. Dengue is a disease of warm climates and of warm seasons. When it has occurred in the summer in temperate climates it has disappeared upon the appearance of frost. The recent experimental investigations of Ashburn and Craig led them to believe that dengue is not a contagious disease, but that it is infectious in the same manner as yellow fever and malaria and that the mosquito at fault is probably *Culex fatigans* (Wied). The liability is universal. Neither age, sex, nor occupation confers immunity. The outbreaks chiefly affect cities, less generally the open country. To this statement, however, there have been many exceptions.

Exciting Cause.—No organism, either bacterium or protozoon, can be demonstrated in either fresh or stained specimens of dengue blood with the microscope (Ashburn and Craig). The pathogenic organism is probably ultramicroscopic.

Symptoms.—The period of incubation varies from three to five days. At the beginning and at the height of epidemics it has not, in some cases, exceeded a few hours. The invasion is abrupt. Prodromes are unusual; when present they consist of lassitude, headache, a furred tongue, loss of appetite, muscular soreness, and chilliness. The attack sets in with intense headache, backache, and severe pains in the joints and muscles. The affected joints are swollen, and the face and neck flushed and turgid. Movements are executed with pain and difficulty. Conjunctivitis, swelling of the eyelids, intolerance of light, and stiffness of the muscles of the eyeballs are common. The tongue is heavily coated, and there is epigastric distress which is followed by nausea and vomiting. Appetite is lost and the bowels are constipated. Thirst is not a prominent symptom. The temperature rises rapidly, often reaching by the end of the first twenty-four hours 106.7° to 107.6° F. (41.5° to 42° C.). The pulse is tense and frequent, 120 to

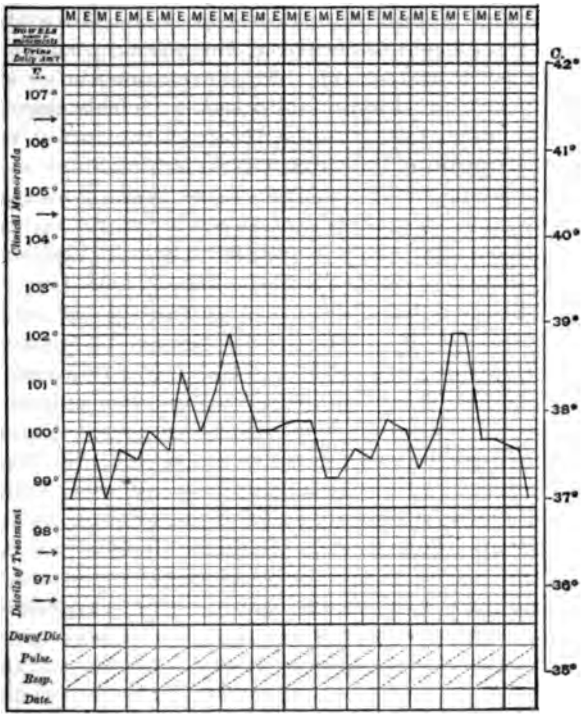


Fig. 239.—Dengue.—Ashburn and Craig.

140. The breathing is rapid, the skin hot and dry. Confusion of thought and delirium occur, and in children the attack may begin with convulsions. An erythematous rash appears in many of the cases.

The duration of the first febrile paroxysm is variable, lasting from a few hours to several days, the average being about three days. Defervescence is commonly sudden and is often attended with critical discharges, such as profuse sweating, epistaxis, or diarrhœa. The eruption now disappears, the skin becomes moist, and there is an amelioration of the pains in the muscles and joints.

The afebrile period lasts two or three days. In some cases it does not occur, or is so brief as to be overlooked. There are cases in which the temperature falls but does not quite reach the normal. Notwithstanding the great relief which the patient experiences, there remain some headache and stiffness of the muscles and joints. At the expiration of several hours, more commonly of two or three days, a second febrile paroxysm sets in. The symptoms are much the same as those of the initial paroxysm but less intense. An eruption of variable character develops at this stage of the disease. It is sometimes macular like the rash of measles, or diffuse and erythematous like that of scarlet fever, or papular. Sometimes it resembles an urticaria, and there are cases in which vesicles occur. Appearing in many instances first upon the hands and feet, this eruption generally invades the greater part of the surface of the body. In other cases it is limited to certain areas. It is attended by distressing itching, and gradually fades after two or three days, being followed by a branny desquamation. The duration of the second paroxysm is from two to three days. Defervescence is gradual; the acute symptoms disappear and the patient enters upon convalescence much enfeebled, the muscular pains and stiffness of the joints often lasting for a considerable time. The small and large joints are alike involved, many being affected at the same time or in rapid succession. The muscles are also stiff and sore, and this, with the swollen condition of the integuments, greatly increases the difficulty and awkwardness of movement, especially in the fingers and hands. In severe cases the mucous membrane of the mouth, throat, and nose is inflamed. The secretion of saliva is sometimes increased and the salivary glands, in particular the parotid, are swollen and tender. The superficial lymphatics about the angle of the jaw and in the groin are in some cases enlarged.

The Blood.—The white corpuscles are diminished in number and there is a relative increase in the small mononuclear variety. The characteristic blood findings are: (1) the absence of a demonstrable protozoon; (2) leucopenia; (3) diminution of polymorphonuclears, and (4) a striking variation in the percentage of other leucocytes at different periods of the attack (Stitt).

The urine during the access of fever is scanty and of high color. With the crisis the quantity is increased. Albuminuria is not usually present.

Diagnosis.—The DIRECT DIAGNOSIS is not difficult. No other disease spreads with such rapidity through a community and attacks so large a proportion of the inhabitants.

DIFFERENTIAL DIAGNOSIS.—Influenza, see p. 702. From acute articular rheumatism dengue differs in its course and duration as well as in the eruptions which attend it; from scarlet fever and measles, in everything except the occurrence of the rashes, which resemble the exanthems of these diseases only in the most superficial way; from relapsing fever in all things except its course, and from yellow fever in many important particulars, among which its extremely low death-rate, the absence of jaundice and black vomit, the infrequency of hemorrhage and albuminuria, and the correlation between the pulse-rate and the temperature are of diagnostic importance. Yet upon these very points experts have failed to agree in the differential diagnosis, as in the State of Texas during the epidemic of 1897. The difficulty is increased by the fact that yellow fever and dengue have the same habitat and very often prevail side by side.

Prognosis.—Dengue is seldom fatal. A few fatal cases have been noted in extensive outbreaks; but in many epidemics in large cities not a single death has occurred.

XIII. DIPHTHERIA.

Definition.—A specific infectious disease, caused by the Klebs-Löffler bacillus and characterized by a fibrinous exudate, usually situated upon and in the mucous membrane of the upper respiratory passages, and by serious constitutional symptoms due to toxins formed in the local lesions.

Clinically the term diphtheria is applied to cases of pseudomembranous inflammation in which the presence of the Klebs-Löffler bacillus can be demonstrated. Cases of pseudomembranous inflammation, which present similar local and constitutional features, but in which Klebs-Löffler bacilli are not present, are designated *pseudodiphtheria* or *diphtheroid angina.*

Etiology.—PREDISPOSING INFLUENCES.—Diphtheria is widely distributed but is especially a disease of temperate climates. The general prevalence is more extensive in winter and it is probable that the frequent occurrence of catarrhal inflammation of the pharyngeal mucous membrane constitutes a definite predisposing factor. Severe epidemics are occasionally encountered in summer. Neither altitude nor the constitution of the soil exerts a special influence. Diphtheria is endemic in cities and towns, where it frequently becomes epidemic, but this is also the case in rural districts, where the population is scattered, and among hamlets and farms it often rages with extreme virulence. The affection spares no class in a community. Rich and poor alike contract it. Crowding, uncleanliness. and neglect of sanitary laws favor the spread of the disease among the poor; therefore epidemics are much more common among the poor than among well-to-do and well-advised persons. These elements of predisposition are fully explained by the fact that diphtheria is a readily transmissible disease. Infants in the first year of life are rarely affected. The period of greatest liability lies between the second and the fifteenth years. The mortality is especially great between the second and the fifth years. The disease may occur at any age and it not infrequently happens that the nurse or a parent contracts the disease from a child, and many physicians have lost their lives from diphtheria contracted in the discharge of duty. In later childhood and adolescence girls are attacked more frequently than boys. The diminished resistance to pathogenic influences on the part of the local mucous membrane which accompanies chronic nasopharyngeal catarrh, adenoid vegetations, hypertrophied tonsils, and laryngitis constitute an especial predisposition to the disease. Certain acute febrile infections, such as measles and scarlet fever, act in the same way. It is important, however, to bear in mind the fact that the pseudomembranous inflammation common as a complication of the acute diseases is more frequently diphtheroid and due to streptococcus infection.

The attack does not confer immunity; on the contrary, certain persons manifest a peculiar susceptibility. There are on the other hand persons who appear to be possessed of a natural immunity and do not contract the disease upon exposure nor, in some instances, even when the bacilli are present in their throats.

Exciting Cause.—The Klebs-Löffler bacilli are non-motile; they vary in length from 2.5 to 3μ and from 0.5 to 0.8μ in breadth, and present different forms, some of which are pointed, others blunt and rounded at the ends, and less frequently forms that are irregular and branching. They are very tenacious of life and have been grown in culture after a non-parasitic existence under varying conditions for periods measured by weeks and months. Attached to clothing, bedding, articles of furniture, even the walls and floor of the room occupied by the patient, they maintain their pathogenic power. Milk serves as a culture media in which they grow rapidly without altering its appearance. The bacilli are found in large numbers in the false membrane of cases of true diphtheria. In this situation they grow and multiply, being rarely found in the blood during life. In the lesions of the bronchopneumonia of laryngeal diphtheria they may be the predominant organism. After death they are occasionally present in the blood and viscera. They vary greatly in virulence. Guinea-pigs, the most susceptible of laboratory animals to the poison, are used for determining the intensity of cultures. Non-virulent bacilli are sometimes spoken of as the pseudobacillus of diphtheria—a misnomer. The pseudodiphtheria bacillus or *Bacillus xerosis* resembles the Klebs-Löffler bacillus morphologically, but differs from it in certain cultural peculiarities and being non-pathogenic. The biological relationship of this organism to the Klebs-Löffler bacillus has not been determined.

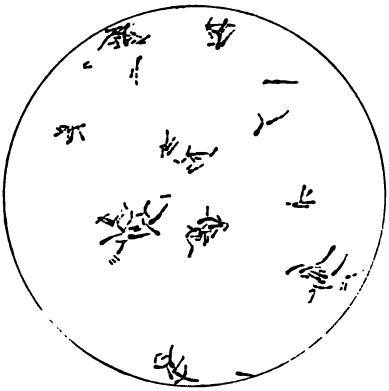


Fig. 240.—Bacillus diphtheriæ from culture on Löffler's blood-serum.

The Klebs-Löffler bacilli may be present upon the mucous membrane of cases which show no membranous exudate and present the clinical picture of a lacunar tonsillitis or a simple erythematous angina. They are very often found in the throat and nose of individuals who have passed through an attack of diphtheria and are fully convalescent. Under these circumstances they may persist for weeks or months, during which time the patient may communicate the disease to others or pass through one or more fresh attacks himself. They are frequently isolated from the throats of healthy persons, both the nurses and attendants upon the sick and others living in the same house with the patient or convalescent. The organisms have been found in the hair and clothing of nurses and in the dust of diphtheria wards. The disease may be communicated by means of infected milk.

The foregoing facts shed considerable light upon the endemic and epidemic prevalence of the disease, as well as the occurrence of sporadic cases. They enable us to understand the persistence of diphtheria in localities, the occurrence of house and neighborhood epidemics, the simultaneous occurrence of scattered cases in a school district, and the development of cases in patients long confined in hospital wards, after visiting days.

The Toxin.—The general symptoms are caused not by the bacilli but by toxins formed by them. Susceptible animals may be rendered immune by the injection of progressively stronger doses of attenuated cultures of the bacilli or increasing quantities of the toxin. By suitable treatment in this manner large animals, as the horse, may be rendered to a high degree immune.

Symptoms.—The period of incubation varies from two or three to seven days. The onset is marked by slight chilliness and sometimes in young children by convulsions. The signs of a febrile infection, elevation of temperature,—102°-103° F. (38.9°-39.5° C.),—malaise, backache, and muscle pains follow. These symptoms vary greatly in intensity. Older children and adults complain of sore throat. An examination of the fauces must be made as a matter of routine in infants who are taken acutely ill. The pseudomembranous exudate rapidly forms upon the mucous surfaces of the tonsils and adjacent parts and shows a marked tendency to spread upwards to the nasopharynx, the nasal chambers and accessory sinuses, the tear duct and the Eustachian tube, or downwards to the epiglottis, larynx, trachea, bronchi. Much less frequently the œsophagus, stomach, and duodenum are invaded. Accidental infection of the vulva and vagina, the ear, conjunctivæ, and wounds occurs. The larynx is primarily involved in a large proportion of the cases.

According to the local lesions the following groups of cases are to be considered:

1. Faucial Diphtheria.—The mucosa is at first reddened and there is difficulty in swallowing. The membrane usually first appears upon one tonsil and, in the course of a few hours, without bridging across, upon the other. After some hours, or it may be a day or two, it has in neglected cases covered the tonsils and spread to the half-arches, the soft palate and uvula, and to the pharynx—pharyngeal diphtheria. Meanwhile, the tonsils are enlarged and the soft palate and uvula are swollen, reddened, and œdematous. The membrane, at first whitish, soon assumes a gray or dirty yellowish-white color. As a rule the patch or patches are distinctly marginate and surrounded by a border of red deeper than that of the general mucosa. The membrane, when forcibly detached, leaves an eroded surface with punctate bleeding, upon which a fresh pellicle soon appears. The

lymph-nodes about the angle of the jaw are swollen and tender. Variations in the character of the exudate occur. It may be throughout punctiform and restricted to the tonsils, or punctiform at the outset but rapidly becomes membraniform, and extends. Again the exudate may be pultaceous rather than pseudomembranous. Finally, there are cases with acute erythematous angina and constitutional symptoms in which no membrane is present but virulent Klebs-Löffler bacilli are found in the secretions. The breath of the patient has a fetid, sickening odor, which surrounds his person and permeates the atmosphere of the room.

2. Nasal Diphtheria. — The Klebs-Löffler bacilli are frequently found in the nasal secretions when the exudate has invaded the pharynx, although no membrane is present in the nasal chambers. When membranous exudate is present two conditions occur. In the first the nares are occupied by a thick, tough membrane, which rarely extends to the adjacent parts and in which Klebs-Löffler bacilli are present, but the constitutional symptoms are very slight or altogether absent. The disease shows very little tendency to affect other children in the family.

The second and far more common form of nasal diphtheria may be primary, but usually arises in the course of the attack by extension from the pharynx or autoinoculation by way of the nostrils. Exceptionally the symptoms are mild, but in the majority of cases both the local and constitutional symptoms are most intense—a fact attributed to the abundant supply of lymph-vessels to the mucous membrane of the nose and consequent free absorption of diphtheria toxin.

3. Laryngeal Diphtheria.—The term membranous croup was at one time used without distinction to designate all forms of membranous laryngitis. This misleading and dangerous custom is fortunately passing away. More than four-fifths of such cases in large series of statistics have shown the presence of Klebs-Löffler bacilli. In a considerable proportion of the rest the result, for various reasons, has been doubtful, while in the small remainder other organisms, chiefly streptococci, have been found. While the clinical symptoms are practically the same, the differential diagnosis can readily be made by laboratory methods, and the membranous laryngitis associated with the Klebs-Löffler bacillus is called laryngeal diphtheria, while that in which other organisms are exclusively present is known as diphtheroid laryngitis or pseudodiphtheritic laryngitis. The latter affection is rarely a primary disease, but usually arises as a complication in the course of some acute disease—scarlet fever, variola.

The local symptoms of laryngeal diphtheria are at first those of an acute laryngitis, with hoarseness and a rough, so-called "croupy" or laryngeal cough. In the course of twenty-four or thirty-six hours the patient—usually a child—suddenly becomes worse, with symptoms of laryngeal stenosis—dyspnœa, slight cyanosis, rapid pulse, aphonia, brassy cough, and restlessness. These symptoms, which commonly develop at night, are at first paroxysmal, with intervals of quiet breathing and sleep. In favorable cases, after two or three paroxysms without marked dyspnœa or cyanosis, the child falls asleep and awakes in the morning greatly improved. Not rarely the attack recurs upon the succeeding night with more intensity.

The respiratory obstruction, which is at first due in part to laryngeal spasm, with the increasing exudate soon ceases to be paroxysmal and becomes continuous with exacerbations and remissions. Inspiration and to a greater extent expiration are increasingly difficult. The auxiliary respiratory muscles are brought into play, the lower intercostal spaces and epigastrium show inspiratory retraction. The voice is reduced to a husky whisper. The cyanosis of the lips and finger-tips becomes more intense. There is urgent air hunger and after a period of extreme restlessness the patient sinks into a semiconscious listlessness, with general relaxation and a freely perspiring skin, only to start up again in the course of a few minutes, tossing about and struggling for air. Occasionally in a severe paroxysm of cough shreds of membrane are coughed up with great temporary or even permanent relief. In other cases, a fold of detached membrane becomes lodged in the glottis and is followed by fatal asphyxia. The fatal issue is, as a rule, preceded by increasing dyspnœa and cyanosis, a period of distressing jactitation, coma, and slight, shuddering convulsions. Pharyngeal exudate may be present, the membrane invading the larynx from above—descending croup; the invasion being from the larynx upward—ascending croup. In many of the cases the membraniform exudate is situated wholly within the larynx, where it may be seen upon laryngoscopic examination—a procedure usually attended, however, with great practical difficulties. If the duration of the attack be prolonged, bronchopneumonia occurs. This complication may be due to an extension of the bronchitis, caused by retained secretions, to the finer bronchial tubes—secondary infection—or to an infralaryngeal extension of the exudate along the trachea and into the bronchial tubes—a true diphtheritic tracheobronchitis. Thus arises respiratory obstruction at two anatomical levels, namely, at the larynx and in the smaller bronchial tubes, a condition often difficult of recognition because of the diminution of tidal air and consequent fainter vesicular sounds and small mucous râles on the one hand, and the loud laryngeal stridor and coarse tracheal râles on the other; a fact of great practical importance because a successful intubation or tracheotomy, which wholly relieves the obstruction at the upper level, can have no effect whatever upon that at the lower level.

4. Other Sites of the Diphtheritic Exudate.—The conjunctiva may be the seat of a primary or secondary diphtheria. In the latter case the extension is by way of the tear duct or by autoinfection. The symptoms may be those of a catarrhal conjunctivitis, the bacilli being present in the secretions, or they may be very serious. The invasion of the middle ear by way of the Eustachian tube may be the occasion of an otitis causing destruction of the tympanic membrane and erosions of the external meatus covered with a characteristic membrane. Vulvar and vaginal diphtheria is occasionally encountered. Diphtheria of the skin occurs in the ordinary forms of faucial and nasal diphtheria when, as is not rarely the case, fissures and abrasions form about the nostrils and corners of the mouth and become infected. The membrane in diphtheria of the anus or genitalia may likewise invade the adjacent cutaneous surfaces. Wounds and ulcerated surfaces in persons suffering with diphtheria are liable to be the seat of an adherent pseudomembrane associated with the Klebs-Löffler bacillus.

The organism may be present in inflamed or necrotic lesions with membrane and, in rare instances, wound infection may occur in the absence of throat affection or traceable exposure to diphtheria cases or fomites. A large proportion of the cases of pseudomembranous inflammation of wounds are due to streptococcus infection or to mixed infection. Local diphtheritic lesions, when severe, are frequently associated with more or less necrosis and gangrene.

In favorable cases the process of separation of the membrane and healing may be observed in faucial diphtheria. After some days the extension of the process is arrested and in the slighter cases the membrane becomes thinner, less distinct at the margins, and gradually disappears. In the more severe forms it appears thicker at the margins, which curl outward from the underlying surface, and separates *en bloc* or by a gradual disintegration. In either case the outlying mucous membrane loses its redness and œdema and shows rapid and marked improvement. Local ulcerations often persist, which, in healing, may give rise to adhesions of the uvula to a tonsil or of the soft palate in part to the wall of the pharynx, and the like.

Infraglottic membranes separate from the underlying surface in more or less extensive membraniform shreds. Pathologically the conditions differ in the mucous membranes above the glottis, which are provided with a squamous epithelium, and in those below it which have a columnar and ciliated epithelium. In the former the membrane is found not only upon, but also in, the substance of the mucosa, while in the latter it is superficial, involving largely the epithelial surfaces and not causing necrosis of the underlying tissues.

Diphtheritic Toxæmia; the Systemic Infection.—There is, in the majority of cases, a general correspondence between the intensity of the local lesions and the severity of the constitutional symptoms. To this rule, however, there are important exceptions. There may be extensive and intense faucial or nasal membranous inflammation with relatively mild general symptoms, or profound toxæmia with limited and apparently superficial local lesions. It has been assumed in explanation of this discrepancy that certain individuals may be more susceptible to the diphtheria bacillus and others more susceptible to its toxins. It is more in accordance with the known facts to explain these differences by assuming that in some instances the bacilli form a larger amount of more virulent toxin than in others and that severe local lesions are in part due to the action of associated organisms—mixed local infections. This explanation finds support in the fact that the graver symptoms are, as a rule, not at first present but arise later when the local disease is at its height. The severest form is septic diphtheria, the outcome of the simultaneous action of the diphtheria bacilli, streptococci, and saprophytic bacteria which are present in the necrotic lesions.

The general symptoms of the attack of diphtheria are those of a mild or intensely severe general infection. The onset is marked by chilliness, a chill, followed by vomiting, fever of atypical course, headache, and anorexia. The temperature varies not only in different cases but also in the course of the attack in the same case. Often but slightly above normal, it sometimes reaches 104° F. (40° C.). In the severest cases the temperature is sometimes subnormal. The pulse in severe cases is small, weak, and irregular, and in some of the gravest cases there is bradycardia. In the septic cases with gangrenous lesions the constitutional depression may be extreme, with frequent thready pulse, high fever, and nervous symptoms, or there may be ashen pallor, great enlargement of the superficial lymph-glands, and a subnormal temperature. A leucocytosis is present alike in the mild and moderately severe cases.

The following visceral changes occur: The toxin of diphtheria acts especially upon the heart muscle and the nervous system. The myocardium shows fatty degeneration. Endocarditis is rare and the bacilli have been found in the lesions. Pericarditis is extremely rare. Pulmonary complications are very common and are often the cause of death, especially in laryngeal diphtheria. The most common condition is bronchopneumonia. Klebs-Löffler bacilli and streptococci are often present, but the organism in the greater number of cases is the pneumococcus. The liver, spleen, and kidneys show the parenchymatous changes present in the severe infections.

Complications and Sequels.—*The Heart.*—Irregular action is common. A faint, blowing, systolic murmur is heard in a majority of the cases. Rapid action, associated with gallop rhythm and epigastric pain, and bradycardia are grave symptoms. Acute dilatation due to granular and fatty degeneration may be the cause of sudden death in the course of an otherwise favorable convalescence. *Paralysis* occurs in from 15 to 20 per cent. of the cases. It is usually incomplete. In rare cases it comes on as early as the seventh day, but commonly not until the second or third week and sometimes later. It is more frequent in adults than in children. Diphtheritic palsy may follow cases in which the local and constitutional symptoms are mild. The palate is most frequently involved, the symptoms being speech having the nasal quality and the regurgitation of fluids through the nose in swallowing. Upon inspection the soft palate is seen to be relaxed and immobile upon phonation. Sensation is likewise greatly diminished. The constrictors of the pharynx may be affected. The intrinsic and extrinsic muscles of the eye are also frequently involved. Strabismus, ptosis, and loss of accommodation result. The loss of power may affect a single limb or the arms or legs together. As a rule it is the result of peripheral neuritis, and the limbs are flaccid, with impairment or abolition of the tendon reflexes. Multiple neuritis is common. The paralysis may affect the extensors of the feet or there may be complete paraplegia. When the arms are involved the patient is often unable to help himself. In other cases an acute ataxia, resembling tabes but without the lightning pains and pupillary phenomena, has been observed. This condition may be attributed to the action of the toxin upon the posterior columns and posterior nerve-roots and is analogous to the derangements of coördination experimentally produced in animals by the injection of Klebs-Löffler bacilli or the diphtheria toxin.

The occurrence of albuminuria is common and may be noted as early as the first day of the attack. This early change in the urine must be regarded as a "toxic" albuminuria. The albumin in favorable cases disappears in the course of some days. In cases of greater severity it persists and red blood-corpuscles and epithelial and hyaline casts appear. The condition is that of an acute nephritis. Anasarca, contrary to the course of the renal affection in scarlet fever, is very rarely encountered and the acute nephritis shows very little tendency to become chronic.

Hemorrhage from the local lesions occasionally occurs in the severer cases of faucial and nasal diphtheria. Epistaxis may be the first symptom.

A diffuse erythematous rash occasionally develops early in the course of the disease. Urticaria is by no means infrequent, and petechiæ and purpuric hemorrhages appear in the later stages of the grave cases. Jaundice, as in other septic conditions, is often present in the worst cases.

Pseudodiphtheria.—The diphtheroid affection is rarely transmitted to other patients or the attendants. As a rule the local process is of moderate intensity and the constitutional symptoms, if present at all, are mild. There are, however, cases in which the most intense streptococcus infection is associated with non-diphtheritic membranous inflammation of the throat or nose.

Diagnosis.—DIRECT DIAGNOSIS.—This rests upon the presence of a false membrane having the characters above described; bacteriologically, upon the presence of the Klebs-Löffler bacillus. There are, however, cases of membranous inflammation in which the Klebs-Löffler bacillus is not present—diphtheroid angina—and the bacillus may be frequently demonstrated in cases presenting the clinical phenomena of an ordinary lamnar tonsillitis or simple tonsillar or pharyngeal catarrh, or in the throats of persons in health. This want of accord between the clinical and bacteriological diagnosis is apparent rather than real. The same thing is seen in other affections, as, for example, tuberculosis. It is a question of the seed and the soil. The bacillus varies in virulence and the individual in power of resistance. The Klebs-Löffler bacillus is the criterion. Membranous inflammations associated with it constitute diphtheria; non-membranous inflammations in which it is present are diphtheritic, as tonsillitis, pharyngitis, rhinitis, and the like. The recognition of the diphtheritic character of many of these milder throat affections marks an important recent advance in practical medicine.

BACTERIOLOGICAL DIAGNOSIS.—For the positive determination of the true character of an acute throat affection a bacteriological examination is often necessary. The material should be taken from the throat as early as possible in the course of the attack and at a time when no antiseptic, and in particular no mercurial preparation, has recently been applied. An immediate diagnosis may sometimes be reached by making a smear preparation. Cultures require about fourteen hours at the body temperature. If the result is negative the examination must be repeated. When the result is positive the examination should be repeated at intervals of ten days or two weeks until the bacilli are no longer found. Pending the result of the examination every acute sore throat in a child must be regarded as suspicious, and measures of isolation and disinfection instituted without delay.

THERAPEUTIC TEST.—ANTITOXIN TREATMENT.—In every case of pseudo-membranous angina the physician without awaiting the laboratory report should at once administer diphtheria antitoxin serum in doses regulated not by the age of the patient but by the severity of the process. The dose varies from 6000 to 8000 units and may be repeated if necessary at intervals of twelve to twenty-four hours. In very grave cases a total dosage of 50,000 or 70,000 may be required. When administered early the serum is followed in the course of a few hours by local and general improvement. The swelling of the faucial mucous membrane subsides, the membrane shrivels and gradually disappears, the sickening odor becomes less intense, the temperature falls to normal, and the pulse loses in frequency and gains in force. Even in apparently hopeless cases improvement and eventual recovery frequently occur. Most remarkable results are seen in laryngeal diphtheria, so that intubation and tracheotomy have become far less common than formerly. The diphtheria antitoxin serum is wholly without effect in pseudodiphtheritic membranous angina.

DIFFERENTIAL DIAGNOSIS.—*Pseudodiphtheritic Angina.*—The majority of the cases of diphtheroid throat inflammation are caused by the Streptococcus pyogenes. They are almost always secondary to other infections, as scarlet fever, variola, measles, or pertussis. The local and general symptoms are less intense. Exceptionally they are severe. The bacteriological findings are diagnostic.

Prognosis.—Since the introduction of the antitoxin treatment the mortality has progressively fallen and is now about 10 per cent. Before this era it ranged from 30 to 40 per cent. Of unfavorable omen in individual cases are extensive or gangrenous exudate, sanious discharge from the nostrils and intense, penetrating, sickening stench, a feeble, thready pulse, cold, clammy hands and feet, and petechiæ. The common causes of death are laryngeal obstruction, bronchopneumonia, sepsis, sudden asystolism, paralysis, and uræmia.

Serum Disease; The Allergic Reaction; Anaphylaxis.—There are two groups of cases: (a) Those following the primary inoculation within five or ten days and comprising local redness, generalized erythemas and urticaria, fever and arthralgias—all of brief duration, and (b) those caused by reinoculations performed not in the course of a few hours or days but after intervals of several weeks or many months and characterized by immediate and intense œdema and urticaria, accompanied in certain cases by shivering, cyanosis, convulsions, coma and even death. Laboratory studies have shown that these effects are due to the serum itself. Other proteid substances acting upon susceptible individuals, when reinoculated, ingested or inhaled, are capable of causing similar though less intense phenomena. Severe reactions are liable to occur in asthmatic persons. Desensitization follows the injection of minute amounts of the serum (0.1 c.c.), the full dose being given an hour later.

XIV. VINCENT'S ANGINA.

Definition.—An acute febrile, pseudomembranous inflammation of the tonsils, associated with *Bacillus fusiformis* and the spirochæta of Vincent, and characterized by a tendency to destructive ulceration of the tissues involved, enlargement of the lymphatic glands at the angle of the jaw, and an irregular, slow course.

This affection is comparatively rare and chiefly affects children and young adults.

Symptoms.—The onset may be acute or subacute, with the symptoms of an ordinary angina or simple membranous sore throat. The constitutional symptoms are often severe. The tonsils are usually at first affected and, in some cases, the local manifestations of the disease are confined to those organs. More commonly the uvula and half-arches are also involved and the ulceration may extend to the pharynx and even to the gums. The exudate is of soft consistency, usually of a greenish or grayish-yellow color, and readily detached, leaving a slightly depressed bleeding ulcer with irregular ragged borders. The process is slowly progressive, not readily yielding to treatment, and may result in extensive destruction of the parts involved, especially the uvula and soft palate.

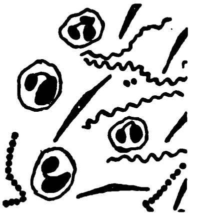


FIG. 241. — Fusiform bacilli and spirilla in Vincent's angina.

Diagnosis.—This form of membranous angina may be recognized by its tardy progress, the destructive tendency of the ulcerative process, and the presence of Bacillus fusiformis and the spirochæta of Vincent. The differential diagnosis from diphtheria rests upon the above anatomical and clinical characters and the absence of the Bacillus diphtheriæ from the exudate. In fact, diphtheria can usually be excluded when the associated fusiform organisms and spirochætæ are present. As a rule, the direct diagnosis depends upon the laboratory findings. Mucous patches may resemble this form of angina or it may develop upon syphilitic lesions.

Prognosis.—Recovery occurs as a rule after a duration varying from four or five days to several weeks. In a recent case in the Pennsylvania Hospital, Vincent's angina was the terminal event in aplastic anæmia in a young adult male.

XV. CROUPOUS PNEUMONIA.

Fibrinous Pneumonia; Lung Fever; Lobar Pneumonia; Pleuropneumonia.

Definition.—An acute infectious disease due to the Diplococcus pneumoniæ of Fraenkel and Weichselbaum and characterized by pulmonary inflammation and fever, usually of abrupt onset, high range, and critical termination.

Etiology.—PREDISPOSING INFLUENCES.—Pneumonia prevails alike in hot and cold countries. It is said to be more prevalent in the temperate climates.

Season plays an important part as a predisposing factor. The incidence of the disease is uniformly greater in winter and the early spring. March is the month of greatest liability. This is in accordance with the fact that exposure to cold and especially unusual exposure to cold and wet are very often soon followed by the initial chill. Steady, low temperatures are less dangerous. *Personal Factors.*—Croupous pneumonia may occur in the new-born and in early infancy. It is common till the sixth year. The liability then diminishes until the fifteenth year. From fifteen to forty-five is a period of special liability. After sixty the disease is very common and often constitutes the terminal event in the aged, both in chronic disease and when the previous health has been well preserved. In infancy and old age the incidence for the sexes is about equal; in the middle period of life, when the mode of living is different, males are more frequently affected than females. There is no special racial predisposition. The negro bears the disease badly. Rich and poor are alike liable. No occupation is conspicuous as a predisposing factor. Overwork and sudden exposure constitute especial risks. Lumbermen and miners frequently suffer. The disease is very common in cities. Pneumonia attacks the robust and hearty and the debilitated and previously ill with impartial energy. The alcoholic is especially liable. The last illness of the chronic invalid is very often pneumonia. Pneumonia sometimes follows injuries, especially contusions of the chest. This may occur in the absence of the signs of injury to the lung. The term "contusion pneumonia" is used to describe this variety.

Immunity.—There is apparently no natural immunity, certainly no permanent acquired immunity. On the contrary, croupous pneumonia is conspicuous among the infections for its liability to recur. Subsequent attacks have been noted in from 15 to 50 per cent. of the cases. I have seen a woman who stated that the attack was the twelfth, and several persons in whom a number of annual attacks occurred.

A powerful protective serum has been obtained by the repeated inoculation of various animals, as the horse, ass, and cow, with dead and living cultures of the pneumococcus. The specific protective substances—opsonins—are formed in the bone-marrow and, circulating in the blood, enter into chemical combination with the pneumococci which are thus rendered an easier prey to the phagocytes. The formation and gradual accumulation of similar substances in the human body during the attack serve to explain the crisis. The organisms are rapidly destroyed within the exudate in the lungs and in the circulating blood. The duration of the local and general immunity which follows is uncertain.

Exciting Cause.—Diplococcus pneumoniæ of Fraenkel and Weichselbaum, Micrococcus lanceolatus, or pneumococcus. This organism is the sole cause of true acute croupous pneumonia. It is present in the expectoration and pulmonary exudate in enormous numbers. Upon examination of the lungs, when death has occurred in the stage of resolution, it is found only in small numbers or may be wholly absent—phagocytosis. It may be obtained in blood cultures during the attack by the more recent methods in more than 75 per cent. of the cases. The pneumococcus occurs in many other diseases, especially bronchopneumonia, pleurisy, endocarditis and pericarditis, meningitis, peritonitis, in forms of arthritis, and in middle-ear disease.

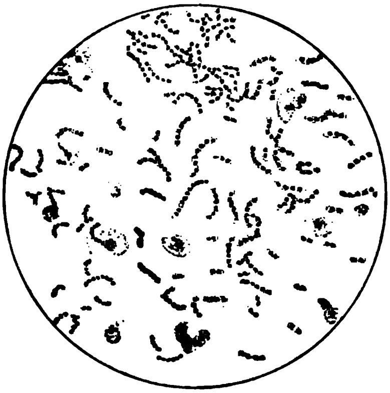


Fig. 242.—Spread of sputum showing pneumococci in pairs and in chains.

Pneumococcus Septicæmia.—An acute general infection without localization in the lungs or serous membranes is occasionally encountered. This variety of pneumonia is analogous to the primary septicæmia due to general infection by Eberth's bacillus—typhoid septicæmia.

In its attenuated forms the pneumococcus is present in the secretions of the mouth and bronchi in a large proportion of healthy individuals. Its presence has been demonstrated in the dust of rooms. Various other organisms are associated with the pneumococcus in croupous pneumonia, as secondary or mixed infections. The most frequently encountered is the Streptococcus pyogenes. Less common are staphylococci, the bacillus of Friedländer, B. typhosus, B. diphtheriæ, and B. influenzæ. These bacteria are often the cause of bronchopneumonia, which in some instances closely resembles croupous pneumonia. The latter is a distinct, specific disease uniformly caused by the pneumococcus, and the recognition of its etiological and clinical independence is a matter of great practical importance.

Pathological Anatomy.—The lesions in the inflamed lung undergo progressive changes, which, since the time of Laennec, have been described as the stage of engorgement, the stage of red hepatization, and the stage of gray hepatization. *Engorgement.*—The vesicular tissue is deep red, firmer than normal to the touch, and on section shows abundant blood and serum. It crepitates upon pressure, and excised pieces float upon water. The capillaries are distended and the air-vesicles contain blood-corpuscles and swollen detached alveolar cells. *Red Hepatization.*—The air-cells and terminal bronchi are occupied by the coagulated exudate, entangled in which are pneumococci, red blood-corpuscles, leucocytes, and alveolar epithelium. The affected portion of lung is solid and airless. It is enlarged and shows the oblique, parallel markings of the ribs. On section the surface is of a reddish-brown color and granular, an appearance produced by the protrusion of the fibrinous moulds in the vesicles. The terminal bronchi also contain branching fibrinous casts. The surface yields upon scraping a reddish, viscid serum. The hepatized lung is extremely friable. *Gray Hepatization.*—The lung tissue is now of a dirty gray color and more friable. The cut surface is more moist, has lost its granular appearance, and yields upon scraping a milky turbid fluid. The air again reaches the alveoli, from which the fibrin and red blood-cells have in great part disappeared, but in which are great numbers of leucocytes. The foregoing stages gradually merge into each other and the process is not equally advanced in all parts of the lesion. The liquefaction of the exudate and resolution are the result of the action of proteolytic enzymes. The part of the lung occupied by the exudate varies from a small patch to the entire lung. Commonly a single lobe is involved. The uninvolved portions are usually congested and œdematous. The pleural surface opposite the exudate is always inflamed when the latter extends to the periphery of the lung. The bronchial glands are enlarged and sometimes softened.

The right heart is dilated. Pericarditis is not rare, especially in left-sided and double pneumonias. Endocarditis is common both in the simple and in the malignant form. Myocardial changes occur. The spleen shows moderate enlargement and, in the kidneys, parenchymatous swelling and the lesions of interstitial nephritis are frequently present. Meningitis is by no means rare and is often associated with malignant endocarditis. Diphtheroid colitis is rare. The liver is slightly enlarged and deeply congested. The distribution of the lesion is as follows: The right lung alone is involved in about 50 per cent. of the cases; the left alone in about

:ent.; both in less than 20 per cent. In double pneumonia the bes are usually affected, or an entire lung with the lower lobe ther. Much less frequently the lower lobe of one lung and the be of the other are involved—*crossed pneumonia.* Very rarely per lobes suffer.

ıpous pneumonia in the majority of the cases occurs as a sporadic At the seasons of greatest prevalence it is the type of an endemic House epidemics are by no means rare. In a family of five I n the mother and two children attacked in rapid succession, two cases proving fatal. More e local epidemics occasionally schools, prisons, and other ɔns.

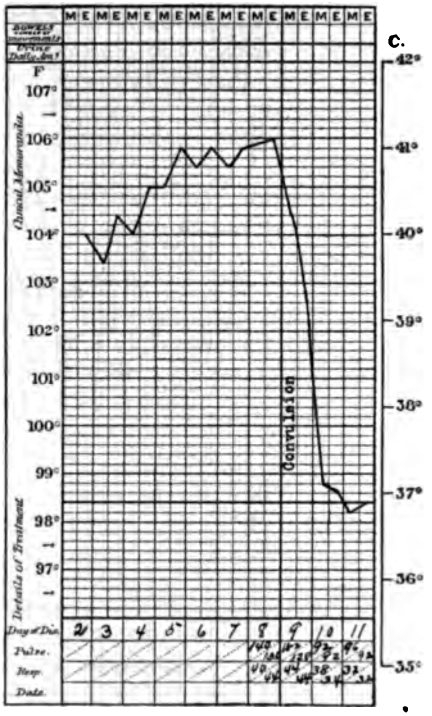


FIG. 243.—Pneumonia ; acute nephritis. Convulsion at crisis. Recovery.

ıptoms.—In the well-devel- ses of croupous pneumonia :k runs a typical course and ıited. The period of incuba- ıf unknown duration. It is · brief. Prodromes are unus- hen present they consist of .tarrhal symptoms. The abrupt, with a chill which is ly severe and prolonged. perature rises rapidly to the ı—104°-105° F. (40°-40.6° :adache, general pains, and .tion of being very ill are fol- the course of a few hours by stitch-like pain in the side. l on full breathing, a short h, hurried respiration, and a ding pulse. Very significant rt, expiratory grunt. The 'ten characteristic. The face y cyanotic, with dusky, cir- ed flushing of one or both the eyes are bright; the ın anxious; the nostrils dilate, and later patches of herpes, usually of many small vesicles, some of which contain blood, appear upon ınd nose. The patient lies upon the affected side or flat upon his y the close of the second day there is scanty, viscid, blood-stained ation—*rusty sputum.* The physical signs of consolidation of the lung tissue are present—small mucous and crepitant râles followed ss and bronchial breathing. At the end of several days deferves- crisis occurs with a remarkable amelioration of all the symptoms.

following symptoms require special consideration: FEVER.— perature rises rapidly, sometimes reaching the fastigium within ours, usually within twenty-four hours. In childhood and old ·ise is more gradual, especially if there is no chill. Its course is subcontinuous and remarkably constant, the morning remissions and evening exacerbations in many cases not greatly exceeding the diurnal oscillations in health. Pseudocrises are not infrequent. They may occur at any period but are more common about the fifth or sixth day. They are usually single, but two or more sometimes occur. In the latter case the temperature range suggests an irregular intermittent. The defervescence in so-called classical cases is by crisis, which occurs between the third and twelfth days, very often upon an uneven day, and commonly upon the fifth or seventh day. A precritical rise of a degree or more is not very rare.

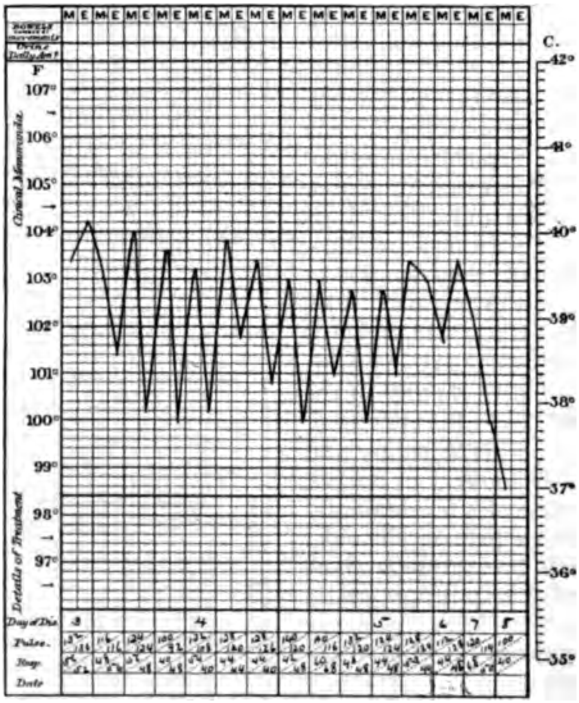


FIG. 244.—Croupous pneumonia. Recovery.

The time occupied by the crisis varies from two to several hours. It is accompanied by an abundant sweat and usually occurs during a deep and prolonged sleep, from which the patient awakes weak but refreshed and comfortable. The fall almost always reaches subnormal ranges—96°-97° F. (35.5°-36° C.), and may be followed by a postcritical rise and subnormal oscillations for a few days. In some instances the crisis is interrupted by a rebound—*interrupted crisis*—or it may extend over twenty-four hours—*protracted crisis*. In delayed cases and in children the defervescence is often by lysis. In fatal cases of the so-called sthenic type there may be a preagonistic rise of temperature and in the asthenic cases an abrupt antemortem fall of several degrees. The crisis is sometimes attended by collapse symptoms. In the aged and in drunkards the temperature is much lower. Afebrile cases are encountered. PAIN.—The pain all the characters of pleurisy. It is stitch-like and lancinating, usually ere, aggravated by deep breathing and cough, and referred to the region the nipple or the infra-axillary region on the affected side. Occasion- it is referred to the epigastrium or the region of the appendix: an ortant point for the diagnostician. In these cases there is a diaphragtic pleurisy. In apex pneumonia pain is less constant and less severe in central lesions it is absent. DYSPNŒA.—The respiration rate is reased in almost all cases, the masked pneumonias of drunkards and aged and the terminal pneumonias of chronic diseases constituting

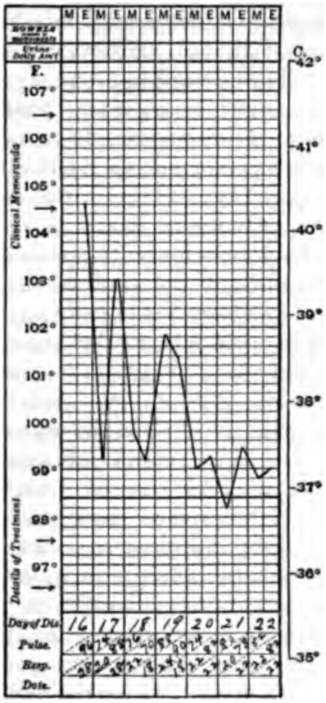


FIG. 245.—Croupous pneumonia. Prolonged febrile movement, terminating by lysis on 21st day of attack.

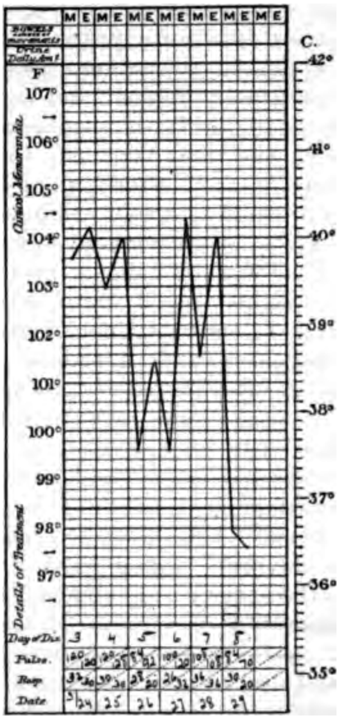


FIG. 246.—Pneumonia; pseudocrisis, 5th day; crisis 8th day.

exceptions. The pulse-respiration ratio may be 2:1 or even 1.5:1. respiration-frequency ranges from 30 to 50, and in children as high as per minute in cases that recover; in fatal cases it is even more rapid. breathing is shallow; the ancillary muscles are brought into play; re is, in many cases, respiratory distress associated with air hunger and haracteristic expiratory grunt or snort. COUGH.—The early cough is to pleural irritation and is short, hard, and dry. Later it becomes uent, somewhat paroxysmal, and productive. It is attended with n, often severe throughout the attack. About the time of the crisis, it omes easier and is attended with free expectoration. Terminal pneunias and those of the aged and drunkards are often without cough.

Absence of cough is also occasionally observed in the pneumonia of infancy. Slight cough after the crisis is often without significance; but severe paroxysmal cough may be a sign of pleural effusion. SPUTUM (see Part III, page 456). PULSE.—At the onset the pulse is small, but it soon becomes strong and full. It is seldom dicrotic. Later it becomes feeble and small. The frequency varies in favorable cases from 80 to 110. A very frequent pulse—130–140—is of unfavorable prognostic import, but less so in children than in adults. In feeble and aged persons the pulse is small and frequent from the onset. After the crisis the pulse remains frequent for a time but gradually returns to normal. The peripheral blood-pressure during the early days of the attack shows little or no change. Later there is often a progressive fall. A sudden drop may be the precursor of death. In the septic cases there is an early fall. HEART SOUNDS.—They are usually distinct and well-defined. The second pulmonary sound is accentuated. With engorgement of the right heart and incomplete systole of the right ventricle, the pulmonary second sound becomes progressively fainter. Great impairment of heart power shows itself in heart sounds of the fetal type. Sudden collapse may occur early in the disease, at the crisis, or during convalescence, and terminate in death. This may happen without warning in previously healthy persons with good hearts. BLOOD.—A decrease of the red corpuscles occurs at the time of the crisis but marked anæmia is not common. A leucocytosis varying from 10,000 to 50,000 per c. mm. is present in most cases throughout the attack. It disappears after the crisis. Its persistence may be the sign of a complication. It bears some ratio to the extent of the pulmonary exudate. In the toxic cases leucocytosis may be absent, and its absence in any case is an unfavorable sign. The blood-plaques and fibrin elements are increased. The eosinophile cells are decreased. Pneumococci can be isolated in some cases. DIGESTIVE SYSTEM.—There is complete anorexia. The tongue, at first covered with a thick white fur, becomes red and glazed and in the severer cases dry and brown. Vomiting is frequent in children. Constipation is the rule. Meteorism is a troublesome condition in the graver cases. Fibrinous exudates have been observed in the mouth and nose and other mucous surfaces. SKIN.—Herpes is very common—20–60 per cent. of the cases. It appears usually upon the lips at the border of the mucosa; less frequently upon the alæ nasi, infrequently upon the genitalia or anus, rarely upon the buttocks. Slight cyanosis may occur. Redness of the cheeks, and especially of the cheek upon the affected side, is very common. A general erythema is encountered in rare cases. As in all grave infections petechiæ may occur. Sweating is not common during the course of the attack, but is profuse at the crisis. NERVOUS SYSTEM.—The symptoms referable to the nervous system are not peculiar to pneumonia but are, in certain cases, of great assistance in the diagnosis. In infants and young children convulsions may take the place of the initial chill. Headache is frequent and often severe. Insomnia is a troublesome symptom, often followed by delirium. The latter may be mild and wandering, becoming progressively more severe, even increasing after the crisis. In the intervals there is marked mental confusion. In a group of cases in children the symptoms suggest meningitis, and the actual condition is very often overlooked. There are cases in which the onset is marked by furibund mania. In alcoholic cases the nervous phenomena closely simulate delirium tremens. Finally there are cases characterized from the onset by dulness and stupor, with no chill and but little fever, in which pulmonary symptoms are nearly or quite absent, but grave depression and wandering delirium constitute the only manifestations of profound toxæmia. The true character of such cases can only be determined by a systematic routine examination of the chest. Apex pneumonia is more frequently attended by severe nervous symptoms. The attack may be followed by postfebrile delusional insanity, which as a rule terminates in recovery. URINE.—The secretion has the usual characters of fever-urine. Toxic albuminuria is common. Later the signs of an acute nephritis may be present. Urea and uric acid, diminished during the attack, are greatly increased upon the occurrence of crisis. The chlorides are diminished or absent.

Physical Examination.—INSPECTION.—The attitude is variable. In lung lesions of moderate extent the patient lies upon his back or upon the affected side; in pneumonia of an entire lung, or double pneumonia, he usually prefers to be supported by pillows. The respiratory movement of the affected side is diminished. In basal pneumonia there may be increased movement over the upper lobe. The increased compensatory excursus on the sound side is often very conspicuous. The frequent breathing, the action of the auxiliary muscles of respiration, and the sudden muscular relaxation in expiration are to be noted. Orthopnœa may be present in severe cases. The affected side may look larger, but the increase upon actual measurement is trifling.

The difference in the expansion of the two sides is very evident upon palpation. The vocal fremitus is greatly increased over the lesion. It may be diminished or absent if the exudate extends into the middle-sized bronchi or a plug of tenacious mucus occludes a tube of some size.

PERCUSSION.—During the stage of engorgement the resonance is of higher pitch and vesiculotympanitic—*Skodaic resonance.* After hepatization has occurred percussion yields dulness, which varies from partial impairment of resonance with the tympanitic quality, to almost complete loss of resonance. Flatness is only present in massive pneumonia when the fibrinous exudate extends some distance into the larger bronchi. Beyond the borders of the lesion percussion often yields Skodaic resonance. As resolution takes place the dulness becomes less marked; the quality becomes vesiculotympanitic and by degrees the normal pulmonary resonance is restored. A certain elevation of pitch and faint tympanitic quality may persist for several weeks. Wintrich's phenomenon is sometimes present in apex pneumonia. In rare cases the percussion sound has an amphoric quality and suggests a cavity. In central pneumonia the symptoms may be well marked but percussion may fail to indicate the site of the exudate until it reaches the periphery of the lung, sometimes a period of several days.

AUSCULTATION. — In the stage of engorgement faint respiratory sounds. The tidal air is not only decreased in volume, it also ebbs and flows with diminished force. Then follow crepitant râles, heard only at the end of inspiration—*crepitus indux.* In the stage of red hepatization, when dulness appears, the respiration becomes bronchial, at first soft and low-pitched, and more distinct upon expiration. Fully developed it is high-pitched, heard alike upon inspiration and expiration, with an interval of silence between the inspiratory and the expiratory sound and often, especially in the young, having a loud, snoring quality. In massive pneumonias in which the exudate fills the bronchi bronchial breathing is absent. Upon resolution small mucous and crepitant râles are again heard—*crepitus redux*—and are sometimes followed by larger bronchial râles which disappear as convalescence advances; more frequently by nearly or quite normal vesicular breathing. In central pneumonias the auscultatory like the percussion signs may be absent for a time. The variety of bronchophony known as ægophony is sometimes present, but it is a sign of trifling importance.

After the diagnosis is fully established it is not desirable to make frequent examinations of the chest. They are exhausting to the patient, especially as the crisis draws near, and in the absence of some special indication in the symptoms or general condition should not be repeated oftener than once in three or four days. In the necessary movements the patient must be carefully assisted and make as little effort on his own part as possible.

Complications and Sequels.—These are not many. PLEURISY.—Inflammation of the pleura corresponding to the exudate is always present when the latter extends to the periphery of the lung. It is usually fibrinous and cannot then be regarded as a complication. When serofibrinous, the effusion usually contains coarse fibrin flakes and there is much soft fibrinous deposit. It is often abundant. Even a moderate effusion coming on during the stage of hepatization may cause urgent pressure symptoms. A rare complication is pleural effusion upon the opposite side.

METAPNEUMONIC EMPYEMA.—This complication is not altogether infrequent. Cases are sometimes regarded as instances of delayed resolution. The pneumococcus is usually present early; the streptococcus afterwards. The signs of pleural effusion may appear during the attack or after the crisis. In the former case there may be pressure symptoms, as dyspnœa, cardiac embarrassment, and sensations of tightness, together with persistence of the fever. In the latter, the temperature rises and becomes remittent or intermittent and there are irregular profuse sweats, marked anæmia, leucocytosis, and not rarely paroxysmal cough. The diagnosis of small encapsulated and interlobar empyemata may often be made with confidence in cases in which their precise location remains obscure.

PERICARDITIS.—This complication is comparatively infrequent—5 per cent. It occurs chiefly in left-sided or double pneumonias. The exudate is usually fibrinous. Precordial pain may be overlooked in connection with the pleurisy. A friction sound may be obscured by bronchial râles. The effusion may be serofibrinous or purulent.

ENDOCARDITIS.—Primary endocarditis may occur, or a fresh attack supervene in chronic valvular disease. The malignant form is occasionally associated with meningitis. The signs are not constant. Of diagnostic importance are murmurs which change their quality or point of maximum tensity, irregular fever with chills and sweating, and signs of embolism. iere are cases discovered post mortem in which no murmur has been cognized during life.

Thrombosis.—This condition may occur during convalescence. The moral vein is commonly affected. Embolism of the larger arteries is ry rare. Aphasia is also rare. It may occur with or without hemiplegia.

Meningitis is a rare complication occurring during the course of the tack or after the crisis. It has been observed more frequently during idemics of cerebrospinal fever. It constitutes a most serious complition. The pneumococcus has been found in the exudate. Very rare deed are multiple neuritis, myelitis, and an ascending paralysis presenting ie characters of Landry's paralysis. Postinfective insanity is rare. In dividuals predisposed to neurasthenia an attack of pneumonia may ecipitate the outbreak.

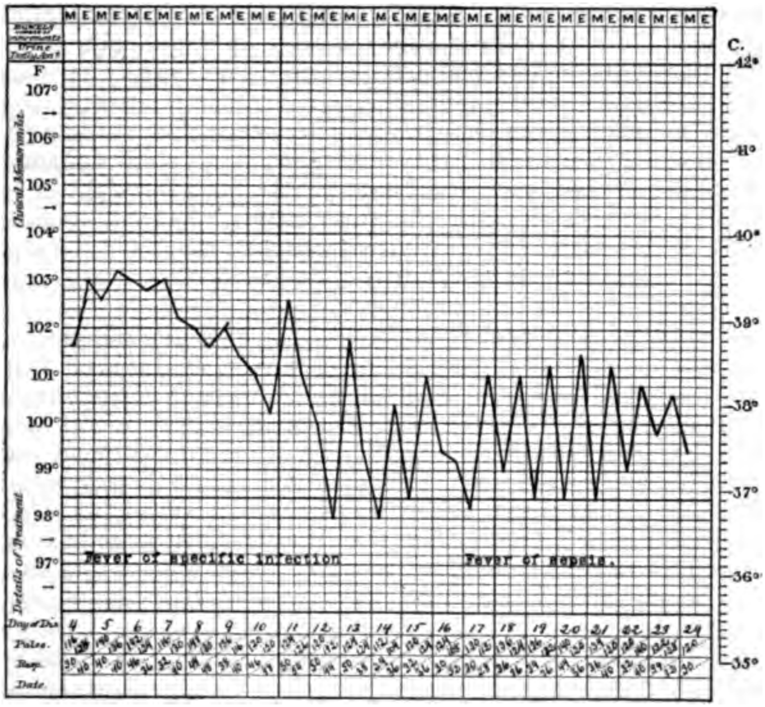


Fig. 247.—Pneumonia followed by empyema.

Abdominal Complications.—Croupous gastritis and colitis and acute latation of the stomach are rare. Appendicitis may occur as an intercurrent ection. Epigastric pain, which is especially common in children, is usually ie to diaphragmatic pleurisy. It may simulate peritonitis—a fact of imrtance in diagnosis, since in rare instances inflammation of the upper ritoneum by extension from the pleuræ has been observed. The abdominal in, if localized and attended with shock, may suggest acute hemorrhagic ncreatitis.

Jaundice.—Mild jaundice is not uncommon. It develops early and has no bearing upon the prognosis. A deep obstructive jaundice may occur.

Meteorism.—Abdominal distention is a common and troublesome symptom in the graver cases. It is the manifestation of the action of toxins upon the nerve supply of the walls of the gut. By mechanically interfering with the action of the diaphragm it adds to the embarrassment of the respiration.

OTHER COMPLICATIONS.—Parotid bubo occasionally occurs, especially in connection with endocarditis of malignant type. Middle-ear disease is common in children, and polyarthritis resembling that of rheumatic fever, sometimes clearly septic in character, may occur during the course of the attack or during the convalescence.

RELAPSE is a rare event. It is important not to mistake the fever of delayed resolution or of empyema for relapse. An initial chill, high fever, cough, rusty sputum, and critical defervescence would justify the diagnosis of relapse.

CONVALESCENCE is commonly rapid. Resolution does not immediately occur. Impaired resonance with the vesiculotympanitic quality and feeble vesiculobronchial respiration may persist for a fortnight or longer. Fever and a leucocytosis after the crisis suggest delayed resolution or empyema. A systematic physical examination and the use of the aspirator needle may become necessary. Persistent dulness is often due to a greatly thickened pleura.

Anatomically the terminations of croupous pneumonia are:

1. RESOLUTION.—The exudate undergoes liquefaction and resorption. Only in small part is it expectorated. Complete restoration of the lung gradually occurs—*restitutio ad integrum.*

2. DELAYED RESOLUTION.—Resolution is more commonly delayed in pneumonias of the aged and in debilitated subjects; it may be delayed also in basal lesions and in previously robust persons. The crisis is followed by an apparent convalescence, but the signs of local consolidation continue. From three to five or six weeks elapse before dulness and bronchial breathing wholly disappear. Complete recovery ultimately follows. In a second group of cases the defervescence is by lysis, with irregular, recurrent fever, sweating, rapid pulse, slight cough, usually little or no expectoration, and impaired nutrition. The condition simulates pulmonary tuberculosis. In the course of four or five weeks, sometimes not until two months or more have elapsed, complete resolution takes place and the patient regains his health.

3. ABSCESS.—Local infection with pyogenic organisms. The abscess cavities are multiple and small or they may coalesce and form an extensive depot. Cough is paroxysmal and accompanied by an abundant purulent expectoration containing elastic fibres, sometimes cholesterin crystals and hæmatoidin crystals. The onset of the condition is commonly attended with increased fever of hectic type and other signs of sepsis. The differential diagnosis between a circumscribed empyema with bronchopulmonary fistula and pulmonary abscess rests largely upon the character of the sputum.

4. GANGRENE.—Infection with saprophytic bacteria. The condition may occur independently of or in connection with abscess. The condition occurs mostly in persons debilitated by previous bad health. It

manifests itself clinically by septic phenomena and extreme prostration and by a disgusting, penetrating fetor of the sputum and breath.

5. Fibroid Induration.—Defervescence either by crisis or lysis may occur, but the signs of consolidation—dulness, increased vocal fremitus, bronchial respiration—continue unchanged. Occasional râles are heard. Cough may be troublesome, but expectoration is scanty. Resolution does not take place gradually as is usual, but the signs become more marked with contraction of the opposite side and the gradual development of chronic interstitial pneumonia—fibroid phthisis. In other cases the fever returns, the expectoration increases, and the course of the case is that of a rapid pulmonary consumption—phthisis florida.

Clinical Varieties.—The clinical picture of croupous pneumonia is made up of two essentially different groups of symptoms, namely, those due to the local pulmonary lesion and those due to the toxæmia. As these vary greatly, it is evident that the individual cases, while conforming to a type, will present differences which are determined among other factors by the relative preponderance of one or the other of these two symptom-groups. Complete consolidation of a lung may, on the one hand, be accompanied by but trifling evidences of general infection, while, on the other hand, severe, even fatal, toxæmia occasionally occurs in cases in which the pulmonary lesion is limited in extent. The other factors are: 1. Local Variations.—(a) *Apex pneumonia* is more common in children. It is frequently associated with marked cerebral symptoms and may simulate meningitis. In the absence of cough and sputum the pulmonary lesion is often overlooked. Pneumonia of the apex in adults may be accompanied by grave constitutional symptoms. (b) *Double pneumonia* is attended with the additional dangers incident to progressive diminution of the respiratory surface. The second lung is usually involved a day or two after the first and to a less extent. The extension of the process is not attended by a chill. (c) *Central Pneumonia.*—The exudate may remain circumscribed about the root of the lung or in the substance of a lobe and not reach the periphery for three or four days, when the physical signs may be for the first time detected. Meanwhile characteristic symptoms—chill, fever, cough, and rusty sputum—are present, but pain does not occur until the inflammation reaches the pleura. (d) *Pneumonia in Emphysematous Persons.*—The symptoms are distinctive, but, owing to the diminished vascular supply and the dilatation of the vesicles, there is not a sufficient quantity of fibrinous exudate to give rise to the signs of consolidation. Several days may elapse before the site of the lesion can be detected. (e) *Massive Pneumonia.*—A rare form in which the fibrinous exudate fills the bronchi. A lobe or the entire lung may be involved. The affected portion is converted into a completely airless mass. The percussion sound is not dull but flat. Upon auscultation neither râles nor bronchial respiration are heard, and vocal fremitus is absent. The signs closely simulate pleural effusion, but adjacent organs are not displaced. (f) *Migratory Pneumonia.*—The inflammation creeps about, involving in succession one lobe after another, resolution, not always complete, taking place in turn as new areas are affected. The migrations are not attended by chills; fever continues and the course of the disease is much protracted.

2. THE INTENSITY OF THE PROCESS.—(a) *Larval or Rudimentary Pneumonia.*—Mild cases occasionally occur. The symptoms consist of slight chill, moderate rise of temperature, and cough. The pulmonary signs are obscure. The expectoration is slight in amount and not usually rusty. Herpes is common. The attack comes to an end in the course of two or three days. Its true nature is often overlooked. (b) *Abortive Pneumonia.*—The attack begins with a severe chill. The fever is high. Pleural pains, cough, rusty sputum, herpes, and characteristic physical signs enter into a symptom-complex which is complete. In the course of the second or upon the third day the temperature falls by crisis and the patient enters upon convalescence. (c) *Intense Pneumonia; Sthenic Pneumonia.*—The adjectives sthenic and asthenic have to a great extent passed out of use, but almost every day one sees in a large hospital service cases of pneumonia of great severity but in strong contrast as regards the character of the symptoms. Those terms serve a useful purpose in this connection. Intense pneumonia occurs in middle life, in individuals previously in good health, usually males, large, deep-chested, hard workers in the open air. The initial chill is severe and prolonged, the temperature high, the pulse bounding, the face flushed, chest pain very intense, the breathing hurried, cough frequent, sputum hemorrhagic, the delirium active, even maniacal, and the signs indicative of an extensive pulmonary inflammation. These cases are attended with especial danger to life. (d) *Toxic, Asthenic or So-called Typhoid Pneumonia.*—This variety, equally severe and even more dangerous, is in the strongest contrast to the variety just described. The ordinary symptoms of pneumonia may be absent and the pulmonary lesions limited. The symptoms are those of profound toxæmia. Early prostration, delirium, jaundice, meteorism, and diarrhœa are present. The hands and lips tremble and stupor alternates with wandering delirium. The condition is one of pneumococcus septicæmia or mixed pneumococcus and streptococcus infection.

3. INDIVIDUAL TENDENCIES.—(a) *Age.*—In the new-born croupous pneumonia is extremely rare. It is common in infants and young children. Convulsions replace the initial chill. The apex is not rarely involved. Cough is slight and suppressed or absent altogether. There is no expectoration. Excitement, jactitation, boring of the head into the pillows, and high fever followed by stupor and convulsions suggest meningitis. The apex pneumonia is often wholly overlooked. Pneumonia in the aged usually develops insidiously without a distinct chill. There is little cough and expectoration. Fever is moderate and irregular and the physical signs not well defined. Great depression, inability to take nourishment, mild delirium, and a tendency to stupor are present. (b) *Sex.*—In women at the middle period of life pneumonia tends to assume the toxic form, in men the sthenic form; in infancy and old age the course of the disease is the same in males and females. (c) *Pneumonia in Alcoholic Subjects.*—Two forms are to be considered—pneumonia in steady drinkers and pneumonia during debauch. In the first the early symptoms do not differ from those of ordinary pneumonia. Delirium with tremor soon develops, vomiting is troublesome, the circulation fails, sleeplessness is uncontrollable, and the signs of nephritis with uræmic phenomena are common. In the second

he onset is insidious, the temperature but slightly raised, cough, expecoration, and sputum trifling or wholly absent, and the clinical picture that f delirium tremens. Only by a systematic physical examination can the ondition be recognized. (d) *Pneumonia in Chronic Diseases; Terminal Pneumonia.*—The terminal event in many chronic diseases, especially ulmonary tuberculosis, valvular and myocardial disease of the heart, rteriosclerosis, nephritis, diabetes, cancer, and diseases of the spinal cord, s croupous pneumonia. The development of this intercurrent disease is ery frequently overlooked, partly because it is very insidious and presents one of its ordinary symptoms, and partly because the patient has reached point in the progress of the primary affection in which a proper physical xamination can no longer be made. The diagnosis is frequently made in he post-mortem room. The *intercurrent pneumonias of the acute infecions,* as enteric fever, diphtheria, and influenza, are not as a rule due to he pneumococcus, but to the specific organism of the primary disease n association with secondary invading bacteria—Streptococcus pyogenes, taphylococcus, or the colon bacillus.

4. Varieties due to Differences in the Determining Causes.—a) *Contusion Pneumonia.*—Contusion of the chest, or violent bodily hock without direct injury to the lung, may be followed in the course of a ay or two by the onset of a well-characterized croupous pneumonia. (b) *ostoperative Pneumonia.*—The cases probably do not all belong to the ame group. True croupous pneumonia is much less common than bronchoneumonia, which may be diffuse or pseudolobar. Croupous pneumonia ay occur after operations of various kinds, irrespective of the anæsthetic mployed. Bronchopneumonia is common after operations upon the mouth nd throat. (c) *Anæsthesia Pneumonia.*—This variety is almost always ronchopneumonia. In many instances the lesions are so massed as to onstitute a pseudolobar pneumonia. The symptoms develop in the ourse of the first or second day after the operation, much more commonly hen ether has been administered and the mouth, throat, or abdomen has een operated upon. It is probably an aspiration pneumonia.

Diagnosis.—Direct.—In well-developed cases of primary croupous neumonia the diagnosis is an easy matter and errors are not often made. he mistakes in diagnosis occur mostly in the aberrant and intercurrent orms in which the disease is latent and the symptoms masked, and are the esult of neglect to carefully and systematically examine every patient, nd especially chronic cases, upon the appearance of fresh local or constitutional symptoms, however trifling they may appear. In certain ases the general symptoms are indeterminate but the local phenomena ecisive, in others the physical signs are obscure, but chill, fever, cough, nd sputum are characteristic. In either of these conditions the diagnosis s clear; still more clear is it when both symptoms and signs are present nd well defined.

Differential Diagnosis.—1. *Acute Pneumonic Phthisis* (see p. 803). . *Hemorrhagic Infarct.*—There are circumscribed dulness and bloody putum. The chill of pneumonia does not occur; there may be complete bsence of fever; the blood is less thoroughly admixed with the sputum, nd finally a condition capable of giving rise to embolism may be discovered. 3. *Pulmonary Œdema.*—The sputum is bloody, but it is also thin and frothy, a condition only exceptional in pneumonia. Dulness is not common and when present involves both bases posteriorly and is far less strictly delimited than in pneumonia. Both conditions may be present. Collateral œdema—fluxion œdema—is common in pneumonia an an inflammatory œdema may develop at the borders of the lesion. — *Bronchopneumonia.*—Massed lesions of considerable extent or involving lobe—pseudolobar pneumonias—are misleading. Croupous pneumon generally occurs as an acute process, attacking persons in previous go health, or as an intercurrent specific disease in various chronic affectio whereas bronchopneumonia is mostly an affection secondary to some ac specific fever, as measles or other condition in which the aspiration infectious matter from the mouth or throat takes place. Croupous pn monia is furthermore an acute, short, well-characterized disease, beginn abruptly with a chill and terminating by crisis, while bronchopneumonia comes on gradually or abruptly with temperature rise but without chill, continues indefinitely, and terminates in favorable cases by lysis. In the cases in which the diagnosis is uncertain, sputum is often absent, but, as a rule, to which, however, there are exceptions, when present it is rusty and viscid in croupous pneumonia and mucopurulent in bronchopneumonia. 5. *Pleural Effusion.*—This question of diagnosis is of daily occurrence at the bedside and demands special consideration (see p. 1099). 6. *Meningitis.*—Doubts arise in some cases of apex pneumonia, especially in children. A knowledge that the pulmonary lesions are often masked will remind the practitioner that in every case in which meningeal symptoms are present the lungs and heart are to be particularly examined. 7. *Enteric Fever.*—There are two principal sources of error. Patients suffering with toxic pneumonia or the asthenic form of the disease present septic symptoms identical with those of enteric fever with mixed infection. Clinically, in the absence of a satisfactory anamnesis the diagnosis is often obscure. A positive agglutination with the Widal test is mostly conclusive. Exceptionally the patient may have passed through enteric fever some weeks or months before. To this condition the term *typhoid pneumonia* is frequently applied. This unfortunate term is also used to designate cases of enteric fever in which bronchopneumonia—inhalation pneumonia—has arisen as an intercurrent condition. Much less common are cases of enteric fever which begin with the symptoms and soon develop the signs of pneumonia—*pneumotyphus.* The diagnosis cannot be made with precision until the eruption and splenic tumor appear or a positive result follows the agglutination test.

Prognosis.—Croupous pneumonia, taking all cases together, is an extremely fatal disease. The statistics are unreliable. When the facts in the natural history of this disease are considered it appears probable that the mortality, according to hospital statistics, is too high; according to the impressions of physicians in private practice, too low. Especially misleading are the figures collected to support the efficacy of certain methods of treatment. It is necessary to be explicit. The clinical varieties are to be considered. Apex pneumonia is more liable to be associated with nervous symptoms and grave toxæmia. Double pneumonia is attended with increasing circumscription of the respiratory surface and stress upon the right heart, as well as by a more intense toxæmia. In the migratory form there is increased danger from the prolongation of the active disease. As regards the intensity of the process, the powers of resistance of the individual play an important part. On the one hand robust and previously healthy persons, free from alcoholism, perish in a few days, while individuals of feeble constitution recover from an apparently hopeless attack. The toxic cases are mostly fatal. Nevertheless the better the previous health, the more favorable the outlook. That wholly unknown influence called by the older writers the epidemic constitution is far from being unimportant. The mortality varies in different years in the same locality between 5 and 30 per cent., and without discoverable cause there are alternating series of favorable and unfavorable cases. The mortality in house epidemics and institutions is high. Negroes in the United States show an increased mortality. The death-rate is distinctly higher in the southern than in the northern states of our country. In high altitudes the prognosis is extremely unfavorable. The question of diagnosis has a distinct bearing upon the statistics. The pneumonias of infancy and old age, secondary pneumonias, and terminal pneumonias are very often not recognized. In infants bronchopneumonia is frequently mistaken for croupous pneumonia or the latter for meningitis. In the aged, pneumonia frequently causes death without characteristic or even suggestive symptoms. Insidiously developing intercurrent pneumonias may be wholly overlooked and the fatal issue ascribed to the primary disease. Certainly this is true of terminal pneumonias—a fact which accounts for the discrepancy in the death-rate from pneumonia as reported from the wards and upon the protocols of the post-mortem room. It is easy to overlook pneumonia in a patient dying in the ward of a chronic disease; impossible to do so upon the autopsy table.

Statistics, to be of value, especially to be of value in determining the relative efficacy of different plans of treatment, must be based upon large numbers of cases analyzed with reference to all the factors which influence the result of the attack in individual instances. In hospitals the mortality ranges from 20 to 40 per cent. In the Pennsylvania Hospital, of 943 cases entered as pneumonia in seven years, 198 or 21 per cent. died. In the German Hospital, of 407 cases treated during ten years 108 died, a mortality of 26.5 per cent. The mortality in private practice varies according to different observers from 3 or 4 to 20 per cent. The series of cases in private practice are usually too small to be of statistical value.

Among the circumstances which bear upon the prognosis in individual cases are the following: Under one year the death-rate is much higher than between two and twelve. Adolescents and healthy young adults bear pneumonia well. The death-rate is very low among recruits and young soldiers, picked men living a regular life in well-constructed barracks. After sixty, 75 per cent. die; yet remarkable recoveries occur. I have now under observation a lady aged 99 who has twice had well-characterized croupous pneumonia since her eightieth year. Women bear pneumonia comparatively badly. When it occurs during pregnancy there is danger of abortion or premature labor, but the danger is not so great as it was at one time thought to be. In those previously ill with chronic disease, the obese, and especially in those habitually given to drink, pneumonia is especially dangerous. In such cases also astonishing recoveries occasionally take place. The outlook is also grave in gouty persons and those suffering from emphysema. Complications add greatly to the gravity of the cases. Pneumococcus meningitis may be regarded as a fatal disease; endocarditis is usually of the malignant type; septic phenomena, whether due to the toxæmia of the primary infection or to secondary infection, are ominous. A low leucocyte count is unfavorable.

Death is commonly caused by the action of the toxins upon the vasomotor centres with progressive lowering of the blood-pressure. In many cases over-distention of the right heart is at fault. Sudden œdema of the lungs frequently precedes the fatal event.

XVI. CEREBROSPINAL FEVER.

Epidemic Cerebrospinal Meningitis.

Definition.—An acute, infectious, epidemic disease caused by the Diplococcus intracellularis meningitidis, characterized clinically by sudden onset, with headache, vomiting, and painful contraction of the muscles of the back of the neck, irregular fever, profound nervous symptoms, rapid course, and high death-rate; anatomically, by inflammation of the meninges of the brain and cord.

Etiology.—Predisposing Influences.—Climate appears to have little influence as a predisposing factor. Outbreaks are more common and extensive in the winter and spring than in the warm seasons of the year. Densely populated cities and sparsely settled agricultural regions are alike subject to its prevalence. Damp, overcrowded, and unclean habitations favor its spread, and persons living on the ground floor are especially apt to suffer. Individuals of all occupations and professions are liable to this disease. Military life involves a special liability. Among adults the proportion of males attacked is greater than that of females. Among children the number of males and females is about equal.

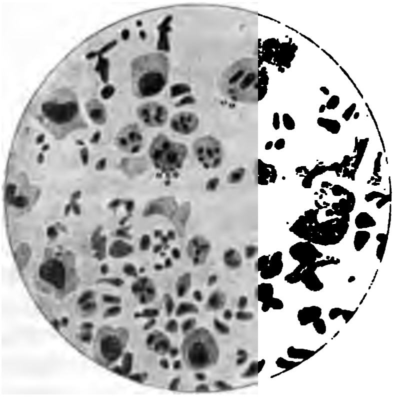


Fig. 248.—Spread of meningococcal exudate showing intracellular meningococci.

Cerebrospinal fever is especially a disease of children and young adults. After 40 it is uncommon, though the diagnosis has been verified post mortem in individuals over 70.

Exciting Cause.—Diplococcus intracellularis meningitidis; meningococcus. This organism is found in the fluid obtained by lumbar puncture and in the meningeal exudate. The cerebrospinal fluid is usually more or less turbid, sometimes very turbid, especially early in the course of the attack. While turbidity of the spinal fluid is of diagnostic importance, its limpidity does not constitute a negative sign, and in either case cultures are necessary to the diagnosis. In the tissues the diplococcus is almost constantly confined to the interior of the polynuclear leucocytes. The diplococci are found only in connection with the lesions of the disease. Mixed infections are not uncommon.

Cerebrospinal fever does not appear to be contagious in the sense in which we use the term in speaking of smallpox, scarlet fever, and typhus. The definite micro-organism which causes it is, in the majority of instances, confined to the meninges of the brain and cord, with little or no opportunity of transmission to other individuals. In cases in which there are lesions in the lungs, ears, and nose, however, infection of neighboring objects or persons may readily take place. Councilman has recently made the following statement: "The presence of sporadic cases is of importance in the occurrence of epidemics. The Diplococcus intracellularis is an organism of feeble vitality: it dies out easily on exposure to drying and light and is incapable of a saprophytic existence. In the absence of intervening infections, it would be impossible for the period of epidemics to be bridged over. Not only this, but there is evidence that this organism can produce other infections and may even live as an inhabitant on the normal mucous membrane."

Second attacks are exceedingly rare. In most cases lasting immunity is established.

Symptoms of the Ordinary Forms.—Cerebrospinal fever presents a great diversity of symptoms in different cases. No other acute disease appears in such various guises. Stillé has well called it a "chameleon-like disorder." The period of incubation is unknown. Prodromes are rare. When present they consist of headache, dragging muscular pains, vertigo, and a sense of fatigue. The onset of the attack is usually abrupt. It is marked by a chill, agonizing headache, nausea, and vomiting. In some cases headache is not a conspicuous symptom. The attack begins with vertigo and the patient acts like a drunken man. Dragging pains in the neck spread along the spine and into the extremities and are followed by motor symptoms which progressively develop. These consist of tetanoid stiffness of the spinal muscles, great pain on attempting to bend the head forward or to turn it from side to side, and awkwardness and difficulty in movements of the extremities. Strabismus, inequality of the pupils, and palsies of the facial muscles are common. In the course of a little time opisthotonos develops, the head is drawn back, the spine curved, the forearms flexed on the arms and the legs on the thighs. Muscular cramps and spasmodic twitchings occur and in young children general convulsions. Hemiplegia has been frequently observed. Paraplegia may also occur. Paralysis may develop during the course of the attack and disappear shortly or persist for some time. The sensory symptoms consist of headache, which may be sharp, lancinating or boring, and is commonly referred to the back of the head; sometimes it is felt as a constricting band; pain in the back of the neck and in the lumbar and epigastric regions and general hyperæsthesia, most marked in the face and neck. Various disturbances of the special senses, as photophobia, intolerance of sounds, ringing in the ears, and vertigo, occur. The psychical disturbances are striking. The patient is restless and distressed. His face is seldom flushed, usually pale, and slightly cyanotic. In children there is great irritability. Delirium occurs early and may be active, even maniacal, or of a busy wandering type. After a time it passes into somnolence or stupor, which may be still attended by more or less restlessness and continual movement on the bed. In the worst cases stupor deepens to coma. The tongue is at first slightly covered with a whitish fur. In conditions of great depression it becomes dry and brown and sordes collect. Taste is lost and the patient refuses food; nevertheless the vomiting persists. Constipation is commonly present throughout the sickness. Toward the end of the attack, however, diarrhœa and involuntary discharges may take place. Slight, exceptionally marked, enlargement of the spleen may be made out. The fever is generally moderate, very irregular, and does not observe a typical course. There are frequent remissions. In some instances fever is slight or absent altogether. On the other hand, the temperature may reach 105° or 106° F. (40.5°–41.1° C.). It may abruptly rise before death. The fever, even when intense, may be of short duration. There is no constant relation between the intensity of the febrile movement and the severity of the other symptoms. Defervescence may take place without improvement in other respects, so that there are cases in which severe nervous symptoms persist for weeks after the temperature has fallen to normal or subnormal ranges. Many of the cases show a temperature range of irregularly remittent type. In the milder cases the temperature is sometimes distinctly intermittent. The subfebrile temperatures are sometimes broken by rapid and transient elevations. The pulse is also irregular. There is no constant correspondence between the pulse and temperature. It may be soft and weak, even slower than in health, and is often intermittent and arrhythmic. Abrupt changes in the force and frequency of the pulse are common. Change in the frequency from 80 to 100 has been observed in the course of a minute. The rhythm of the respiration may be disturbed and Cheyne-Stokes breathing may occur in the graver cases.

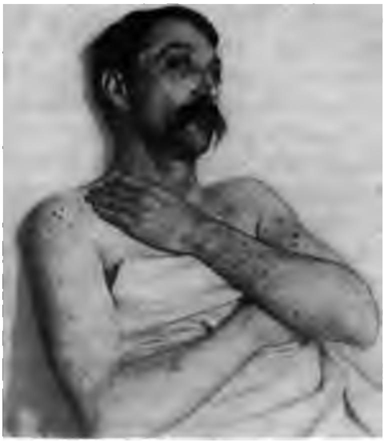


FIG. 249.—Petechial eruption: epidemic cerebrospinal meningitis.- Royer.

Leucocytosis is present throughout the disease, diminishing toward the end of the attack in cases which recover.

Lesions of the skin are common. To their prominence is due the old name of spotted fever. They vary greatly in different epidemics. In many cases they are absent altogether. They are often polymorphous. Herpes is far more common than any other eruption. It usually appears on the lips and nose, but may involve other parts of the face or body and may vary from a crop of a few fine vesicles to an abundant eruption of large vesicles.

A petechial rash resembling flea-bites is frequently observed, and in some instances extensive hemorrhagic areas develop in the skin. These lesions are distributed over the whole surface, but particularly about the knees and elbows. In some cases the rash is abundant and develops with great rapidity. Patches of erythema, dusky mottlings, and rose spots disappearing on pressure, like the rash of enteric fever, have been observed. Among the rarer cutaneous manifestations are urticaria, erythema nodosum, pemphigus, and gangrene. The urine is, as a rule, increased. It may be much increased even with high temperature. The reaction is usually acid. Albumin is frequently present. There is a special form of cerebrospinal fever characterized by symptoms of an acute nephritis and corresponding to the renal form of enteric fever. Glycosuria occasionally occurs, and in malignant cases hæmaturia has been observed. Retention of urine is common in the graver cases. Polyuria is frequent in children, and in some cases has persisted for years after convalescence.

The eye lesions are referable to three causes: First, neuritis, due to the involvement of the nerve in the exudate at the base without extension of the inflammatory process to either the orbit or the eye. This condition may affect the oculomotor and the optic nerve. Second, inflammation from the meninges may extend directly into the eye along the pia-arachnoid of the optic nerve, causing purulent choroido-iritis and in very rare instances suppuration in the orbit. Keratitis may arise in consequence of an extension of the inflammation from the iris and ciliary region. The third cause is neuritis of the fifth nerve, with loss of sensation and keratitis and purulent conjunctivitis. Symptoms relating to the auditory apparatus are common. The auditory nerve is swollen and surrounded by the exudate. Extensive degeneration of the nerve-fibres is frequently found, being most marked in the chronic cases. The abortive form of epidemic cerebrospinal meningitis is the cause of many cases of early acquired deafness. Deafness is frequently due to disease of the labyrinth. Otitis media and mastoid disease occur. The diplococci are found in the pus-cells. Coryza has been frequently observed in the course of the attack. Weigert first advanced the opinion that in meningitis the nose forms the portal of entry for the infectious organisms. It may be, however, that their presence is due to an extension from the brain and not to primary invasion. Epistaxis also occurs. The wasting in severe cases is rapid and extreme. An early, sudden, and great loss of strength is a frequent and prominent condition in this disease.

The symptoms may be divided into those due to the inflammatory lesions of the cerebrospinal organs and those due to a general infection. In the malignant cases both these groups of symptoms are of overwhelming severity. In the mild cases the nervous symptoms are predominant. The foregoing symptoms indicate the nature and severity of the disease in its ordinary form.

Anomalous Forms.—1. MALIGNANT (*Meningitis Cerebrospinalis Epidemica Siderans*).—The patient is struck down without warning and speedily falls into a state of collapse. A violent chill is followed by cyanosis, coldness of the surface, profuse perspiration, intense headache which alternates with drowsiness, and brief delirium followed by unconsciousness. There may be contraction of the neck. Respiration is slow and labored;

the pulse rapid and feeble; the urine scanty and loaded with albu[illegible] Purpuric blotches appear on the surface. Cases of this kind have occur[illegible] in many epidemics and with greatest frequency at the beginning of [illegible] outbreak. They may occur sporadically. Death may ensue in the cou[illegible] of a few hours. 2. ABORTIVE (*Meningitis Cerebrospinalis Epidemica Ab[illegible]tiva*).—The onset of the attack is severe. In the course of a few days [illegible] symptoms subside and convalescence is rapid. 3. MILD (*Meningitis Ce[illegible]brospinalis Epidemica Ambulans*).—Patients complain of headache, stiffn[illegible] in the neck and spine, and malaise. Vomiting occurs. Fever is, as a ru[illegible] absent. Cases of this kind can only be recognized in the light of a prev[illegible]ing epidemic. 4. INTERMITTENT (*Meningitis Cerebrospinalis Epidem[illegible] Intermittens*).—This form is common. Not only the fever, but other symptoms of the disease show extraordinary exacerbations and remissions, which may be repeated at intervals of twenty-four or forty-eight hours. These cases may be due to successive involvement of areas of the meninges or to fresh growths of the organisms. They rarely present the well-marked periodicity of the malarious diseases. 5. CHRONIC (*Meningitis Cerebrospinalis Epidemica Chronica*).—Cases of this form occur in all epidemics. The disease lasts, with numerous complications, remissions, and exacerbations, for several weeks or in some instances for five or six months. Emaciation is extreme. The symptoms may be due to the persistence of conditions left by the acute attack, such as chronic hydrocephalus or abscess of the brain, or general neuritis.

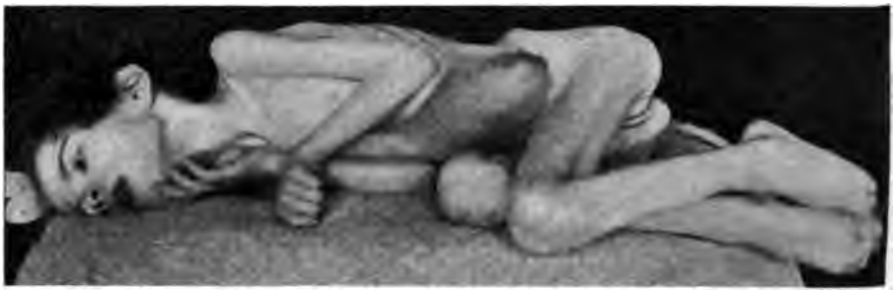


FIG. 250.—Cerebrospinal fever; 53d day of the attack.—Pennsylvania Hospital.

Complications and Sequels.—Among the complications and sequels are pleurisy, endocarditis, and pericarditis. Bronchial catarrh and deglutition pneumonia are very common. Croupous pneumonia has been common in some of the epidemics. It occurs more frequently at the close than at the beginning of an epidemic. Whether cases of pneumonia reported in connection with epidemic meningitis have been cases of true croupous pneumonia or cases of meningococcus pneumonia is uncertain. Arthritis, commonly slight, but in rare instances suppurative, has been noted. The wrist-joints are most commonly involved. Swelling of the parotid glands is an occasional accident of the disease. It may be slight or may run on to suppuration—*parotid bubo*. Intestinal catarrh may occur as a complication. Malarial and enteric fever, and measles, scarlet fever, and cholera have been encountered as intercurrent affections.

The convalescence is irregular and uncertain. After severe cases it is apt to be tardy. Relapses are not uncommon and are often fatal.

Among the more important sequels are prolonged debility and emaciation, palsies and various forms of paralysis, impairment of intelligence n consequence of chronic meningitis and chronic hydrocephalus, especially in children, and more or less complete deafness and loss of vision. General motor weakness and paralysis of individual cranial nerves or of the lower extremities may persist for a long time. They depend on lesions of the brain or spinal cord, or pressure exerted by extensive organized nflammatory exudate, or on peripheral neuritis.

Meningismus.—This term is used to indicate a group of symptoms suggestive of meningitis in cases in which recovery usually takes place rapidly and completely or in which after death the lesions of meningitis are not found. The fluid obtained by lumbar puncture is normal and free rom organisms. It has been observed in various febrile infections, especially in children. The initial symptoms are frequently severe and the early differential diagnosis sometimes impossible.

Diagnosis.—DIRECT DIAGNOSIS. —The recognition of cerebrospinal fever by ordinary clinical methods is a matter of difficulty in sporadic cases and at the beginning of outbreaks. The diagnosis of any form of meningitis is occasionally obscure. Sudden onset, chill, fever, vomiting, delirium, tremor, and painful rigidity of the back of the neck may occur in pneumonia, the malignant form of variola, typhus, and especially in the cerebrospinal form of enteric fever. Kernig's sign is found to be present in 80 to 90 per cent. of the cases of meningitis and only exceptionally present in other diseases. This test is often attended by evident pain on the part of the patient. Brudzinski's sign is common.

If meningitis be present there is usually no great difficulty in recognizng cerebrospinal fever during an epidemic. The ordinary and anomalous forms alike show a symptom-complex that in the course of a little time is distinctive. In all cases when practicable lumbar puncture should be performed. If carried out early in he attack, at the time when the diagnosis is often as important as it is difficult, the result is commonly conclusive.

Lumbar Puncture (Quincke).—The operation is devoid of danger nd can be performed without genral anæsthesia. Freezing of the kin may be dispensed with, as it is as painful as the puncture and auses unnecessary delay. In children excitement may be avoided by a few whiffs of chloroform. Surgical antisepsis is to be strictly observed. Suitable pointed cannulas are sold in the shops. A small aspirator needle may be used. The instrument is introduced into the subarachnoid space between the fourth and fifth lumbar vertebræ. The

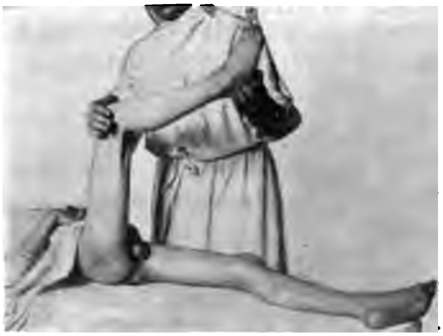


FIG. 251.—Kernig's sign in epidemic cerebrospinal meningitis.—Royer.

point of entrance may be determined by drawing a line connecting the highest points of the crest of the ilium posteriorly. This line passes over the spine of the fourth lumbar vertebra. The point of entrance is about one centimetre below and one centimetre to the right of the intersection of the transverse line and the median line. Some prefer the third lumbar interspace. The patient should lie upon the right side, the spine being strongly bowed, the thighs and knees flexed, and the left shoulder drawn forward. The thumb of the left hand being used as a guide, the needle is thrust with a rotary movement in an upward and inward direction to a depth varying, according to the age of the patient and the thickness of the tissues, from about two and a half centimetres in infants to between four and six centimetres in adults. The fluid runs drop by drop or in a stream, the normal pressure being about 120 mm. of water. In meningitis the pressure may reach 250–300 mm. Normal fluid is clear and limpid, but no conclusions can be reached without careful laboratory investigation, including, in doubtful cases, the inoculation of a guinea-pig.

If the patient has meningitis the fluid withdrawn is, as a rule, but not invariably, more or less cloudy; if cerebrospinal fever, the Diplococcus intracellularis meningitidis—meningococcus—will be found on direct microscopic examination or in cultures. Positive conclusions can only be drawn from positive results. When the result is negative the operation must be repeated.

DIFFERENTIAL DIAGNOSIS.- -1. *Pneumococcus Meningitis.*—This may occur alone or in connection witn croupous pneumonia. The pulmonary lesion may be latent. Symptoms indicating extensive infection of the meninges, of the cord and spinal roots, and extension of the infective process along the cranial nerves are less marked or absent altogether. Contraction of the muscles of the neck may be absent, delirium and coma are present and occur early, and this form of meningitis is fatal, while cases of the epidemic form may recover.

2. *Streptococcus Meningitis.*—This form is secondary to infection elsewhere. Fracture of the skull, especially fracture of the base, local abscess formation, acute endocarditis, erysipelas of the face and scalp, otitis media with extension to the mastoid or the meninges are forms of primary infection. Opisthotonos is neither common nor well developed. The symptoms develop slowly and are often for a time obscure. The association of painful rigidity, intense headache, and vomiting is not conspicuous. Eye symptoms are common.

3. *Tuberculous Meningitis.*—This, perhaps the most familiar form of meningitis, is to be distinguished from cerebrospinal fever by a protracted period of prodromes, more gradual onset, slower course, slow and irregular pulse, great irregularity of the respiration, and the absence of eruption. Antecedent tuberculous disease, failure of health following measles or influenza, tuberculous glands, or a hereditary predisposition to tuberculous infection are found in the history of the patient. In children or during the prevalence of an epidemic of cerebrospinal fever and in cases in which the tuberculous process involves the spinal meninges (Hirsch), the diagnosis is far from easy. The result of spinal puncture is conclusive.

4. *The Cerebrospinal Form of Enteric Fever* (see p. 633).

5. *Scarlet Fever.*—In some instances the sudden onset, high febrile movement, vomiting, convulsions, and stupor suggest cerebrospinal fever as it occurs in children. The presence of the peculiar redness of the palatine half-arches, rapidly followed by general erythematous angina, are important. In the course of twenty-four or thirty-six hours the efflorescence will clear up any uncertainty.

6. *Typhus Fever.*—At one time cerebrospinal fever was confounded with typhus or regarded as a variety of that disease. To Stillé is due the credit of having finally settled every question of doubt concerning the identity of these two diseases in this country. They are in strong contrast in respect of their causes, symptoms, course, lesions, and sequels.

7. *Hysteria.*—Cases of cerebrospinal fever, occurring in nervous females at the close of epidemics or sporadically, have presented a delirium so peculiar and an array of symptoms so little characteristic that they have been looked upon as manifestations of hysteria. This error in diagnosis is no longer possible.

Prognosis.—In individual cases the prognosis can never be made with certainty. The abortive and fulminant cases run the most rapid course. Hirsch has emphasized the fact that certain cases, which at the onset present the symptoms of cerebrospinal fever, recover after an illness of a few hours which terminates in free sweating. The malignant cases, on the other hand, prove fatal in a few hours or two or three days. Moderately severe cases may last one or two weeks or several months. The first week is the time of greatest danger. Symptoms rendering the prognosis unfavorable are intense excitement, early depression, persistent vomiting, irregular respiration, and convulsions alternating with coma. The average mortality is about 40 per cent. It varies in different epidemics from 20 to 75 per cent.

Relapses are not infrequent and are often fatal.

XVII. ERYSIPELAS.

Definition.—An acute, infectious, endemic affection caused by the Streptococcus erysipelatis and characterized by fever, a peculiar circumscribed inflammation of the skin, and ready transmissibility.

Etiology.—PREDISPOSING INFLUENCES.—Erysipelas is a widely prevalent disease which occurs in every climate and to which all races are liable. It is endemic at all seasons of the year and may prevail in local epidemics at any time if the conditions are favorable to its spread. Such epidemics are more common and extensive in the spring. Erysipelas belongs to the group of wound infections and spreads chiefly by accidental inoculation. Neither age nor sex therefore essentially predisposes to the disease. Incidentally certain conditions of the individual and his surroundings render him especially liable. The integuments afford less complete protection against infection at the extremes of life than at other periods. During the first two weeks, the infant is very liable to erysipelas, which most commonly starts from the umbilicus, though it may appear at any wound or abrasion. Aged persons frequently suffer from chronic diseases of the skin, such as eczema, acne, furunculosis, prurigo, varicose ulcers and fissures where the skin and mucous membranes merge, and hence especially liable to accidental inoculation. But these lesions m occur at any period of life. Wounds and injuries are more common males than in females and for this reason the former suffer from erysipe more frequently than the latter. Those who have recently underg surgical operations and lying-in women are peculiarly liable to infecti Exhausting diseases, conditions of cachexia, chronic nephritis, and al holism are important predisposing factors. Among local conditions fi overcrowding, defective ventilation, and deficient sunlight are most portant. Unsanitary apartments and buildings frequently become t abiding place of the poison. The appearance of a single case of erysipelas in a surgical ward or lying-in hospital is an imperative reason, not only for immediate disinfection, but also for abandoning its use for a period. Notwithstanding these precautions the disease occasionally continues to recur in modern institutions of approved construction. The greatly diminished death-rate after surgical operations and among puerperal women at the present time is largely due to the infrequency of erysipelas, and this to the scientific cleanliness of a modern technic. A family or hereditary predisposition is sometimes observed. An apparent personal predisposition is not uncommon. Certain individuals contract the disease several times, at intervals varying from some months to a year or more.

Exciting Cause.—The specific cause of the disease is the Streptococcus erysipelatis, or S. pathogenes longus. This organism belongs to the group S. pyogenes. The Streptococcus erysipelatis is thrown off from the inflamed surface throughout the whole course of the disease and during the desquamation. It is capable of an indefinitely prolonged existence. Under ordinary circumstances it is not intensely virulent, but when a number of susceptible persons are crowded together under bad hygienic conditions the results are disastrous. It is extremely tenacious, adhering to the clothing and bedding of the patient and the furniture and walls and floor of the room occupied during his illness. It clings also to the clothing of individuals who come into contact with the patient, to the hands of operators and attendants, and to surgical instruments. By these means it may be and frequently is, in the absence of proper precautions, communicated to persons at a distance, who in their turn become centres of infection. It gains access to the organism in the vast majority of cases by demonstrable wounds or abrasions of the skin, or, less commonly, of the mucous membranes. The most minute lesion of the integument, readily overlooked or already healed when the erysipelatous flush first appears, or, if not healed, concealed by the blush itself, may serve as the point of entrance. Fissures at the angle of the mouth, nose, or eye, a lesion of the lachrymal duct, a crack in the fold behind the ear, a fissured nipple, an abrasion about the genitalia or at the anus, the prick of a needle, the piercing of the lobule for ear-rings, a scratch, in fact any solution of the continuity of the integument whatever, may be the starting point of the disease. In like manner any lesion of the mucous membrane of the upper respiratory tract or of the oropharynx may become the seat of primary infection. The condition of the mucous membrane of the genital tract in the puerperal woman especially invites infection, which is invariably followed under these circumstances by serious results. The relationship of certain forms of puerperal infection and erysipelas is obvious. Chronic affections of the nasal or laryngeal mucous membrane, varicose ulcers and diseases of the skin render those suffering from them liable to repeated attacks of erysipelas. Abrasion of the skin for vaccination, or the use of the hypodermic syringe without proper precautions as to disinfection and cleanliness, may open the way for the erysipelatous infection.

Symptoms.—The incubation varies from a few hours to several days. Its extreme limit is not more than a week. The local and constitutional symptoms of erysipelas of the face and head and of other parts of the body are identical. When the face and head are involved it not infrequently happens that the local injury to the skin by which the infection has taken place cannot be discovered. Such cases are sometimes spoken of as idiopathic—*E. verum*, *E. cryptogeneticum*. When the cutaneous inflammation affects other parts of the body, a lesion of the skin or mucous membrane can almost always be found. In general terms the severity of the constitutional symptoms is in proportion to the extent and intensity of the local inflammation.

Prodromes are as a rule absent. The onset is commonly marked by a rigor or shivering, followed by a rapid rise of temperature to 103°–105° F. (39.5°—40.5° C.). Headache, pains in the back and limbs, nausea and vomiting also frequently occur. In the course of some hours the signs of local inflammation appear. In many of the milder cases the constitutional symptoms are at first slight and the patient becomes aware of the trouble by burning and pricking sensations in the affected skin. The skin is reddened, tense, and glossy. It is swollen and œdematous and the borders of the affected area are abruptly marginate. It is hot and firm to the touch and the patient complains of subjective sensations of burning, tension, and stiffness. It is a peculiarity of the erysipelatous inflammation that, involving a limited area at first, it tends to spread broadly in various directions, advancing by a well-defined border slightly raised above the level of the surrounding skin. This advance is usually from an ear across the

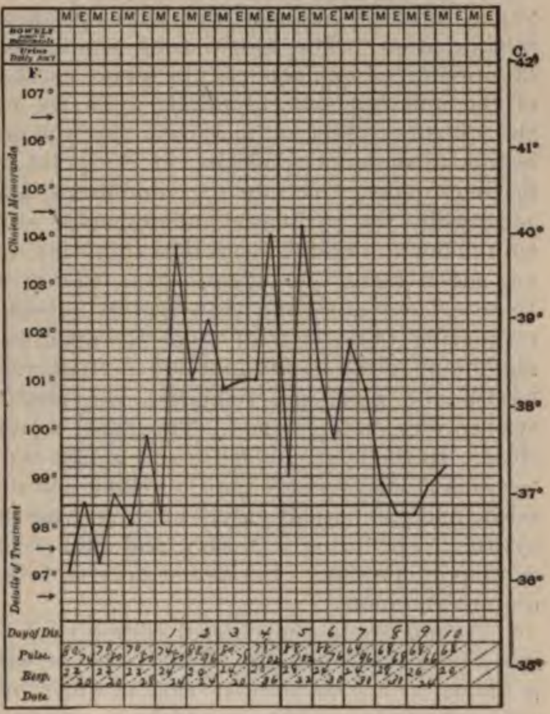


FIG. 252.—Facial erysipelas.

[illegible] temperature falls by [illegible] is marked [illegible] Relapses [illegible]

[illegible] and to the con- [illegible] is abundant [illegible] The [illegible] serum. Pus [illegible] may be ruptured by acci- [illegible] resorption. Thin [illegible] time separate without [illegible] connective tissue is not [illegible] is that in which, in [illegible] swelling and tension result [illegible] The inflammation [illegible] to spread—*E. migrans.* The [illegible] over the neck and chest, sub- [illegible] has traversed the greater [illegible] may extend over many [illegible] or from a complicating pneu- [illegible] symptoms consist in the absence of fever—*E. afebrile;* in hyperpyrexia which is apt to terminate fatally with progressive cardiac weakness and coma; or in great constitutional depression from the outset, a condition to which cachectic and aged persons and those given to excesses in alcohol are especially liable. Erysipelas of the newborn, starting at the navel, shows an abruptly marginate area of redness and induration, which may be superficial but commonly involves the deeper tissues. It extends rapidly and may invade the greater part of the trunk. Suppuration and gangrene sometimes occur. The prognosis is ominous.

Complications and Sequels.—The visceral complications are due to general septic infection. Purulent meningitis may occur in erysipelas of the face and head. When the inflammation involves the mucous membrane of the throat or invades the neck, acute œdematous laryngitis may occur and prove rapidly fatal. Croupous pneumonia is sometimes encountered. Bronchopneumonia is a common late complication in fatal cases. Otitis media and acute nephritis are occasional complications. Septic inflammation of one or more joints may occur. Malignant endocarditis, pericarditis, and pleurisy are important complications. Among the sequels are areas of cutaneous hyperæsthesia or anæsthesia and persistent neuralgias. Repeated attacks of erysipelas have in rare instances been followed by thickening and induration of the skin. The hair falls out after erysipelas of the scalp, sometimes after severe attacks involving distant parts of the body, as in the other acute infections.

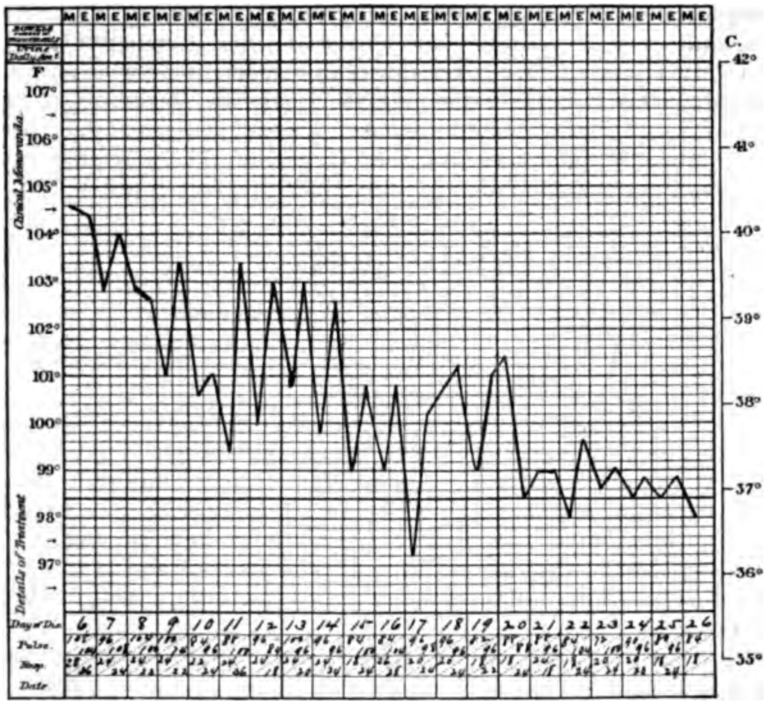


FIG. 253.—Erysipelas ambulans. Woman aged 50. Recovery.

Diagnosis.—The DIRECT DIAGNOSIS of erysipelas rarely presents difficulty. It rests upon the sudden onset, the chill or shivering, fever, and other constitutional symptoms, and the peculiar character of the inflammation of the skin, in which rapid advance from an infected centre, elevation above the level of the surrounding skin, distinct margination, and the tendency to resolution in the region first involved while evolution is taking place at the border, are characteristic.

The DIFFERENTIAL DIAGNOSIS between erysipelas and other forms of dermatitis can be easily made. The main fact is that erysipelas is an acute infectious process attended with fever which begins abruptly and ends by crisis.

Prognosis.—Previously sound individuals except at the extremes of life usually recover. In the new-born erysipelas is commonly fatal, in the aged very often so. In broken down and cachectic persons and drunkards the prognosis is unfavorable. Death is usually the result of intense general infection or grave complications. Erysipelas is not only less frequent, but it is also much less fatal than formerly. The mortality is about five per cent. It is higher in hospitals than in private practice.

XVIII. SEPSIS.

Septicæmia; Pyæmia; Septicopyæmia.

Definition.—A disease produced by the general invasion and growth in the body of pyogenic micro-organisms.

Pyæmia is the term used to designate the condition in which pus collections occur in various parts of the body in consequence of the lodgement of infected emboli; *septicæmia*—*bacteræmia*—that condition in which purulent collections are absent, but with or in the absence of a local infection there is invasion of the blood and tissues by bacteria together with the signs of profound disorder of the entire organism, and *septicopyæmia*, the condition in which bacteræmia and pyæmia are present at the same time. *Sapræmia* is the condition caused by the absorption into the blood of septic or putrid products. The general term *septic infection* or *sepsis* is more convenient and more in accordance with the facts. Some other definitions are necessary. *Infection* is the term used to designate the pathological processes caused by the implantation and growth of pathogenic micro-organisms, most of which are specific. *Toxæmia* is used to denote, (a) the presence of soluble toxic substances or toxins in the blood, mostly elaborated by pathogenic micro-organisms in their growth and multiplication, and (b) the morbid processes which those poisons produce.

Septicæmia and toxæmia are sometimes associated, as in enteric fever, in which the pathogenic organism—Bacillus typhosus—may be grown in cultures from the blood, while the symptoms of the disease are those of a continuous intoxication; sometimes separate, as in tetanus and diphtheria, in which the infection is local and the pathogenic organisms develop in circumscribed areas, while their poisonous products produce characteristic constitutional effects.

Etiology.—PREDISPOSING INFLUENCES.—The predisposition to septic infection is general. It occurs everywhere, at all periods of life, and under the most varying circumstances. PATHOGENIC AGENTS.—The pyogenic cocci—streptococci and staphylococci—are the most common. Other organisms capable of causing sepsis are Micrococcus lanceolatus (pneumococcus), gonococcus, Bacillus coli, Bacillus typhosus, Bacillus proteus, Bacillus pyocyaneus, and Bacillus influenzæ.

Symptomatology.—The symptom-complex is that of a severe constitutional disease. In many of the cases the general symptoms are simply those of any intense infective process, without, however, definite signs of visceral or other localization. In another large group of cases to the foregoing symptoms are added those of an inflammatory implication of the endocardium or pericardium with which the evidences of myocardial changes are shortly associated. Again, the evidences of inflammation of the bones and joints dominate the situation. In a very extensive group of cases the tegumentary structures are especially involved in septic inflammatory processes, such as forms of erythema, scarlatiniform eruptions, malignant erysipelas, and acute septic phlegmon. Subcutaneous hemorrhages are common. Finally, we recognize a great group of cases in which inflammatory and suppurative disease of the various viscera, as for example, the lungs, kidney, liver, stomach, and intestines or spleen, give rise to the chief manifestations of the septic process.

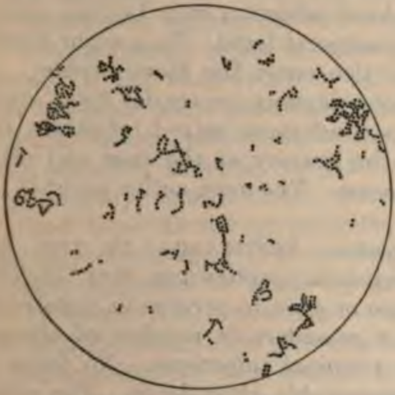


FIG. 254.—Streptococcus pyogenes.

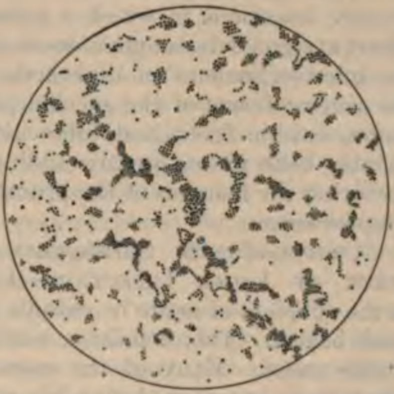


FIG. 255.—Staphylococcus pyogenes aureus.

The onset may be gradual, with chilliness or chills, general malaise, dragging pains in the limbs, and irregular fever. More commonly a decided rigor initiates an active febrile movement. Pallor, faint cyanosis, rapid and feeble pulse, anorexia, constipation alternating with diarrhœa, a tendency to profuse sweating, and a mind strikingly clear and alert are symptoms encountered in well-developed sepsis. Soreness of the bones and muscles, tender joints, great irregularity of temperature, with wide oscillations and a profound feeling of lassitude are also common. A leucocytosis of varying degree, sometimes high, occurs in the majority of cases. In the graver cases even with abscess formation at various points, an increase of leucocytes may be absent.

TOXÆMIA—LOCAL INFECTION WITH THE ABSORPTION OF TOXINS.—Familiar examples of mild and transient forms are the chilliness and fever which attend an attack of angina tonsillaris or a local phlegmonous inflammation. Most important are erysipelas, pneumonia, diphtheria, and tetanus, diseases in which the pathogenic organisms develop locally, while the constitutional symptoms are caused by the absorption of the poisonous products of their growth.

SEPTICÆMIA; BACTERÆMIA—LOCAL INFECTION WITH THE INVASION OF MICRO-ORGANISMS.—Streptococcus and staphylococcus infection is commonly at first local, the toxins undergoing absorption and causing symptoms—toxæmia. The process may be arrested at this point; if not, the cocci enter the blood stream and are carried to all points of the body without causing foci of suppuration. The cause of the infection may frequently be traced, as in puerperal sepsis or injuries of the extremities, along the lymphatic vessels.

The case now becomes more severe; the symptoms more urgent. Specific infections in which a primary local infection may become generalized are gonorrhœa, pneumonia, and puerperal fever. To a slight extent the micro-organisms of diphtheria may also enter the blood stream. In the severer forms of the specific infectious diseases, as scarlet fever, diphtheria, enteric fever, and tuberculosis, secondary or mixed infections frequently take place, greatly adding to the gravity of the case and often obscuring the features of the primary disease. The most active agent is the streptococcus.

CRYPTOGENETIC SEPTICÆMIA—GENERAL SEPTICÆMIA IN THE ABSENCE OF LOCAL INFECTION.—Cryptogenetic septicæmia may develop in the subjects of acute or chronic disease or even in persons in apparently good health. This condition explains a considerable number of obscure febrile cases. Many of the cases are terminal infections. No focus of infection is apparent during life or demonstrable after death. The streptococcus is the common infecting organism, but the staphylococcus, pneumococcus, B. proteus and B. pyocyaneus may be found.

SEPTICOPYÆMIA; PYÆMIA—GENERAL INFECTION WITH SUPPURATIVE FOCI.—The organisms may colonize in various parts of the body and give rise to abscess formation. These suppurative processes are due to the lodgement of infected emboli. The lesions are known as embolic or metastatic abscesses. In infected wounds, septic phlegmon, and osteomyelitis they are frequently encountered in the lungs; in suppurative lesions in the intestines, or elsewhere in the parts tributary to the portal system, metastatic abscesses occur in the liver and may be accompanied with suppurative pylephlebitis. Endocarditis is of common occurrence in septicæmia. The most common organisms in septicopyæmia are streptococci and staphylococci.

TERMINAL INFECTIONS.—Secondary or terminal infection is the cause of death in many acute and chronic diseases. The infection may be local and take the form of visceral disease. The serous membranes are especially liable to these terminal inflammations, and acute pleurisy, pericarditis, peritonitis, endocarditis, or meningitis are final events in many cases of nephritis, arteriosclerosis, cirrhosis of the liver, and cerebral and spinal disease. Not rarely the terminal process is an acute miliary tuberculosis. The enterocolitis so common toward the end of chronic diseases may be classed with the terminal infections. Less frequently the terminal infection is general. This especially occurs in chronic renal and cardiac disease, tuberculosis, leukæmia, and Hodgkin's disease.

Diagnosis.—Direct Diagnosis.—It cannot be made in the beginning of the milder cases. When the symptoms are severe or the illness has lasted for some time, the history of an abortion or confinement, an infected wound involving the skin or mucous membrane, an attack of tonsillitis and especially middle-ear disease, and the occurrence of chills, irregular fever, copious sweating, progressive pallor, and asthenia render the diagnosis probable. Gonorrhœal infection, the reinfection of old scars, and the previous occurrence of disease of the bones, periosteum, or joints are also of diagnostic significance. The signs of an endocarditis are important, especially if the murmurs undergo changes in the course of a short time or are associated with embolic abscesses. Blood cultures may not show the presence of pathogenic micro-organisms.

Cryptogenetic sepsis is more difficult of diagnosis. The symptom-complex is not characteristic. Chills and irregular fever of wide range, with irregular, even prolonged intermissions, copious sweating, profound asthenia, pallor, emaciation, followed after intervals by visceral inflammations, especially affecting the heart, rapid respiration, circumscribed patches of pulmonary consolidation, pleural effusions, enlargement of the liver, localized bone disease or arthritis, and polymorphous cutaneous lesions, especially erythema and hemorrhage, are suggestive. The diagnosis must in many instances be made by exclusion.

Differential Diagnosis. —*Acute Miliary Tuberculosis.* —The general bronchopneumonia affecting the whole of one or both lungs which is characteristic of this disease is not seen in sepsis. If circumscribed tuberculous foci are present, the diagnosis of miliary tuberculosis is probable. Implication of the serous membranes, the pleuræ, pericardium, meninges, or peritoneum increases the resemblance to sepsis and renders the diagnosis more difficult. *Chronic Ulcerative Phthisis.*—In the gravest cases and particularly in the *stadium ultimum,* symptoms occur which are not caused by the Bacillus tuberculosis. These secondary infections are due to streptococci and are mostly terminal. Other organisms are present in the sputum. *Influenza.*—Severe cases of influenza may give rise to difficulties in diagnosis, especially in those cases in which pneumonia, bronchopneumonia, pleurisy, and local pus formation occur. The epidemic prevalence of influenza, the sudden onset with characteristic symptoms, the prominence of catarrhal symptoms in the majority of instances, and the severe neuralgias early in the attack are of diagnostic value. *Enteric Fever* (see p. 634). *Malaria.*—Perhaps no more common error in diagnosis occurs. In phthisis, in internal abscess, in suppurative disease of the liver or hepatic fever from impacted calculus, in malignant endocarditis, the recurrent chills, irregular high temperature, and profuse sweating too often betray the incautious practitioner into a false diagnosis of malaria. The presence of the malarial parasite in the blood and the therapeutic test, or either of them, are conclusive.

Prognosis.—The prognosis is ominous. Very mild cases recover. Many cases make an apparent recovery, only to recur. The fulminant cases are fatal in a short time. Of visceral localizations, septic endocarditis is the most grave. In the absence of local abscess formation recovery may, in rare instances, take place after very grave constitutional symptoms have lasted a long time. Brilliant results sometimes follow the early evacuation of pus and effectual drainage.

XIX. RHEUMATIC FEVER.

Acute Rheumatism; Acute Polyarthritis.

Definition.—An acute febrile disease of undetermined causation, characterized by polyarthritis of fugacious character and a tendency to inflammation of the endocardium and pericardium.

Etiology.—PREDISPOSING INFLUENCES.—Rheumatic fever is a disease of northern and temperate climates. The effect of season is modified by local conditions. Cold and dampness, and especially a combination of these two seasonal conditions, constitute predisposing factors of great importance. Rheumatic fever is rare in the first years of life and after the age of fifty. Adolescents and young adults are especially liable. The two hemidecades of greatest liability are those from 15 to 20 and from 20 to 25. First attacks are very rare after the fortieth year. The liability of the sexes is much modified by age and occupation. If these factors are disregarded males appear to be more liable than females in about the proportion of 2:5 to 1.0. Under twenty the disease is more common in females. The predisposition appears to be hereditary, usually from the maternal side. Those avocations which involve exposure to cold and damp and sudden violent changes of temperature constitute a predisposing influence of great importance. We find a great majority of the cases among coachmen, cooks, bakers, housemaids, sailors, gardeners and outdoor laborers. It is sometimes possible to trace the attack to sudden or prolonged exposure to cold and damp.

EXCITING CAUSE.—The causal relation of the diplococcus described by Poynton and Paine (1900), to which they gave the name *Diplococcus rheumaticus* or *Streptococcus rheumaticus*, is now generally admitted. The phenomena produced by the inoculation of various streptococci may present only a superficial resemblance to rheumatic fever. The hypothesis that the infection is septic rather than specific is thought to find support in the character of the fever, the joint affection, the tendency to implication of serous membranes, the sweating, anæmia, leucocytosis, and the tendency to relapse. On the other hand, pyæmic joints undergo suppuration and pursue a wholly different course, and the pains of sepsis are not influenced by the salicylates. The chemical, metabolic, and nervous hypotheses have merely a historical interest.

Symptoms.—Prodromes are not common. When present they consist of sore throat, slight pains in the joints, and malaise. Not rarely a well-marked attack of angina tonsillaris precedes the joint affection.

The onset is usually abrupt. There is very often the chilliness which attends the development of a mild infective process. Fever of moderate intensity—101°-103° F. (38°-39.5° C.)—and irregular type follows and in the course of twenty-four or thirty-six hours the nature of the attack is established. One or more, usually several, joints are now swollen, reddened, and painful. The pulse is frequent, full, and soft. The tongue is covered with a soft, thick, white coating; appetite is lost; there are thirst, constipation, and scanty, high-colored, and very acid urine. There is frequently abundant, highly acid, and ill-smelling perspiration. The joints are involved successively but without regular order. The large joints, as the knee, ankle, shoulder, are most frequently affected; the smaller joints of the hands and feet somewhat less so. The wrists and ankles are often enlarged and exquisitely tender and painful from the simultaneous implication of many joints and the sheaths of the tendons. The arthritis is curiously fugacious. As one joint is attacked the inflammation subsides in another previously involved. This constitutes a characteristic clinical feature of the disease. The inflammatory exudate is endo- and peri-articular. Suppuration does not occur and ankylosis is very rare, being not a phenomenon of rheumatic fever but a secondary process from want of use and fixation and encountered chiefly in the knee, elbow, or wrist in hysterical girls. Symmetrical bilateral arthropathy is often seen. In severe and protracted cases numbers of joints are implicated and the vertebral articulations do not always escape.

Pain is a constant and conspicuous symptom. It is spontaneous and usually agonizing upon movement and pressure. Frequently the weight of the sheet cannot be borne. Prostration, inability to sleep, and abject helplessness add to the sufferings of the patient. The temperature range does not often exceed 103° F. (39.5° C.) and rarely, except in hyperpyrexia, surpasses 104° F. (40° C.). It does not conform to type and is extremely irregular, with marked remissions and exacerbations, which correspond more closely to the presence or absence of the abundant sweats than to the intensity of the arthritis. The defervescence is by gradual lysis. Recrudescences are common and relapse frequently occurs. Anæmia of high grade develops with great rapidity. A leucocytosis of moderate degree is present. Febrile albuminuria is common. The saliva is sometimes acid or neutral in reaction. There are subacute forms with less intense symptoms which sometimes tend to become chronic. In children rheumatic fever may be attended with very slight or obscure joint affection but with marked and disabling heart lesions. The attack does not confer immunity against subsequent attacks; on the contrary, like croupous pneumonia, diphtheria, and erysipelas, rheumatic fever tends to recur and many persons in the course of time experience several attacks.

Rheumatic Hyperpyrexia; Cerebral Rheumatism.—In rare cases a day or so after the onset, but usually during the course of the second week, a rapid rise of temperature to 108°-110° F. (42.5°-43.5° C.) occurs. Delirium, stupor, a feeble, frequent, and flickering pulse, and extreme prostration accompany the hyperpyrexia. In the course of a few hours the patient usually falls into a comatose state. This form of rheumatic fever is almost always fatal. If the temperature is reduced by cold baths or external cold it rises again. In some instances convulsions precede the coma. Rheumatic hyperpyrexia is extremely rare in this country.

HEART.—Endocarditis, pericarditis, and associated myocardial changes are so frequent that they must be regarded as pathological processes incident to the disease rather than accidental complications. The incidence is variously estimated at 33 to 50 per cent. It is probably higher than these figures indicate. *Endocarditis.*—This is by far the most common of the heart affections and rheumatic fever is by far the most common cause of chronic valvular disease. The liability is greatest in the rheumatic fever of childhood and decreases with age. On the other hand it increases with the number of attacks. The mitral valve system is most frequently involved, the aortic next, and both next, the ratio being about 90-25-20. . Ulcerative endocarditis is of infrequent occurrence in rheumatic fever. *Pericarditis.*—Pericardial inflammation may be associated with endocarditis, as is commonly the case, or occur independently. Well-marked pericardial signs may mask an indistinct endocardial murmur, which very often becomes plainly audible as the friction sounds subside. The exudate may be fibrinous, serofibrinous, or purulent. The last occurs more frequently in childhood. *Myocarditis.*—Changes in the heart muscles—granular and fatty degeneration—are associated in varying degree with the endocarditis and pericarditis, and manifest themselves clinically by enfeebled action and the signs of dilatation.

LUNGS AND PLEURÆ.—Pleurisy may occur and the exudate is often serofibrinous, the effusion not, however, commonly attaining a great volume. Pneumonia is an occasional complication. Acute pulmonary congestion is a grave accident. These conditions are more liable to develop in the cases in which heart lesions are present.

NERVOUS SYSTEM.—Grave nervous symptoms, delirium, stupor, convulsions, and coma arise in the cases of hyperpyrexia and are sometimes manifestations of uræmia. Delirium may be due to the salicylates or other drugs and in cases of idiosyncrasy may result from ordinary therapeutic doses. The mental condition in rheumatic fever is as a rule, even in severe cases, remarkably clear. Chorea, while it does not often appear during the attack of rheumatic fever, follows it in about 15 per cent. of the cases.

CUTANEOUS AFFECTIONS.—These occasionally appear, as in the other acute febrile infections. They are not important and comprise sudamina, miliaria, urticaria, forms of erythema and petechiæ. *Subcutaneous nodules* occasionally develop upon the tendons and fasciæ, about the wrists and hands, and elsewhere. They vary in size up to that of a pea. They grow rapidly and slowly disappear. They are not usually tender to the touch, nor painful. These subcutaneous fibrous nodules are encountered in greater frequency in children than in adults.

Diagnosis.—DIRECT.—The direct diagnosis of rheumatic fever is not usually attended with difficulty. It rests upon the association of the foregoing symptoms, especially the rapid onset, the fugacious polyarthritis, irregular fever, abundant acid sweats, rapidly developing anæmia, and tendency to cardiac complications.

DIFFERENTIAL.—1. *Sepsis; Septicopyæmia.*—Arthritis, irregular fever, and endocarditis are common to both diseases. But in septic conditions the arthritis is fixed, not fugacious, affects a few joints, not many, and tends to suppuration and disorganization instead of *restitutio ad integrum* as in rheumatic fever. The fever in sepsis is as a rule more distinctly intermittent, with higher maxima, and is interrupted by periodical chills sometimes of ague-like regularity. The endocarditis of sepsis is severe, often malignant, with embolic phenomena and retinal hemorrhages. Cases occur in which for a time the differential diagnosis between relatively mild septicopyæmia and severe rheumatic fever cannot be positively made. 2. *Acute Osteomyelitis.*—When the lower end of the femur or the tibia is affected the differential diagnosis may be at first obscure. In the rare cases in which several bones are involved the resemblance to rheumatism is increased. The epiphysis is the seat of the disease rather than the joint, and the local and constitutional symptoms are more severe. 3. *Acute Arthritis of Early Infancy.*—The knee or the hip is usually affected. The affection is mostly monarticular and goes on to early suppuration. It is commonly pyæmic; sometimes gonorrhœal. 4. *Gonorrhœal Arthritis* (see p. 833). 5. *Gout.*—Many cases of podagra are falsely diagnosticated as rheumatism. An arthritis confined to one or two joints, especially the metatarsophalangeal joint of the great toe, the knee, or the ankle, of extremely acute onset and great intensity, with cyanotic redness of the skin which is tense and glossy, exquisitely painful both at rest and on movement, and so tender that the weight of the bedclothes can scarcely be borne, speaks for gout, especially if tophaceous masses are present in the helix of the ear or around the small joints and the patient has reached middle age. 6. *Arthritis Deformans.*—The acute outbreaks of joint inflammation by which certain forms of this disease advance cannot be differentiated from rheumatic fever in the early course. There is fever, together with redness, swelling, tenderness and pain, mostly affecting the small joints. When these symptoms pass, however, there remains the evidence of changes in the joints and periarticular thickening. Fresh attacks of more or less intense arthritis occur and after each one the signs of damage to the joints are more pronounced. 7. *Meningitis.*—When in rheumatic fever the vertebral articulations are involved, there may be severe pain upon movement of the neck, together with painful rigidity of the muscles. As fever is present, the condition, especially in the absence of joint affection of the extremities, may closely simulate meningitis. The absence of severe headache, pupillary derangements, hyperæsthesia, Kernig's sign, and negative results upon examination of the fluid obtained by spinal puncture are of diagnostic importance. 8. *Peliosis Rheumatica.*—The multiple arthritis and fever of Schönlein's disease may suggest rheumatic fever. The simultaneous appearance of purpura, purpura urticans, and erythema exudativum, especially when associated with hemorrhage from mucous surfaces or the evidence of internal bleeding, is decisive. 9. *Hysteria.*—A hysterical arthritic neurosis, usually involving the knee, elbow, or wrist, does not often closely simulate rheumatic fever.

Prognosis.—The course of rheumatism varies from two or three to six weeks or longer and is marked by many remissions and exacerbations, both of the fever and other constitutional symptoms and the arthropathy. Rheumatic fever tends to recovery. The mortality does not exceed 2 or 3 per cent. and death is the result not of the disease in its ordinary manifestations but of the heart affection or hyperpyrexia. It acquires, however, a sinister importance on account of the frequency of the implication of the heart, as the result of which arise initial lesions of the valves and myocardium, especially progressive, constituting the conditions of deformity and impaired function known as chronic valvular disease, irreparably damaging to function and ultimately the cause of death.

XX. YELLOW FEVER.

Definition.—A febrile disease of tropical and subtropical countries due to an unknown infectious principle transmitted by the bite of a variety of mosquito—*Stegomyia fasciata*—and characterized by jaundice, albuminuria, slow pulse, and black vomit.

Etiology.—PREDISPOSING INFLUENCES.—Yellow fever has frequently been transported to the seaboard cities of the United States and toward the end of the eighteenth century prevailed in frightfully disastrous epidemics in Philadelphia and other northern cities. It is a disease of the seaboard and low levels. It rarely shows itself above an altitude of 1000 feet. It occurs chiefly in cities and, during outbreaks, is most prevalent in the low, badly drained, and overcrowded districts occupied by the poor, and in the hot season. The epidemics in the United States have always appeared during the summer and autumn and come to an end upon the occurrence of frost.

THE ACTUAL CAUSE.—The specific germ of yellow fever has not yet been demonstrated. The following are the conclusions of the Yellow Fever Commission of the United States Army:

1. The mosquito—*Stegomyia fasciata*—serves as the intermediate host for the parasite of yellow fever. 2. Yellow fever is transmitted to the non-immune individual by means of the bite of the mosquito that has previously fed on the blood of those sick with this disease. 3. An interval of about twelve days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection. 4. The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack. 5. Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of this disease. 6. An attack of yellow fever, produced by the bite of the mosquito, confers immunity against a subsequent attack of the non-experimental form of this disease. 7. The period of incubation in thirteen cases of experimental yellow fever has varied from forty-one hours to five days and seventeen hours. 8. Yellow fever is not conveyed by fomites, and hence disinfection of clothing, bedding, or merchandise, supposedly contaminated by contact with those sick with this disease, is unnecessary. 9. A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease. 10. The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes and the protection of the sick against the bites of these insects. 11. While

mode of propagation of yellow fever has now been definitely deter- ned, the specific cause of this disease remains to be discovered.

Symptoms.—The period of incubation is three or four days. In 13 perimental cases it varied from 41 hours to 5 days and 17 hours. The rse of the attack may be divided into a stage of invasion and a stage of lapse. These periods are, however, not always well characterized. 1. VASION.—The onset is sudden, without prodromes, and commonly in early morning. It is marked by chilliness, headache, severe pains in the ck and limbs, a rapid rise of temperature to 102°–105° F., and pungent at and dryness of the surface. The tongue is moist and covered with a ck white fur. There is usually some soreness of the throat, together with usea and vomiting, which become more severe upon the second and third y, and constipation. The facies even upon the first day is suggestive, en characteristic. It is flushed and there is slight tumefaction of the elids and lips. The conjunctivæ are injected and icteroid. Later the ense jaundice from which the disease takes its name rapidly invades entire surface. The fever having attained its fastigium during the first y maintains its elevation for two or three days and subsides in favorable ses by lysis. In abortive cases the temperature may fall to normal in enty-four or thirty-six hours. 2. THE REMISSION OR STAGE OF CALM.— is period lasts two or three days. The symptoms ameliorate and the ndition of the patient is in every way more satisfactory. Convales- nce may now set in with rapid improvement, or there may be febrile ction lasting from one to three days and terminating in rapid lysis, or patient may pass into: 3. THE STAGE OF COLLAPSE.—This period is ended with characteristic "black vomit" and other hemorrhages. The miting, in the grave cases, is uncontrollable and copious, being attended th great abdominal pain and exhaustion. The oozing of blood from the cous surfaces and the occurrence of petechiæ usually precede death.

The pulse upon the first day does not usually exceed 110 per minute d, notwithstanding the persistence of a relatively high temperature, comes during the second or third day progressively slower until it may ch, with a temperature of 102°–103° F., a rate as low as 50, 40, or even per minute. This low pulse-rate, with a persistent or even rising febrile vement, is a characteristic and striking feature of the disease. Albu- nuria occurs about the third day of the attack. In the mild cases it is nsient, but in the severe cases it is continuous, abundant, and accom- nied by the ordinary signs of acute nephritis. Suppression may occur d the manifestations of uræmia, convulsions and coma, or these in alter- ion, lead to a rapidly fatal issue. Delirium may be present early in the rse of severe cases. The mental condition is usually, however, one of narkable clearness and alertness.

VARIETIES.—1. *Mild cases*—"walking yellow fever"—present sim- a transient fever and slight jaundice and would not be recognized ept in the light of the prevalent epidemic. These cases are especially ngerous, since they may be the source of contamination of mosquitoes d the subsequent infection of non-immune persons with the fever in its erer forms. 2. *Average cases with high fever* and the characteristic tures of the infection—jaundice, vomiting, fever, slow pulse, albuminuria, black vomit and other hemorrhages, and prostration. 3. *Malignant.*—The patient is overwhelmed by the infection and death occurs in the course of the second or third day.

Convalescence in favorable cases is rapid and complete, the albuminuria usually passing away in the course of a little time. In severe cases terminating in recovery, the convalescence may be protracted by parotid bubo, suppurative processes elsewhere, or persistent diarrhœa. Second attacks are exceedingly rare.

Diagnosis. — DIRECT DIAGNOSIS. — The symptom-complex in well-developed cases is so characteristic that a positive diagnosis would appear to be a simple matter, especially when a number of cases have occurred in a circumscribed region. Commercial interests and considerations of local policy, have, however, in many instances, interposed insuperable difficulties to the recognition of the early cases—difficulties that have frequently led to wide-spread and disastrous epidemics. In some such instances the disease has been reported as dengue, in others as malarial fever.

DIFFERENTIAL DIAGNOSIS.—*Dengue.*—The facies, jaundice, albuminuria, slow pulse, great severity, and high mortality clearly differentiate yellow fever from dengue. The difficulties relate to the initial cases, which may be mild, and the fact that the two diseases may coexist in the same locality. Every suspect should be at once isolated in a screened hospital. *Malaria.*—The differential diagnosis concerns the estivo-autumnal variety which especially prevails in the regions and at the season of the year in which outbreaks of yellow fever are liable to occur. The facies, early jaundice, early albuminuria, slight enlargement of the spleen, hemorrhages, especially black vomit, and the absence of the blood parasite justify the diagnosis of yellow fever. In estivo-autumnal fever the facies is not characteristic, jaundice and albuminuria are later, the splenic tumor is more marked, black vomit and bleeding gums are wholly exceptional. In hemorrhagic malarial fever, hæmaturia, a rare symptom in yellow fever, is most conspicuous.

Prognosis.—The mortality ranges from 10 to 80 per cent. It varies greatly in different epidemics. Among the working classes and hard drinkers it is especially high. Of favorable prognostic significance are mild fever, slight jaundice, a free secretion of urine, and the absence of black vomit. High fever at the onset is ominous. Black vomit, though serious, is not invariably followed by death. Suppression of urine and uræmic symptoms are rarely followed by recovery.

XXI. CHOLERA.

Cholera Asiatica; Cholera Infectiosa.

Definition.—An infectious disease, endemic and epidemic in certain districts of India, and occasionally epidemic in Europe and America, caused by the comma bacillus of Koch and characterized by violent purging, rice-water discharges, and early collapse.

Etiology.—PREDISPOSING INFLUENCES.—Of great importance is exposure in an infected district, but the chief danger lies in the drinking of water contaminated with the fecal discharges of cholera patients. Cholera is endemic upon the delta of the Ganges. Thence it is from time to time transported along the lines of commerce to various parts of the world. Cases on ship-board have reached the New York Quarantine Station on several occasions in the last three decades, but the disease has not gained foothold upon our shores since 1873. It has prevailed extensively in the East in recent years and is still to some extent epidemic in the Philippines. Outbreaks are more common in warm climates—India, Egypt, the Islands of the Malay Archipelago—but the disease has prevailed fiercely in Siberia and Northern Russia and to some extent in Canada. Warm weather favors the spread of cholera, but cold does not arrest it. It is especially a disease of seaport cities and commercial centres, being transported by persons and effects. It journeys in the East with caravans and pilgrims. It is not conveyed by the atmosphere and does not advance at a faster rate than that of ordinary commercial intercourse. In epidemics those who handle the soiled linen of the sick or remove the discharges are especially liable to contract the disease. Physicians and nurses on the contrary are seldom attacked. Students in the study of the germs have contracted "laboratory cholera." The drinking of contaminated water or milk, articles of uncooked food as salads and the like washed with such water, other articles of food accidentally contaminated, are common causes of the disease. The part played by the house-fly in mechanically transporting the pathogenic organism from the stools to articles of food is most important. Every period of life is liable.

Exciting Cause.—The "comma bacillus," discovered by Koch in 1884, is the cause of the disease. This organism is present in all cases of Asiatic cholera and does not occur in other diseases. It is a spirochæta, morphologically appearing as a slightly curved rod, about half the length of the tubercle bacillus but much thicker than that organism, sometimes presenting an S-shaped appearance, and occasionally assuming spiral curves. As other organisms present similar forms, the characteristic growth in cultures becomes important. Comma bacilli are found in the stools from the onset of symptoms and in the rice-water discharges and contents of the intestine after death in almost pure culture. They are rarely present in the vomited material and then only after violent or protracted retching. They are not present in the circulating blood or in the viscera, but are sometimes found in the intestinal glands and submucosa. They have been demonstrated in water tanks and in other drinking water supplies during epidemics. The symptoms are due to a virulent toxin, caused by the bacilli, which acts chiefly upon the vasomotor system. The immunity of certain persons during epidemics, and the fact that virulent cholera bacilli have been

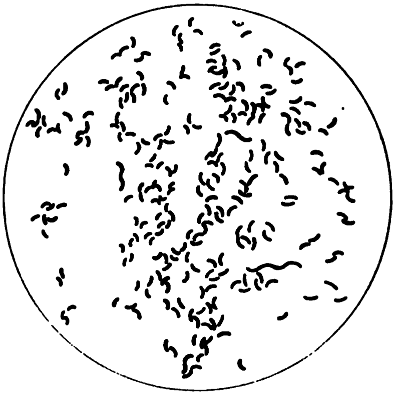


Fig. 256.—Spirillum of Asiatic cholera.

isolated from the stools of healthy individuals raises the question as to natural immunity. Artificial immunity can be established in the cases of laboratory animals and human immunity by the methods of Haffkin. General epidemics in a community are caused by contamination of the water supply and usually arise with great rapidity. Circumscribed outbreaks develop more slowly and the source of the infection cannot always be traced.

Symptoms.—The period of incubation varies from two to five days. The course of the attack may be divided into four stages. Any one of these stages may be absent.

1. PREMONITORY DIARRHŒA.—Looseness of the bowels may begin abruptly or be preceded by colicky pains and vomiting with or without fever. At the time of an epidemic every case of diarrhœa must be regarded as a "suspect," until the true nature of the symptoms is settled by bacteriological examination of the discharges. The stools and any linen that is soiled must be efficiently disinfected. 2. SEROUS DIARRHŒA.—Diarrhœa becomes more urgent, with frequent large liquid stools, which presently assume the rice-water appearance. Or the attack may begin in this way without premonitory symptoms. There are griping pains in the abdomen and much bearing down, with great prostration. The tongue is covered with a thick, whitish fur and there is extreme thirst. In the course of a few hours vomiting occurs. Severe muscular cramps, especially in the legs and feet, add to the sufferings of the patient. Notwithstanding the severity of the symptoms recovery may, in favorable cases, set in at this period. The pains and tenesmus may cease, the rice-water character of the discharges give place to stools that are fecal and bile-stained, the gastric irritability subside, and little by little the ability to retain water and nourishment return. 3. STAGE OF COLLAPSE.—In other cases collapse symptoms rapidly develop. The appearance of the patient is due to the rapid withdrawal of fluid from the tissues. The skin is ashy gray, shrivelled, wrinkled and inelastic, and covered with a clammy perspiration; the features are shrunken, the eyeballs sunk in the sockets, the nose pinched, the cheeks hollow, and the surface cyanotic and mottled. The external temperature is subnormal but the internal registers 103°-104° F. (39.5°-40° C.) or higher. The pulse is feeble, thready, and uncountable. Diarrhœa frequently ceases and there is merely a continuous oozing of rice-water material from the anus. The voice is husky and whispering. The mental condition often remains singularly clear and alert. At the last, coma supervenes. This is the fatal stage of cholera. It lasts from a few hours to a day or two. The thin liquid stools are of a grayish-white color, resembling turbid whey or rice-water. They contain much granular matter and small whitish flakes of mucus. In other cases they are tinged with blood and have the appearance and odor of the washings of meat. They are alkaline in reaction, highly albuminous, and contain sodium chloride in large proportion. Under the microscope epithelial cells and bacteria, often comma bacilli in nearly pure culture, are seen. The urine is greatly diminished or anuria may be present. That which is voided is intensely albuminous. Microscopically it presents the characters of an acute parenchymatous nephritis. Saliva is scanty but the

function of the sweat-glands is maintained. *Cholera Sicca.*—In rare instances the contents of the bowel are retained and collapse terminates in death without diarrhœa. 4. Reaction.—In the cases which survive the stage of collapse the symptoms characteristic of that condition gradually subside. The action of the heart grows stronger, warmth and color return to the skin, which regains its natural turgor, cyanosis disappears and is often replaced by a reddish mottling or erythematous blush, the stomach becomes retentive of water and bland fluids, the colicky pains and violent muscular cramps cease, the stools are much less frequent, and the secretion of urine is re-established. With these signs of improvement the disparity between the external and internal temperatures passes away. The patient now enters upon convalescence which is often protracted, but frequently interrupted by a relapse, which usually proves fatal.

Cholera-typhoid.—The stage of reaction may pass into a septic condition characterized by so-called typhoid symptoms, due to secondary infection. In some instances the predominant features are those of general sepsis, in others pulmonary, and frequently they are uræmic. Feeble, rapid pulse, dry tongue, muttering delirium, and stupor are followed by coma, which terminates in death. As in other epidemic diseases cases of every degree of severity occur.

Complications and Sequels.—The attack in the graver cases is so severe and rapid in its course that complications as such are not common. Important sequels are inflammation of the mucous membranes, as diphtheroid colitis, pleurisy, and pneumonia, and abscess formation, especially parotid bubo. Muscle cramps may persist and subacute gastro-intestinal symptoms are often present for a long time.

Diagnosis.—Direct.—During an epidemic no doubt arises in well-developed cases. The clinical picture is unmistakable. The uncertainty in regard to first cases and suspects is usually quickly dispelled by the course of the attack. Bacteriological examinations are necessary.

Differential.—*Cholera Nostras: Cholera Morbus.*—The symptoms and course of severe cases do not differ from those of Asiatic cholera. The cases are sporadic and occur in hot weather in temperate climates. There is often a history of improper food, chilling, or exposure. Vomiting, diarrhœa with rice-water stools, colic, muscular cramps, suppression of urine, cyanosis, and collapse may terminate fatally in the course of ten or twelve hours. The differential diagnosis can only be made by laboratory methods. *Arsenical and Other Poisoning.*—Vomiting, diarrhœa, and collapse are constant symptoms in acute poisoning by the preparations of arsenic, mercury, and the poisonous fungi. The absence of cholera, the sporadic occurrence of poison cases, the anamnesis, the evidences of the poisonous substance, or the vial or box in which it was contained, are important.

Prognosis.—The mildest cases recover; the severe cases almost invariably die. The mortality ranges in different epidemics between 30 and 80 per cent. In any given case alcoholism, old age, or diminished powers of resistance from other causes, marked cyanosis, a temperature much below normal, and early collapse are of ominous prognostic import.

XXII. BACILLARY DYSENTERY.

Definition.—An intestinal disease, usually acute but sometimes becoming chronic, occurring sporadically and in local epidemics, due to a specific bacillus and characterized by tormina, tenesmus, and frequent discharges of mucus and blood.

Etiology.—Predisposing Influences.—Dysentery is a widely spread disease. It occurs in all parts of the world but is especially common in tropical and subtropical countries. While bacillary dysentery is much more prevalent and disastrous in hot climates, it is also common in temperate climates both as a sporadic and an epidemic affection. Overcrowding and neglect of sanitary requirements both in military and civil life are predisposing influences of great importance. It follows that dysentery constitutes one of the most serious difficulties in warfare, especially in tropical campaigns, and that its epidemic outbreak in overcrowded institutions not infrequently occurs.

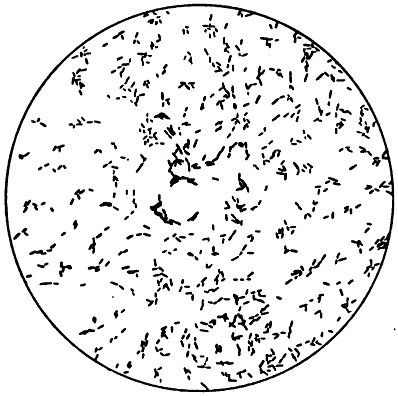


Fig. 257.—Bacillus dysenteriæ (Shiga).

Exciting Cause.—*Bacillus Dysenteriæ.*—Shiga, in 1898, discovered in the stools of these cases a bacillus, having specific characters, which he regarded as the cause of the disease, and to which he gave this name. This organism has been found in the dysentery of the Philippines, Porto Rico, in that occurring in various points in the United States and Europe. It has been demonstrated in the summer diarrhœas of infancy.

There are several strains, as determined by the relative agglutinating power of the immune serum upon the bacilli in pure culture and the action of the bacilli upon various sugars, but the lesions produced are the same. Flexner's types are, (1) the Shiga, (2) the Flexner-Harris—the strain prevalent in the United States, and (3) Bacillus Y. The Bacillus dysenteriæ has never been isolated except from the stools or lesions in human beings. The mode of infection has not been demonstrated. The lesions comprise intense hyperæmia of the mucosa of the large intestine, with scattered points of hemorrhage, superficial necrosis over limited or extended surfaces, and enlargement of the solitary follicles. Deep ulceration is not present in the cases that are early fatal. In the most intense cases great thickening of the mucosa and other coats of the colon occurs, together with extensive necrosis and gangrene. The ileum may be involved.

Symptoms.—The period of incubation does not exceed forty-eight hours. The onset is sudden, with abdominal pain and frequent discharges consisting at first of fæces, followed by mucus which soon becomes bloody. There is urgent inclination to go to stool, with twisting abdominal pain and violent rectal tenesmus. The pyrexia, moderate at first, soon rises to 103°-104° F. (39.5°-40° C.). Thirst is intense and there is complete loss of appetite. The pulse is rapid, small, and feeble. In the very severe cases the patient becomes delirious and death occurs in the course of the third or fourth day. In favorable cases the urgency of the intestinal symptoms gradually declines, the temperature falls, and convalescence may be fully established in three or four weeks. There are other cases in which, with subacute symptoms, the cases run a protracted course. Many of our soldiers return from the island possessions with chronic dysentery.

Among the important complications and sequels are malarial infection, subacute septic arthritis, pleurisy, pericarditis, endocarditis, and sepsis. Albuminuria, anæmia, œdema of the legs and feet, and various palsies due to neuritis occur in the protracted cases. In contrast to amœbic dysentery, abscess of the liver is extremely rare.

Diagnosis.—DIRECT.—Laboratory methods are necessary. In nonamœbic dysentery the B. dysenteriæ must be sought in the stools. It is isolated most conveniently from the shreds of mucus. In the acute cases the blood-serum agglutinates the bacillus in the Flexner-Harris strain in dilutions of 1–1000 up to 1–1500; the Shiga strain agglutinates less readily.

DIFFERENTIAL. — Bacillary dysentery is to be distinguished from amœbic dysentery only by the methods of the laboratory.

Prognosis.—The outlook in the sporadic cases in temperate climates is favorable; in local epidemics less so, especially with bad sanitary arrangements. In active campaigns and tropical dysentery the death-rate is high. The dysentery of Japan has a mortality of about 25 per cent. After recovery there is very frequently prolonged ill health with gastrointestinal symptoms and diarrhœa.

XXIII. THE PLAGUE.

Bubonic Plague.

Definition.—An infectious febrile disease of the Orient, caused by Bacillus pestis, and characterized by glandular swellings or buboes, carbuncles, pneumonia, and, in many cases, hemorrhages beneath the skin and from the mucous surfaces.

Etiology.—PREDISPOSING INFLUENCES.—This disease, the great pestilence of Europe and Great Britain for eleven hundred years, practically disappeared towards the close of the seventeenth century. It has not appeared as an epidemic in England since the Great Fire in London in 1666, the year following the Great Plague. Its cessation is due to modern methods of living. It has been said of Europe that when the shirt came in the plague went out. Always smouldering and frequently flaring up in the East, the plague occasionally slipped over into Lower Italy, Egypt, and other countries bordering on the Mediterranean, but not until the outbreak at Hong Kong in 1894 did it again threaten to become a world pest. Since that date it has continued its ravages, especially in India, where during the first six months of 1905 nearly 900,000 persons died of it, the highest mortality for a half year made in the epidemic of eleven years' duration. During the past decade the plague has appeared in Egypt and other parts on the Mediterranean, South Africa, Oporto, Glasgow, New

York, Mexican and South American ports, Australia and New South Wales. Occupation, age, and sex are without influence as predisposing factors. The disease spreads chiefly among the poorer classes. The prevalence is greatest in the hot season, but outbreaks sometimes occur during the coldest weather. Personal and household cleanliness are important. In Bombay few attendants upon the sick were attacked, and not a case occurred among the British soldiers engaged in police duty and disinfection. Only an occasional case occurs among Europeans living in infected regions.

EXCITING CAUSE.—Bacillus pestis was discovered by Kitasato. It appears in the form of short, non-motile rods, with rounded ends, staining readily and more densely at the poles than in the middle, and decolorized by Gram's method. This organism has a characteristic growth in culture. It is present in the blood and lesions of the plague and in the dust of houses in which cases have occurred, and in the earth of the floors and adjacent parts. Dogs and cats and household vermin, as rats, mice, flies, and fleas, suffer from the infection, transmit it to others and to man, and die infected by its germ. Plague bacilli are present in enormous numbers in the hemorrhagic sputum in the pneumonic form of the disease, and may be found in the ordinary bubonic form.

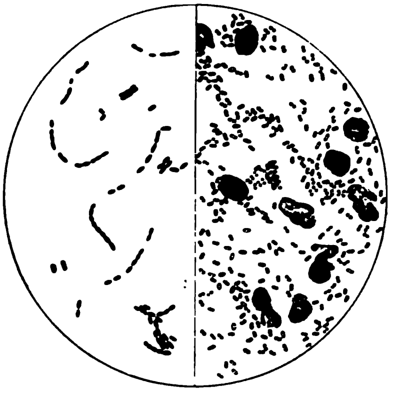


FIG. 258.—Bacillus pestis. *a*, organism from culture; *b*, smear preparation from spleen.

Mode of Transmission.—The plague is not contagious in the ordinary sense. It is dependent on the disease in the rat, and is transmitted from rat to man by the rat flea. Cases commonly occur singly in a house. Multiple cases are usually simultaneous. Rat fleas are carried by personal and other fomites. Insanitary conditions except as to rats have no relation to plague.—Plague Commission, 1908.

Symptoms.—The period of incubation varies from two to five days. The following forms are described: 1. RUDIMENTARY FORM; PESTIS MINOR.—The patient is not in all cases ill enough to go to bed. There is moderate fever, with enlargement and tenderness of the inguinal glands, which sometimes undergo suppuration. These cases constitute a danger to the community by the presence of the bacilli in the discharges. 2. THE ORDINARY FORM; BUBONIC PLAGUE.—The onset is sudden, with a chill which is immediately followed by fever, headache, backache, muscular soreness, great anxiety, and depression of spirits. The temperature progressively rises until the third or fourth day, when there is a more or less marked remission, followed by a further rise coincident with the development of the buboes, and accompanied by signs of septic infection, dry, brown tongue, delirium, stupor and collapse symptoms—secondary fever. Death frequently occurs at this stage. The swelling of the superficial lymph-nodes occurs between the third and fifth days, the inguinal nodes being involved in more than half the cases, less frequently those of the axilla or neck. The adenitis may undergo resolution, suppuration, or, in rare cases, gangrene, with the formation of deep sloughs. Necrosis of the subcutaneous tissue may give rise to more or less extensive carbuncles. Enlargement of the spleen occurs. Petechiæ are common, and the extensive subcutaneous hemorrhages that characterized the disease in certain epidemics gave to it in the Middle Ages the popular name of the Black Death. Hæmoptysis and other hemorrhages from the mucous surfaces have been especially noted in some outbreaks. 3. THE SEPTIC FORM.—The symptoms are from the onset overwhelming, and death occurs in the course of three or four days without the appearance of buboes. Hemorrhages constitute a prominent feature. Metastatic abscesses are frequently found in the viscera. 4. THE PNEUMONIC FORM.—The disease may appear as a primary specific pneumonia, with the usual characters of infectious inflammation of the lungs. The type is bronchopneumonic, the fever high, the respirations rapid, the sputum hemorrhagic and laden with the bacilli. The attack lasts only a few days and almost invariably terminates in death.

Diagnosis.—DIRECT.—Cases imported during the stage of incubation or the first cases in an outbreak may be readily overlooked. In suspected cases all uncertainty can be at once settled by a proper laboratory investigation. The bacteriological examination of the blood, pus from the buboes, the urine, and the sputum yields positive results. If necessary cultures should be made and inoculation experiments upon guinea-pigs. The danger of the importation of the disease at the present time renders an efficient inspection at the quarantine station at every port of entry imperatively necessary, with bacteriological studies in the case of every suspect.

Prognosis.—Bubonic plague is the most fatal of the acute infectious epidemic diseases. In the larval forms recovery is the rule; in the septic and pneumonic forms death; in the ordinary bubonic form a great majority of the cases die. The statistics of the Middle Ages are unreliable, but it is stated that the Black Death of the fourteenth century destroyed one-fourth of the population of Europe.

XXIV. MALTA FEVER.

Definition.—An acute general infectious disease, caused by the Micrococcus melitensis and characterized by irregular fever of long duration and remittent or intermittent type, with periods of apyrexia, by profuse sweating, rheumatoid pains, arthritis, and enlargement of the spleen.

Etiology.—PREDISPOSING INFLUENCES.—The disease prevails widely upon the littoral and islands of the Mediterranean and is known as Mediterranean fever, rock fever, Neapolitan fever, Danubian fever. It has also been encountered in China and India, Manila and in the West Indies, and imported cases have been studied in this country. It is prevalent in summer as an endemic disease, not occasionally in circumscribed epidemics. It is not directly transmissible from the sick to the well. Malta fever is especially a disease of young adults. It has been particularly studied by the surgeons of the British Army stationed at Gibraltar and Malta.

Exciting Cause. — The Micrococcus melitensis was first isolated and studied by Brun in 1887. This organism has not been found in the circulating blood, but is present in the spleen during life and after death. It is pathogenic for monkeys, and cases of accidental infection in laboratory work have been reported. The serum of the patient after the fifth day causes agglutination of cultures of the organism in dilution of 1 to 10 or 1 to 50.

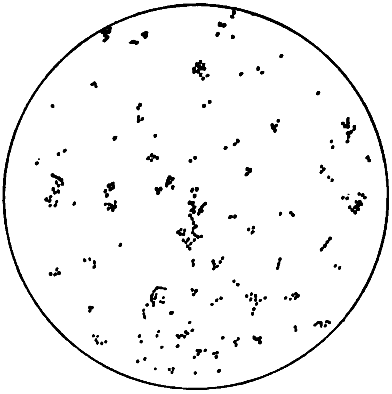


Fig. 259.—Micrococcus melitensis.

The milk supply at Malta is largely derived from goats, and Zammit, in 1905, made the important discovery that the goats of the island are infected with Micrococcus melitensis and isolated this organism from the milk and urine of those animals.

Symptoms.—The period of incubation lasts from six to ten days. The onset is preceded by prodromes not unlike those of enteric fever. The course of the disease is characterized by undulations of fever, 102°–104° F. (39°–40° C.), of distinctly remittent type, lasting as a rule from one to three weeks, and separated by intervals of incomplete or complete apyrexia of two or more days' duration. In rare cases during the pyrexial period the fever conforms to the intermittent type, without, however, manifesting the regular periodicity of the malarial infections. This irregular fever is indefinitely prolonged, lasting from three months, the average time, to six months, and being in some instances prolonged by a series of relapses to two years. Obstinate constipation, progressive anæmia, and debility are common symptoms; the spleen is enlarged and tender; neuralgias, inflammation of the joints, with intra-

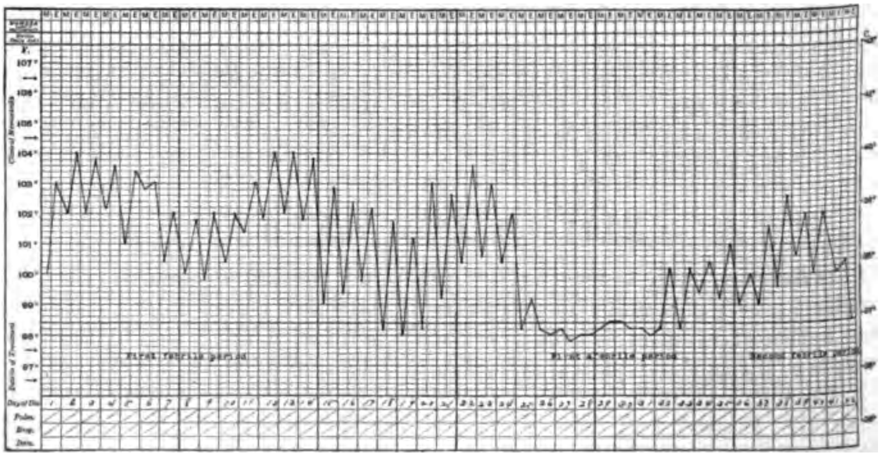


Fig. 260.—Malta or undulant fever.—Jackson. An average case may extend more than twice the duration shown on this chart.

articular effusion, painful inflammatory conditions of certain fibrous structures, and orchitis occur as complications. The wave-like range of the temperature has suggested the name "undulant fever." A malignant type, fatal in the course of a week or ten days, is recognized, and a so-called intermittent type with a daily rise of temperature toward evening has been described.

Diagnosis.—DIRECT.—In districts in which Malta fever is endemic the direct diagnosis is not attended with difficulty. It rests upon the character of the fever and its protracted course with intervals of apyrexia, the tendency to relapse, the headache, lassitude, and joint affection. Finally, the agglutination test is conclusive.

DIFFERENTIAL.—The true nature of the disease may not be apparent in imported cases. *Malaria* reveals itself upon an examination of the blood. The temperature is higher, the periodicity, as a rule, more distinctly defined. Joint pains are not prominent. The judicious use of quinine is curative—therapeutic test. *Enteric fever* differs from Malta fever in its temperature range and duration. If relapses occur they constitute a repetition of the primary attack and like it are attended by subcontinuous temperature, rose spots, and marked intestinal and nervous symptoms. A positive Widal reaction is conclusive. Endemic and epidemic influences are suggestive. *Dengue* is a pandemic disease and spreads with great rapidity. The joint affection is among the earliest and most conspicuous phenomena. The initial febrile paroxysm is of short duration and the recurrent paroxysm is characterized by a polymorphous rash. *Rheumatic fever* bears only a superficial resemblance.

Prognosis.—The mortality is about 2 per cent. Death is due to the debility resulting from indefinitely prolonged fever, the anæmia, or complications. Convalescence is hastened by change of climate.

XXV. BERI-BERI.

Kakke.

Definition.—An endemic and epidemic multiple neuritis of undetermined etiology, widely prevalent in tropical and subtropical countries of the East, and characterized by motor and sensory palsies, atrophy of the muscles, and dropsy.

Etiology.—PREDISPOSING INFLUENCES.—This disease is widely prevalent in China, Japan, the Philippines, and the Malay Archipelago. Local epidemics have been observed in Australia. It is very common in Brazil and occurs in the West Indies. Imported cases are not uncommon in the seaport cities of the United States, especially on the Pacific Coast. In a few instances American fishermen have suffered from it, both upon the Grand Banks and in shore fishing. In its prevalence as determined by season and locality it is not unlike malaria, being most common in the hot and rainy seasons. Absence of sunlight and air, and overcrowding are important favoring conditions, hence beri-beri is frequent in jails and asylums. It is also a common and troublesome disease on shipboard, both in naval and commercial service. It is especially a disease of fishermen, a fact attributed to exposure and wet. In districts where the disease is endemic, and even in epidemics, the native races chiefly suffer. The imported coolies are especially liable to it. Young men from 16 to 30 are most frequently attacked, but no period of life is exempt. Males suffer much more often than females.

EXCITING CAUSE.—Beri-beri has long been regarded as a food disease and attributed to rice which has undergone certain unknown changes, or fish eaten raw or improperly cooked. The Dutch physicians, who have first-hand knowledge of the disease upon an extensive scale, believe that beri-beri is an infectious disease. Recent observations conducted by the Board for the Study of Tropical Diseases in the Philippine Islands have shown that beri-beri is a food disease dependent upon an excess of polished rice—i.e., rice from which the pericarp has been wholly removed—and that (a) a reduction in the rice ration with an increase in the legumen and (b) the substitution of under milled rice—i.e., rice in which some part of the pericarp remains upon the grain—were followed by the disappearance of beri-beri from the native scout organization during the last half of the year 1910. This body is 5,000 strong, and no other cause could be shown to be operative, nor was there any corresponding decrease in the incidence of beri-beri in the general Filipino population or in the native constabulary.

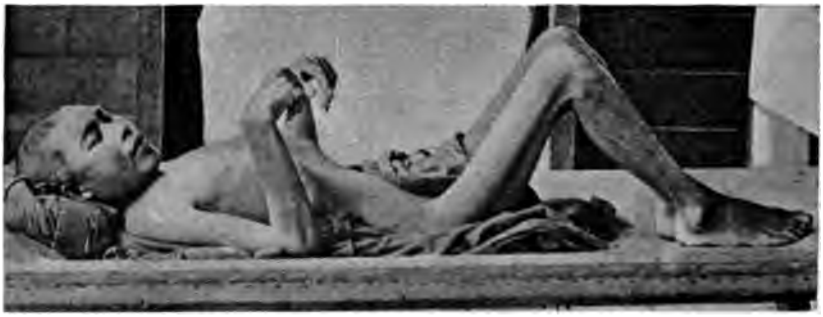


FIG. 261.—Atrophic variety of beri-beri showing muscular atrophy and wrist-drop.—(*Journal of Tropical Medicine*).—*International Clinics*.

Symptoms.—The period of incubation is unknown. Wright thinks it is short. A dry and wet form were once recognized. The following clinical forms are described but they are not sharply differentiated: 1. UNDEVELOPED OR LARVAL FORM.—The onset is marked with catarrhal phenomena which are followed by nervous symptoms, such as modifications of sensation, paræsthesias, hyperæsthesia and pain, and loss of power in the limbs, chiefly in the legs and feet. The muscles are not only weak. they are also tender upon pressure. There is in many cases even in this form œdema of the feet and ankles, but it is slight. Cardiac palpitation, dyspnœa, and abdominal distress may be present. This symptom-complex is of variable duration. After a week or two, or several months, recovery takes place, but there is a tendency to recurrence. 2. ATROPHIC FORM; SO-CALLED DRY FORM.—Early symptoms are the same as in the incomplete form. There is, however, more pain, wasting of the muscles

and progresses rapidly so that in a short time paralytic phenomena
ınounced and the power of movement is greatly impaired. Cardiac
ements and dropsy are not prominent. 3. DROPSICAL, OR SO-CALLED
'ORM.—With similar initial
ɔms œdema occurs early and
ecomes general with effu-
ıto the serous sacs. Muscu-
l neural symptoms are less
ınced than in the atrophic
nd the wasting is not appar-
til after the anasarca sub-
4. ACUTE CARDIAC, OR
ɔɪous FORM.—The symp-
vhich characterize the rudi-
ry form are followed by the
ces of acute cardiac asthe-
ıich may persist for several
or terminate fatally in the
of a day or two.

iagnosis.—DIRECT.—In
ies in which beri-beri is
ic there is little difficulty
diagnosis.

IFFERENTIAL.—Only in an
d imported case could mul-
ıeuritis from other causes,
ɔhol, arsenic, etc., occasion
lty. Here the anamnesis
t important and dropsical
oms most suggestive.

rognosis.—The disease is
ally subacute and chronic
ısts a varying time, meas-
ɔy days or months. The
cardiac forms usually be-
chronic after a number of
ır weeks. Recurrences are common, especially in the wet season.
ıortality varies from 2 or 3 to 50 per cent., and is very high
coolie laborers imported into a beri-beri district.

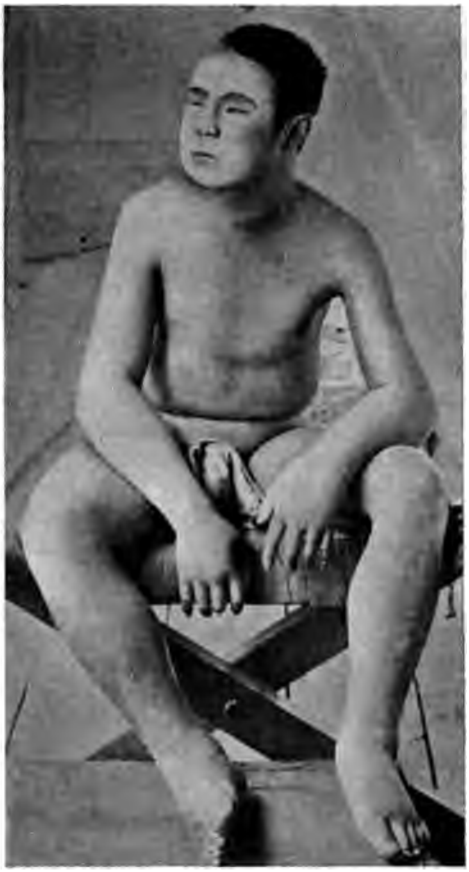


FIG. 262.—Œdematous variety of beri-beri.—(*Journal of Tropical Medicine.*)—*International Clinics.*

XXVI. TETANUS.

Lockjaw.

efinition.—An infectious disease caused by a bacillus found in
earth and in the excrement of animals, especially the horse, char-
ed by tonic spasm of the muscles, with paroxysmal exacerbations.

tiology.—PREDISPOSING INFLUENCES.—Tetanus is essentially a
infection. The view at one time entertained, that the disease may

be cryptogenic and idiopathic, and due to rheumatism or exposure to cold, is no longer accepted. Many cases arise in consequence of insignificant wounds which are overlooked or entirely healed at the beginning of the attack. Internal injuries involving the mucous membrane, which elude observation, are to be considered in this connection. Tetanus occurs in all latitudes but is much more common in tropical than in temperate climates, and among the colored than the white races. This is especially true of puerperal tetanus and tetanus of the new-born. In tropical America and the West Indian Islands tetanus has prevailed as a veritable public scourge.

Since the discovery of the tetanus bacillus and the diffusion of the knowledge of its saprophytic existence the prevalence of the disease has greatly diminished. Tetanus occurs more frequently in the summer and early winter than at other seasons of the year. It has been attributed to exposure to cold and to sleeping upon the damp ground. Aside from the frequency of tetanus among the new-born, age is without influence. The disease is said, however, to be less common after the sixteenth year. Sex is without influence except in so far as occupation renders males more liable than females. Occupation is an important predisposing influence. All those which involve liability to wounds of the extremities, with coincident fouling with earth, manure, or the excrement of animals, especially with that of the horse, or with dust or dirt containing such material, predispose to tetanus. Hence stablemen, teamsters, gardeners, and soldiers, especially cavalrymen, are particularly liable to the disease. In some military campaigns tetanus has contributed largely to the mortality.

The majority of cases occur after lacerated and crushed wounds, especially those involving large nerves. The disease is comparatively infrequent after incised wounds. The extent and severity of the wound has no direct relation to the liability to tetanus. The condition of the wound as regards its tendency to heal is entirely without influence; tetanus may occur when a wound has completely healed and a cicatrix has formed. The disease may follow the most trifling traumatism—the extraction of a tooth, the use of cupping glasses, the sting of an insect, the application of a blister. It is far more frequent after injuries of the extremities than in other parts of the body. The disease has been attributed to the use of the hypodermic syringe. At one time tetanus was common in hospitals and occasionally became epidemic in maternity institutions, a large proportion of the lying-in women and their children dying of this disease.

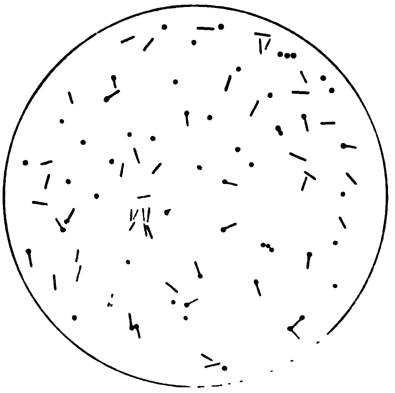


Fig. 263.—Bacillus tetani and free spores.

Exciting Cause.—The tetanus bacillus has been found in the cultivated surface soil of all countries, but not beyond the depth of 30 cm. It has also been found in the dust of streets and in the woodwork of houses and furniture. It is frequently present in the excrement of animals and man. The comparative infrequency of tetanus is in strong contrast with the wide distribution of its cause.

Symptoms.—The period of incubation is extremely variable. In general the onset occurs between the eighth and fourteenth days, rarely later than the fourth week. Prodromes are infrequent. They consist of nausea, tenderness in the wound or scar, increased suppuration or spontaneous reopening of the wound, accompanied by restlessness and loss of sleep.

The attack is occasionally marked by shivering or an actual chill. The characteristic symptoms are continuous tension of the voluntary muscles, and the occurrence at irregular intervals of spasms of varying intensity. The tension and the spasms are commonly relaxed during sleep. In inoculated animals, these symptoms usually commence in the region of the wound and extend to other parts of the body. In man they often first involve the muscles of the neck and suggest an ordinary torticollis from cold. Tension and spasm of the masticatory muscles—*trismus*—soon occur and may be the earliest manifestations of the true condition. There is inability to open the mouth or protrude the tongue—*lockjaw*—and efforts to perform these actions provoke more or less persistent spasm of the facial muscles—*risus sardonicus*. Presently the abdomen is felt to be hard and board-like, as in the early stage of peritonitis, from contraction of the muscles. A sensation of oppression or pain in the precordia, extending to the spine, is frequently experienced, and has been attributed to spasm of the diaphragm. The pain at this time is not usually severe in proportion to the violence of the spasm; later it becomes more intense. In a short time the spasms extend to the voluntary muscles in all parts of the body and affect them with about equal severity. The dorsal and lumbar muscles may contract more violently than their antagonists, giving rise to *opisthotonos*. Forcible contraction of the abdominal muscles, causing *emprosthotonos*, or of the muscles of one side—*pleurothotonos*—are far less common and less marked. On the other hand, transient or persistent rigidity of the trunk and limbs—*orthotonos*—is frequently observed. The fingers are but slightly involved in the general stiffness and spasmodic contraction. The laryngeal muscles may be involved, causing noisy respiration, dyspnœa, or fatal asphyxia. The spasms may occur spontaneously and are apt to come on when the patient awakes from sleep. They are caused by trifling external impressions, as efforts to move, the arrangement of the bedclothing, or an examination of the pulse or heart. Attempt to swallow frequently brings on violent general spasm. Viscid saliva accumulates in the mouth and is swallowed with difficulty. Respiration is disturbed; there may be cyanosis; the expression is greatly changed. The spasms are followed by great exhaustion, with periods of quietude, drowsiness, and sleep. The duration of the paroxysms is variable. The intellect as a rule remains clear throughout the attack. In rare cases delirium has occurred. The pulse is increased in frequency during the paroxysms; in the intervals the pulse and respiration are commonly normal. The temperature is frequently normal until toward the close of the attack; in some instances there is fever from the onset, 104°–105° F. (40°–40.5° C.). A preagonistic rise is usual and often extreme, 106°–110° F. (41.1°–43.5° C.). Abundant perspirations occur during the paroxysms. The urine is decreased in quantity, concentrated, dark, and frequently albuminous. Its toxic coefficient is increased and its injection into animals has caused fatal tetanus. The bowels are constipated. The sphincters are contracted. The administration of a clyster may be difficult and catheterization impossible. These procedures are not only hindered by the state of the muscles but they also cause violent general spasms.

The acute cases usually prove fatal about the third day. The milder cases may continue for two or three weeks or even longer. Recoveries usually take place in the prolonged cases. The disease is most severe and quickly fatal when it develops shortly after the injury. Death occurs in the acute cases, usually during the paroxysm, from asphyxia, œdema of the glottis, or cerebral hemorrhage; less frequently from sudden collapse or coma. In the prolonged cases it may be the result of exhaustion or of inhalation pneumonia. If the patient survives the fourth day the intensity of the symptoms may gradually diminish. Tetanus neonatorum is due to infection at the navel. The disease usually shows itself between the first and fifth days after the separation of the cord. The symptoms are the same as those of ordinary traumatic tetanus. The duration is variable, death commonly occurring about the third or fourth day. Recovery is rare.

The term puerperal tetanus is applied to those cases which develop in the lying-in woman. It frequently occurs as the result of abortion or from neglect of antiseptic precautions in confinement. Tetanus may occur after operations or injuries of the genitalia in non-pregnant women. Tetanus bacilli have been found in the uterus and vaginal discharges.

The Cephalic or Facial Tetanus of Rose originates from a lacerated wound in the region of the facial nerve and is characterized by trismus, difficulty in swallowing, and paralysis of the facial muscles upon the same side.

Diagnosis.—DIRECT.—This rests upon the occurrence of stiffness in the muscles of the neck, trismus, substernal pain extending to the back, followed by risus sardonicus, general tension of the muscles with paroxysms of spasm, and finally upon the bacteriological examination. The previous occurrence of trauma is most important. Courmont has shown that neither spontaneous nor experimental tetanus develops any agglutinating property in the blood.

DIFFERENTIAL.—1. *Torticollis* following exposure to cold—so-called rheumatic torticollis. A history of trauma and the rapid development of the attack speedily settle any doubt. 2. *Trismus* associated with quinsy. Parotitis and local abscesses of the jaws or teeth may suggest tetanus, but a careful examination and the absence of general symptoms render the diagnosis clear. It is important in this connection to note that the rigidity of the masticatory muscles in tetanus is bilateral. 3. *Strychnia Poisoning.*—The spasms are very suggestive but trismus is rarely marked, and in the intervals between the paroxysms there is, in strychnia poisoning, no rigidity. 4. *Cerebrospinal Meningitis.*—Opisthotonos is a common and prominent symptom and in rare cases general spasms may occur, but the high fever and grave mental derangements stand in contrast to the absence of fever at the beginning and the mental clearness in tetanus. 5. *Tetany.*—Spasm of the extremities, the peculiar position of the wrists and hands and the feet and toes, the occurrence of the symptom described by Trousseau and that of Chvostek, together with the conditions under which the disease occurs, usually render the diagnosis a simple matter. 6. *Hydrophobia.*—The history of the case, showing the bite of an animal or man, the long period of incubation, the absence of trismus, the influence of attempts to swallow in producing the spasms, and the great restlessness and jactitation are of diagnostic importance. 7. *Hysteria.*—The paroxysms are occasionally attended by convulsions which suggest tetanus. The absence of antecedent trauma, the neurotic temperament, the interparoxysmal state, the emotional manifestations, the curious sensory disturbances, the existence of hysterogenetic areas render the differential diagnosis between this condition and tetanus easy.

Prognosis.—The worst days are the first four. The aphorism of Hippocrates still holds good: "The patient who survives the fourth day may recover." Traumatic tetanus is less frequent and less fatal in women than in men. The general mortality is about 80 per cent. The later the disease shows itself after the occurrence of the wound of infection the milder is its course, and the more intense the initial symptoms the greater the danger. The mortality is greater in children than in adults. Restriction of the spasms to the muscles of the neck and jaw, and the absence of fever are favorable.

XXVII. HYDROPHOBIA.

Rabies; Lyssa.

Definition.—An acute infectious disease of warm-blooded animals, caused by an unknown specific virus contained in the saliva, characterized by convulsive and paralytic symptoms and communicated by inoculation to man.

The terms "hydrophobia" and "rabies" denote prominent symptoms. The Greek term "lyssa" is frequently used. The distinction between lyssa humana and lyssa animalis is unnecessary.

Etiology.—PREDISPOSING INFLUENCES.—A distinction was formerly made between the hydrophobia of man and that of animals. Experimental medicine has established the fact that the disease in man and animals is the same. Climate is without influence. The disease occurs in all parts of Europe and is common in France, Holland, and England. At one time frequent in Germany, it has in recent years become rare as a result of the strict enforcement of laws regulating the muzzling and care of dogs. It is very common in Russia. In North America it is comparatively nfrequent. The greater number of cases develop in the summer months, a fact which finds ready explanation in the out-door life and lighter clothing at this season. Men and children are affected more frequently than women who are less exposed and whose clothing affords some degree of protection.

Epidemics among animals can often be traced to a single case. The spontaneous occurrence of the disease as the result of cold, heat, thirst, or other such cause has not been established. It is equally contrary to experience that hydrophobia arises spontaneously in man. Inoculation takes place in the vast majority of cases by the bites of animals suffering from the disease. Not every bite of an animal suffering from rabies produces the disease, since the saliva may be wiped off by the clothing. Especially dangerous are bites upon the face, hands, and the uncovered legs and feet.

Rabies occurs most frequently in the dog, wolf, and cat. Nearly all the domestic animals occasionally suffer as the result of inoculation. Rats and, in this country, the skunk are particularly liable. The disease is commonly propagated by the dog, which is on the one hand peculiarly susceptible and on the other hand liable to infect other animals and human beings, the disposition to bite being an especial manifestation of the attack. Next in importance in disseminating the disease is the cat. The propagation of hydrophobia by other animals is comparatively rare. Recent researches show that rabbits, guinea-pigs, and other warm-blooded animals in which hydrophobia was not formerly observed are susceptible.

EXCITING CAUSE.—The nature of the poison is unknown. It is most abundant in the central nervous system and especially in the medulla oblongata. It is present also in the peripheral nerves, though in much smaller amounts.

Symptoms.—The period of incubation in man is irregular. Its average duration is from 20 to 60 days. It may, however, be prolonged to three months and exceptionally to six months.

The period of incubation is influenced by: (a) the susceptibility of the patient—it is shorter in children than in adults; (b) the animal communicating the infection. The incubation is shortest after the bite of the wolf and increases in the following order—the cat, the dog, and other animals. (c) The amount of the virus introduced—puncture wounds and extensive laceration wounds being followed by shorter periods; and (d) the part of the body upon which the inoculation takes place. Bites upon the face and head are followed by shorter periods of incubation, and next in order are bites upon the hands.

STAGE OF PRODROMES.—There is irritation about the scar or the wound, together with pain. In some instances the healed wound reopens. Sensations of numbness may occur. Trembling and fibrillary contraction of the muscles of the affected member have also been observed. Other prodromal symptoms consist of a feeling of depression, aching, and a sensation of pressure in the head. The patient becomes sad, loses interest in his surroundings, and prefers to be alone. He is unwilling to talk about the injury to which his illness is attributed. He is restless, apprehensive, and seeks relief from these symptoms in long solitary excursions. Children become depressed, lose interest in their ordinary play, and have irregular and disturbed sleep. There is increased general sensibility and bright light and noises are extremely distressing. There is laryngeal irritation which may show itself in slight hoarseness and occasional spasmodic cough. There may be difficulty in swallowing. Shivering may occur, which is followed by some elevation of temperature and increased pulse-frequency. Loss of appetite, nausea, and epigastric pain occur. This stage commonly lasts from two to eight days. It may however be much prolonged. In some instances the prodromal symptoms are altogether absent. STAGE OF EXCITEMENT.—There is an intense sensation of dyspnœa, interrupted by sighing and accompanied by a feeling of oppression and precordial distress. Difficulty in swallowing becomes more marked and it is commonly on account of these symptoms that the patient consults a physician. The difficulty in swallowing is characteristic. Attempts to drink or even to swallow the saliva produce violent painful reflex spasm of the muscles of the larynx and throat, which passes away to return with renewed intensity upon further attempts. So distressing is this symptom that the patient avoids taking fluid until compelled by the urgency of thirst. As the case progresses any irritation—a sound, a draught of air, the sight of water or the mere suggestion of it—may bring about the spasm. The superficial and deep reflexes are increased. The pupils are usually slightly dilated. This stage is characterized also by great excitability and restlessness. The patient is tormented by occasional hallucinations, his speech is short and broken, his voice hoarse, and during the convulsive attacks inarticulate sounds may be uttered which have been compared to the barking of a dog. In the maniacal excitement the patient sometimes bites himself. Very rarely, however, does he attempt to injure his attendants. The saliva is much increased. It is frothy and discolored and freely expectorated. At the beginning of the attack there is usually free perspiration. The urine is diminished and of high specific gravity. Albumin and casts are sometimes present. Sugar has been observed and in some cases hæmoglobinuria. Trembling of the tongue and hands is common and in some instances there is persistent tremor of the muscles of the face. Priapism occurs. The temperature is commonly elevated and may reach 100° to 103° F. (37.8°–39.5° C.). In many cases there is a preagonistic rise. The pulse is increased in frequency, irregular and intermittent. The respirations are irregular and shallow in the intervals between the convulsive paroxysms and sometimes assume the Cheyne-Stokes rhythm. The expression is anxious. Hallucinations and sometimes furious maniacal excitement attend the convulsive paroxysms. In the interval the mind is usually clear. Death may occur from asphyxia during the paroxysm. This stage commonly lasts from 2 to 3 days. PARALYTIC STAGE.—The spasms become less frequent and less severe. There gradually develops muscular relaxation. Difficulty in breathing disappears and it becomes possible to swallow. The patient sinks into unconsciousness, the heart's action becomes progressively more feeble, and death occurs in collapse. The paralysis may be limited to certain groups of muscles, as the tongue, the facial muscles, or the muscles of the eye. In other cases hemiplegia or paraplegia may occur. In rats as a rule, and occasionally in rabbits, the stage of excitement does not occur and the onset of the attack is marked by paralytic symptoms—*dumb rabies*. The disease occasionally assumes this form in human beings. It is apt to follow multiple and severe wounds inflicted by the rabid animal, and has been attributed to proportionally large doses of the virus—an opinion supported by experimental investigations.

Diagnosis. — DIRECT. — There is nothing characteristic about the wound. It may heal promptly by first intention or by suppuration and granulation in the ordinary manner. The diagnosis depends upon the history of the bite of an animal suspected or known to be rabid, the occurrence of irritation in the wound or scar, the characteristic spasm of the muscles of deglutition and respiration, the intense reflex excitability, the short duration of the disease and the fatal issue. It is important in doubtful cases to determine by inoculation the presence or absence of the disease in the suspected animal. A small quantity of the substance of the central nervous system of a rabid animal inoculated subdurally will produce in a rabbit the paralytic form of rabies in the course of fifteen or twenty days.

DIFFERENTIAL.—The following diseases may present superficial resemblances to hydrophobia in the human being: 1. *Hysteria.*—The reflex irritability may suggest hydrophobia, but the spasms lack early localization to the muscles of the throat and neck and the intensity of the later general convulsions. The stigmata of hysteria are present and the manifestations are indefinitely prolonged. 2. *Lyssophobia.*—The points of differential diagnosis are those which are to be considered in hysteria. The symptoms arise in hysterical and neurasthenic persons who have been bitten by dogs or other animals and who fear that they have been infected with hydrophobia. The pseudohydrophobic spasms are the result of autosuggestion. Cases of this kind are doubtless among the reported recoveries from hydrophobia. 3. *Tetanus.*—The incubation period is shorter; the convulsions are tonic; trismus is an early and persistent symptom, and the disease follows indifferent wounds and injuries rather than the bites of rabid animals. 4. *Various diseases—epilepsy, poisoning by datura stramonium, sunstroke, cerebral tumor, acute mania*—may present symptoms suggestive of hydrophobia and give rise to difficulties in diagnosis in cases in which there is the history of the bite of an animal suspected of rabies. The symptom-complex, course, and termination in these affections render the diagnosis as a rule a comparatively easy matter. 5. *Landry's paralysis* presents symptoms in some respects suggestive of dumb hydrophobia. The anamnesis is of great importance. Where there is a history of the bite of a rabid animal, or of an animal supposed to be rabid, and the patient has been subjected to the Pasteur treatment, the differential diagnosis between the attenuated form of paralytic rabies, which occasionally develops subsequently, and Landry's paralysis is extremely difficult.

Prognosis.—Hydrophobia occurs in only 15 or 20 per cent. of individuals bitten by rabid animals. The danger is greatest when the wounds are lacerated or deep and especially when they involve the muscles. It is also greater in bites of the face and head than in those of the extremities. The probability that the patient may escape increases with the lapse of time. From the fourth month after the inoculation the danger rapidly decreases. According to Woodhead the mortality of patients bitten by rabid animals varied from 5 to 50 per cent. prior to the introduction of the Pasteur treatment, and the general mortality of those bitten by rabid animals was 16 per cent. Bollinger's statistics indicate that of patients bitten by dogs undoubtedly rabid 47 per cent. suffer and die from hydrophobia. Where the wounds have not been cauterized 83 per cent. of the cases succumb: where they have been cauterized 33 per cent. die. When the symptoms in the human being are well developed the prognosis is absolutely unfavorable, death taking place in a period varying from 12 hours to 4 days. Laveran, Roux, and others have reported cases in which some symptoms of hydrophobia have appeared in individuals undergoing the Pasteur treatment who have ultimately recovered. At the Pasteur Institute at Paris, of 27,719 cases of all kinds treated up to January, 1904, 117 died, a mortality of 0.42 per cent. These figures do not include a small number of cases in which the disease appeared during or within fifteen days after the treatments. Recovery occasionally takes place in animals inoculated in the laboratories.

XXVIII. GLANDERS.

Farcy.

Definition.—An infectious disease of the horse caused by the Bacillus mallei and characterized by the development of nodules in the nose which undergo ulceration—glanders; nodules in the skin—farcy—and lymphangitis. It occurs occasionally in man as the result of accidental inoculation.

Etiology.—PREDISPOSING INFLUENCES.—Among animals the ass is especially liable to glanders. Mules and horses are less so. Other animals may contract the disease. Wild animals in confinement, as in menageries, frequently develop it. Animals contract the affection usually through direct contact and commonly by the respiratory tract. Glanders in man occurs as a rule from exposure to the disease in horses. Stablemen, teamsters, coachmen, veterinary surgeons, and cavalrymen are especially liable. As a rule the infection is derived from chronic cases in the horse the nature of which for a long time remains obscure. Acute glanders in the horse is generally recognized and its further spread controlled by the destruction of the animal. The infection in man usually occurs through some more or less trifling lesion of the skin. Infection by way of the respiratory surfaces is much less frequent. Infection may take place by way of the mucous membranes. The disease in a pregnant animal may be communicated to the fœtus.

EXCITING CAUSE.—The specific germ—Bacillus mallei—morphologically resembles the tubercle bacillus but is shorter and thicker. It is chiefly communicated from the sick to the well by the discharges, including the urine and milk. When the bacilli are present in the circulating blood the course of the disease is very rapid. The patient perishes with acute symptoms.

Cases of glanders in animals have been reported in which recovery has followed the repeated injection of small but increasing doses of mallein: a substance isolated by Kalning and later by Hellman from cultures of the bacilli.

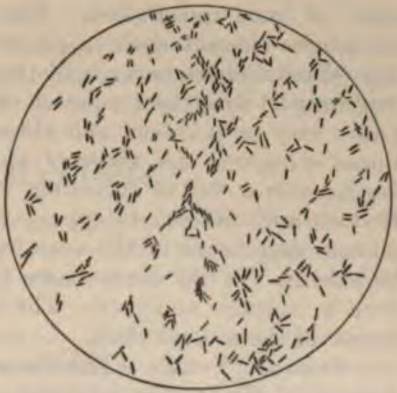


FIG. 264.—Bacillus mallei.

Symptoms.—For purposes of description it is convenient to make a distinction between glanders and farcy. They are due to the same cause and are very often associated. Acute and chronic forms occur. The period of incubation of acute glanders varies from three to five days. Prodromes are not uncommon, and consist of loss of appetite, nausea, and pain in the head and extremities. In acute glanders the *stage of invasion* is marked by general febrile disturbance. The mucous membrane of the nose is swollen and respiration interfered with. There is a scanty, bloody secretion which rapidly becomes more abundant and purulent. The nose is reddened and swollen. An erysipelatous inflammation may extend over the face. The nodules upon the nasal mucous membrane rapidly break down, with the development of extensive ulcers which may go on to necrosis. The lymph-nodes of the neck and the salivary glands are commonly much enlarged. Suppurative lesions of the skin, lymphangitis, and an inflammatory enlargement of the superficial lymph-nodes occur. *Stage of Eruption.*—The eruption is sometimes abundant, especially on the face and extremities, particularly the larger joints. It has been mistaken for variola. Only exceptionally do the pustules show umbilication. In some instances they are confluent. There is troublesome cough with abundant sanguinolent or mucopurulent foul-smelling expectoration. Suppurative arthritis may occur. Hemorrhages into the skin and mucous membranes have been noted. Colliquative sweating, diarrhœa, stupor, convulsions, and coma are followed by death, which usually takes place in the course of the second or third week of the attack. When an acute attack develops in the course of chronic glanders death may occur as early as the second or third day.

ACUTE FARCY.—The infection in man usually takes place from inoculation by way of the skin. There is intense inflammatory reaction with phlegmon formation which rapidly breaks down into an ulcer with irregular, abrupt edges, from which extend painful reddened lines marking the course of the lymph vessels. The corresponding lymphatic glands are swollen, tender, and painful. The swollen lymphatics are known among veterinarians as *farcy-pipes;* the nodular dilatations in their course as *farcy-buds* or *buttons.* If the lesion be situated upon one of the extremities the limb rapidly becomes œdematous. Phlebitis may occur and abscesses form in the subcutaneous connective tissue. In other cases there are no signs of local inoculation. The sickness begins with the constitutional symptoms characteristic of glanders. In the course of from three to seven days small nodules occur in distant parts of the body which rapidly undergo suppuration with the formation of deep ulcers and areas of gangrene. The joints may be involved and abscesses form in the muscles. The constitutional symptoms are those of an acute infection. The attack frequently begins with a chill or shivering. The fever is constant and may be intense. It does not conform to type. Remissions and intermissions occur. The mucous membrane of the nose may not be involved and the eruption may be absent. In the acute cases the bacilli have been found in the urine, both in animals and man. The termination is commonly in death in the course of the second week.

CHRONIC FORMS.—The disease develops insidiously. Fever as a rule is absent. If the infection takes place through a lesion of the skin, similar manifestations to those in acute farcy may occur, developing however more slowly, and only after some time do symptoms of glanders or farcy appear. Symptoms referable to the organs of respiration are prominent. They consist of sensations of fulness in the nose, hoarseness, cough, an abundant nasal secretion, and later dark, dry crusts. Upon examination catarrhal inflammation and ulceration are discovered. The condition is often looked upon as a chronic nasal catarrh. The process may last for months. In some cases recovery has taken place. More commonly the acute form of the disease develops or the patient dies of exhaustion. In chronic farcy the patients experience for some weeks pains in the limbs and joints. At the end of this time subcutaneous nodules develop. These undergo suppuration and form more or less extensive abscesses and ulcers. In some instances they show a tendency to heal; in others healing may take place and the scars after a time break down. The lymph-nodes are not often inflamed and the eruption is rare. Chronic farcy may last, with periods of rest and recrudescence, for two or three years and end in recovery. Most of the cases, however, terminate fatally with acute symptoms.

Diagnosis.—DIRECT.—The diagnosis of glanders or farcy depends upon the occurrence of the foregoing symptom-complex. With a clear history of the case and a knowledge of the occupation of the patient the diagnosis in acute cases is not difficult. In chronic cases no suspicion of the true nature of the disease may be entertained. A positive diagnosis can be reached by bacteriologic methods. Strauss recommends for diagnostic purposes the injection of cultures of the secretion into the peritoneal cavity of a male guinea-pig. After two days, in positive cases, there develop swelling of the testicles and granular inflammation of the tunica vaginalis; later a specific orchitis, which undergoes suppuration. The animal dies in the course of two or three weeks and the visceral lesions of glanders are found. Mallein is frequently used for diagnostic purposes.

DIFFERENTIAL.—In the beginning of the acute cases the symptoms and course of the temperature may suggest enteric fever, and the joint pains rheumatic fever. Later glanders is to be distinguished from erysipelas and pyæmia. The urgency of the symptoms, the well-defined local manifestations, and the course of the attack will usually render the diagnosis a comparatively simple matter. The indolent serpiginous ulcers of chronic farcy may suggest tuberculosis or syphilis.

Prognosis.—The prognosis is in a high degree unfavorable. Recovery takes place very rarely in the acute cases. Chronic glanders usually terminates in death. In chronic farcy, recovery occurs in about 50 per cent. of the cases.

XXIX. ACTINOMYCOSIS.

Definition.—A chronic infectious disease caused by the Streptothrix actinomyces or ray fungus, characterized by granulomatous new formations and multiple abscesses, in the pus of which are found peculiar bodies containing the organisms.

Etiology.—PREDISPOSING INFLUENCES.—This disease occurs in all parts of the world. Those cereals armed with stiff or thorny processes may serve as carriers of the fungus. Barley and rye may be especially named. Cattle are most exposed to the danger of infection at the time of the second dentition and in the autumn and winter. Low and damp localities favor the infection. The fungus penetrates the tissues by way of pre-existent lesions of the mucous membranes or through wounds inflicted by the spears of grain or pointed straws. The usual region of infection both in man and animals is the mouth; less commonly the gastro-intestinal canal, the lungs, or the wounded or abraded skin. The infection may be acquired by drinking water contaminated by the discharges from the mouth of an animal suffering from the disease. There is no reason to believe that infection occurs by means of the milk or flesh of diseased animals. Cases have occurred at all ages, from five to seventy years. Men suffer more frequently than women in the proportion of 5 to 3. Those occupations which involve habitual contact with cattle and their food must be regarded as predisposing causes.

EXCITING CAUSE.—The parasite has been variously classified. Israel and Boström described it as a cladothrix; more recently it has been regarded as belonging to the streptothrix group. It appears in the pus as minute specks, which are yellowish or brownish by reflected light—sulphur granules—and often greenish by transmitted light. These granules vary in diameter from one-half to two millimetres and consist of a central core of filaments among which are cocci in varying numbers surrounded by a mass of radiating filaments, many of which present bulbous or clubbed extremities. The earliest developmental forms consist of smaller granules of a gray color and translucent appearance composed of a thick mass of threads either single or branched, closely interwoven at the centre and possessing the ray-like arrangement. The organism is polymorphous. In animals the club-shaped forms are more common; in man the filamentous. Both threads and clubs are present in cases in which the process is active. Ordinary pyogenic bacteria are present in varying numbers. The ray fungus has been grown upon artificial culture media and actinomycosis has been successfully inoculated both directly and by the artificially grown organism.

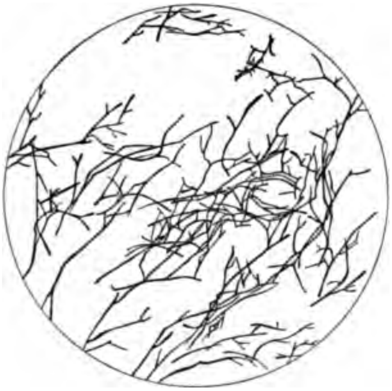


FIG. 265.—Streptothrix actinomyces.

Symptoms.—Actinomycosis is at first a local disease. Its course is generally chronic, and as distant organs become involved it presents the clinical picture of a chronic pyæmia. In very rare cases rapid dissemination may occur by way of the blood-vessels, and the disease run an acute course.

GASTRO-INTESTINAL FORM.—The infection takes place by way of lesions in the mucous membrane of the mouth or throat or through the tonsils. The jaw is very commonly involved in cattle, much less frequently in man. There is swelling of the side of the face, usually involving the lower, rarely the upper jaw. The appearance may suggest sarcoma or a phlegmon. Sinuses form and the characteristic pus is discharged. Burrowing may take place in various directions. Indolent ulcers are common. The duration is variable. Very rarely the fatal issue occurs as the result of secondary infection or embolism in a few weeks. The usual course is chronic and may extend over years. The tongue may be involved either primarily or secondarily. One or more circumscribed nodules form and in the course of a few weeks undergo softening and may be incised. Intestinal actinomycosis commonly involves the region about the cæcum and the appendix, or the sigmoid flexure and the rectum. Metastases are common. Pericæcal abscesses have been reported. The anus may be involved. Actinomyces have been found in the stools. Peritonitis is a common termination, but the disease may run a very chronic course with septic phenomena and cachexia.

Respiratory Form.—Actinomycosis of the neck may directly involve the larynx or may give rise to laryngeal œdema. The lungs may be involved primarily or secondarily. The lesions are less characteristic. In many cases they are merely those of a chronic bronchial catarrh. In others the tissue of the lungs is studded with gray nodules, resembling miliary tubercles and consisting of granulation tissue surrounding masses of the parasitic growth. In other cases the lesions are those of chronic bronchopneumonia with interstitial changes and a tendency to softening and the formation of cavities. As the process advances it involves the pleura, which may become adherent and greatly thickened or undergo suppurative changes leading to circumscribed empyema. Fistulous tracts are formed which open at the inner border of the scapula or elsewhere along the spinal column. Erosion of the vertebræ and necrosis of the ribs and sternum may occur. The clinical phenomena are those of pulmonary tuberculosis or fetid bronchitis. Actinomycotic granules are not always present in the sputa. As the disease advances there are septic symptoms with progressive emaciation and night-sweats. In rare instances the condition may simulate enteric fever. The duration varies from a few weeks to two or three years. Recovery is rare.

Cutaneous Form.—Cutaneous actinomycosis is very rare. It appears in the form of circumscribed tumors of a mottled purplish red and yellow color, varying in diameter from 1 to 3 or 4 centimetres, presenting one or more crater-like ulcerative openings, from which is discharged a clear sticky fluid sometimes containing the characteristic granules. In some instances the ulcerative process, while undergoing cicatrization at the centre, advances at the periphery. The condition is chronic and intractable.

In some few instances other regions have been primarily involved, especially the reproductive organs in the female, and the orbit. Bollinger reported a case of primary disease of the brain. In the other recorded cases the cerebral lesions have been the result of metastasis. The symptoms are those of cerebral tumor or abscess.

Diagnosis.—Direct.—This rests upon the presence of the actinomyces in the pus. Local tumor formation with a tendency to implication of bone and formation of multiple sinuses should arouse suspicion. Visceral actinomycosis gives rise to obscure symptoms. Tumors involving the lower jaw and the neck with multiple fistulæ are very suggestive. In the examination of the sputum some forms of degenerate epithelium and the Leptothrix buccalis may present strong points of resemblance to detached threads of the ray fungus.

Differential.—Actinomycosis of the lungs may resemble forms of chronic bronchitis and tuberculosis. Tuberculosis of the gastro-intestinal tract may give rise to local peritonitis, infiltrations, abscess formations, and fistulæ, which cannot in the absence of the actinomyces be distinguished from similar conditions due to other causes. Cutaneous actinomycosis may resemble lupus and the lesions in the tongue may be mistaken for carcinoma, cysts, or syphilitic gummata.

Mycetoma or Madura Foot. — This curious disease of hot climates presents points of resemblance to actinomycosis. It is a chronic destructive local inflammation of the foot, or more rarely of the hand, resulting in an excessive proliferation of connective tissue. There are two varieties of the disease: the pale or ochroid form which is characterized by yellowish-white or brownish granules in the discharge, and the melanoid form which is characterized by dark brown or black masses of varying size. The disease shows no tendency to formation of visceral deposits. It was early described by Van Dyke Carter as a fungus disease. An organism has been cultivated from the pale variety which has been thought to be closely related but not identical with actinomyces.

XXX. ANTHRAX.

Wool-Sorter's Disease; Malignant Pustule.

Definition. — An acute, infectious, epidemic disease of vertebrate animals, particularly sheep and cattle, caused by the Bacillus anthracis, and occurring sporadically in man as the result of accidental inoculation.

Etiology.—Predisposing Influences.—Anthrax is readily communicated from the domestic animals to man. Those occupations which involve direct or indirect contact with living or dead animals suffering from the disease constitute the chief predisposing cause. Individuals especially liable may be grouped as follows: 1. Farmers, shepherds, drovers, farriers, and veterinary surgeons. 2. Slaughterers and butchers. 3. Tanners, skin dressers, rag sorters, and workers in wool, hair, and horn. 4. Those who come in contact with persons following the foregoing occupations or who live in their neighborhood. 5. Anthrax may be transmitted from one person to another, and is under certain circumstances communicable from the human dead body to those coming into contact with it.

Anthrax is the most widely spread and destructive of the epizootics. All vertebrate animals are susceptible to anthrax, the herbivora being most liable, the omnivora less so, and carnivora only under unusual circumstances.

Exciting Cause —The Bacillus anthracis. This organism usually finds access to animals by way of the gastro-intestinal tract from infected fodder or infected pastures or water. Pasteur held that the earth-worm plays an important part in bringing to the surface and distributing bacilli from the buried carcasses of infected animals. Certain localities thus become permanently infected. The disease is directly inoculable and the infection may take place by the bites or stings of insects. Omnivorous animals, as the hog, dog, cat, and rat, though less susceptible, sometimes contract the disease by feeding upon infected carcasses. The disease does not spread by mere contact or association. The danger of infection ıtly diminished if the carcasses of animals dead of the disease are unopened. Occasionally local outbreaks of anthrax among cattle, and other animals, in regions in which the disease does not prevail uously, have been traced to imported hides, wools, and hair. These, eing thoroughly disinfected, are d, the water being discharged treams and sewers. In some ces the refuse from the manu- e of such articles is utilized for e, and farms and fields thus e infected.

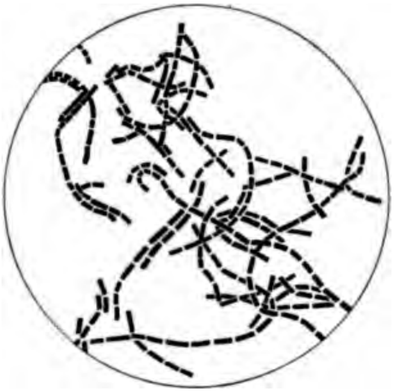


Fig. 266.—Bacillus anthracis.

ymptoms.—The cases may be d, according to the seat of the y lesion by which the infection place, into (a) external or cutaanthrax, and (b) internal or l anthrax, of which there are nary and intestinal forms.

) External or Cutaneous Anthrax. ALIGNANT PUSTULE OR VESICLE. term "malignant pustule" is inappropriate and misleading. The ion is in some cases not malignant and the lesion does not sup- . Anthrax is literally a burning coal. The general condition is as anthracæmia. The term *charbon*—coal—is applied by the to the local lesion of the skin, and *fièvre charbonneuse* to the general disease. The inoculation almost always occurs on some exposed part, as the arm, face, neck, or chest. The period of incubation varies from a few hours to two or three days. The early symptoms are local irritation and itching. A papule forms which rapidly becomes vesicular. There is surrounding redness and considerable brawny swelling. By the third day the vesicle ruptures, leaving a brown base exuding serum. In the course of twenty-four hours a black, dry, depressed eschar forms, around which at a little distance are several small secondary vesicles, sometimes discrete, sometimes confluent. The œdema extends for some distance very tense and deep. The related lymph-nodes are swollen nder. Lesions upon the face or neck cause extraordinary swelling isfigurement. Implication of the larynx and mediastinal glands ise to great difficulty in breathing and swallowing. Pus does not n favorable cases until the eschar begins to separate, usually toward

—Anthrax pustule; early stage.—Royer.

the end of the second week. The severity of the general symptoms has no constant relation to the amount of local disease. Cases with marked local lesions may show but slight constitutional disturbance. Commonly symptoms of general infection rapidly follow the appearance of the papule, or they may be deferred for some days. There is a feeling of illness, chilliness, thirst, vomiting, and restlessness. In many of the cases the symptoms are those of the internal affection. Death may take place in from three to five days. In favorable cases the constitutional symptoms are slight, the eschar suppurates, and the wound heals. 2. MALIGNANT ANTHRAX ŒDEMA.—Swelling appears in the eyelids or elsewhere on the head, hands, and arms. Neither papule nor vesicle develops and there is no characteristic eschar. The œdema may be very extensive and occasionally follows the constitutional symptoms. Extensive areas of gangrene may result, with grave constitutional symptoms. A remarkable characteristic of the external forms of anthrax is the mental condition of the patient. With the gravest symptoms the mind may be perfectly clear and the patient manifest no indications of anxiety or distress up to the time of death.

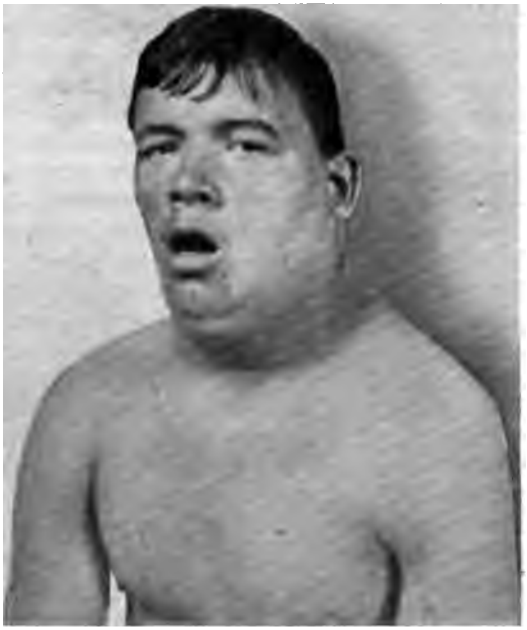


FIG. 268. Anthrax—fifth day; œdema of neck and thorax. Royer.

(b) **Internal or Visceral Anthrax.** — 1. PULMONARY ANTHRAX (*Wool-sorter's Disease; Anthracæmia*). — This form of anthrax occasionally develops in those exposed by their occupations to the inhalation of anthrax spores in dust arising from the products of diseased animals. Wool and hair imported from Russia, Asia, Egypt, and South America appear to have been the cause of the disease in a large proportion of the cases. The symptoms are often indefinite until the approach of death. Prodromes are not common. The onset is usually acute. The patient suddenly feels out of sorts, has shivering, chilliness, uneasiness about the chest and stomach, and sensations of great weakness and weariness. In the course of a day or two, without having expressed sensations of being seriously ill, the patient may fall into a condition of collapse which terminates a few hours later in death. The tongue is moist and coated, thirst is moderate, and there may be weight and uneasiness at the stomach with complete loss of appetite. Vomiting and diarrhœa also occur. Symptoms referable to the respiratory system consist of a feeling of oppression, quickened respiration, cough, not commonly severe, with or without expectoration. The pulse is usually weak and rapid, out of proportion to the severity of the other symptoms, and toward the close of the case becomes irregular and uncountable. The heart sounds are greatly enfeebled. Wandering or active delirium, convulsions, and coma have been observed. The skin is moist. The temperature rises to 102°-103° F. (38.9°-39.5° C.) and may reach 105°-106° F. (40.5°-41.1° C.). It is commonly four or five degrees higher in the rectum than in the axilla. The urine is scanty, dark-colored, and of high specific gravity. Albuminuria is common. 2. INTESTINAL ANTHRAX (*Mycosis Intestinalis*).—This form is rare in man. Infection occurs by way of the stomach and intestines in consequence of eating the flesh or drinking the milk of diseased animals. The symptoms are those of intense poisoning, with gastro-intestinal irritation, and consist of nausea, persistent vomiting, abdominal pain, and diarrhœa. There is great weakness, restlessness, and difficulty in breathing. The pulse is small and rapid, the surface of the skin cold and moist, the face and extremities are slightly cyanotic. The rectal temperature is but slightly above normal. Hemorrhage from the mucous surfaces may occur and is sometimes accompanied by petechiæ and cutaneous abscesses. The spleen is enlarged. The blood is dark and fluid and contains the bacilli. Convulsions and coma are followed by collapse, and death occurs in from two to seven days. Instances have been recorded of local outbreaks in which the symptoms have developed at about the same time in a number of individuals.

The external form of anthrax may be associated with both the pulmonary and intestinal forms of the disease. Eppinger has shown that rag-picker's disease is a local anthrax of the lung and pleura with general infection, and a consideration of the pathological anatomy justifies the conclusion that the intestinal form also begins as a local process to which the constitutional symptoms are secondary.

Diagnosis.—DIRECT.—In both the external and internal forms of anthrax the occupation of the patient is of diagnostic importance. In external anthrax the direct diagnosis rests upon the character of the papule on an uncovered portion of the body, the rapid development of a vesicle, the redness and extensive brawny induration extending along the lymphatics to the neighboring glands. Microscopical examination of the contents of the vesicle may show the presence of anthrax bacilli. Cultures and inoculation experiments in a guinea-pig or white mouse give conclusive results in the course of two days, the animal dying and the internal organs showing anthrax bacilli in enormous numbers. These organisms may not appear in the blood until shortly before death. The appearance of the local lesion upon the third or fourth day is very characteristic. The central depressed eschar, the surrounding vesicles, redness, extensive œdema, with comparatively little pain, are significant.

DIFFERENTIAL.—An ordinary *boil* or *carbuncle*. This rests upon the absence of suppuration and of a moist yellow slough. *Phlegmonous Erysipelas and Cellulitis.*—Anthrax may be distinguished by the absence of pain and of marginal secondary vesicles in the case of slough. *Chancre.*—The differential diagnosis rests principally upon the rapidity of the progress and more serious constitutional symptoms of anthrax. *Glanders.*—There is an absence of the profuse purulent discharge from the nostrils. The direct diagnosis of pulmonary anthrax in the early stage is impossible. Later the gravity of the illness in connection with the symptoms above described, in an individual exposed to infection in his occupation, is highly suggestive. The direct diagnosis can, however, be made in some instances by microscopical examination of the blood. If this be negative inoculation experiments should be performed. The progress of the case in intestinal anthrax is so rapid, and the symptoms so closely resemble those of gastro-intestinal poisoning due to other causes, that a positive diagnosis during life is usually impossible.

Prognosis.—Every case of anthrax may be regarded as a grave illness, but cases of spontaneous recovery are not altogether uncommon. The mild cases are most frequent in children and the intensity of the attack in man is said to correspond to the intensity of the disease in the animal from which the infection is derived. The prognosis is much more favorable in localized external anthrax than in the internal form. Malignant œdema of the face or neck is dangerous to life, partly by its extent and partly through the pressure exerted upon the structures of the neck, especially of the great vessels. The prognosis in anthrax œdema is by far graver than that of malignant pustule. Inhalation anthrax—the *rag-picker's disease*—gives a mortality of 50 per cent. The graver cases with severe fever, rapid prostration, and the evidences of extensive pulmonary inflammation terminate in death. Bell states that "no case demonstrated during life to be intestinal anthrax has ended in recovery."

XXXI. LEPROSY.

Lepra; Elephantiasis Græcorum.

Definition.— A chronic, infectious, endemic disease caused by the Bacillus lepræ, characterized by a disseminated nodular infiltrate in the skin and mucous membranes—tubercular leprosy—or lesions of the nerves—anæsthetic leprosy. In the complete or generalized disease both sets of lesions are present—the mixed form.

Etiology.—PREDISPOSING INFLUENCES.—No race is exempt. Leprosy occurs in all latitudes, in moist and dry climates, alike at the sea level and in mountainous settlements. Congenital leprosy is very rare; in fact its occurrence is doubted. The disease sometimes shows itself in childhood, but the vast majority of the cases develop in early adult life. In some instances it has first appeared in extreme old age. Males are affected in greater proportion than females. The mode of life is not without influence. The poor suffer more frequently than the well-to-do, but the latter do not escape. It has been thought that the habitual or exclusive use of certain articles of diet, as vegetables, salted food, food without salt, fish or pork, predispose to leprosy, either by the ingestion of the bacilli or by rendering the tissues less resistant to their development. Leprosy prevails in mountainous districts, as Kurdistan and Kashmir, where a fish diet is unknown, and among the Brahmins who never taste fish. Furthermore systematic examinations of fish and preparations of fish in countries in which the disease is endemic have failed to reveal the presence of the Bacillus lepræ.

THE EXCITING CAUSE.—The Bacillus lepræ constitutes the specific infecting agent. Nothing is known of the distribution of the Bacillus lepræ outside of the human body. It has not been found in the tanks in which lepers bathe, nor in the soil about the graves of lepers, although in some few instances it has been discovered in the soil of the paths and banks surrounding asylums.

It has been asserted that vaccination may be the means of transmitting leprosy. The danger cannot arise where bovine vaccine is used. The majority of lepers have never been vaccinated, and in countries where leprosy is steadily diminishing vaccination is becoming more general.

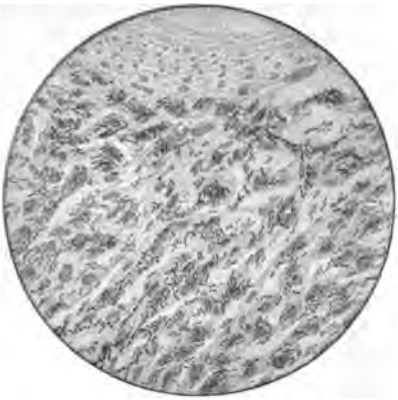


FIG. 269.—Bacillus of leprosy; section of skin.

Until recently a belief in the hereditary transmission of leprosy was generally entertained. The present drift of opinion is against this view. The transmission of the disease takes place under conditions that are not well understood. It has been suggested that the lepra bacillus undergoes some developmental change in an intermediary host. It is probable that the bacilli find access to the body through the skin and mucous membranes, especially the mucous membrane of the nose. When leprosy is carried by immigrants into highly civilized countries it rarely spreads. In countries where the disease has been endemic its diffusion is largely influenced by the degree of association of the lepers with the healthy. When the intercourse with lepers is controlled by legal enactments or a general dread of the disease, its prevalence is circumscribed and limited. The transmission of the disease by the conjugal relation is rare. There are, however, many recorded cases indicating that the communication has taken place after marriage. Physicians in charge of hospitals and asylums for lepers rarely contract the disease. From 9 to 10 per cent. of the helpers in the leper settlement at Molokai have developed the malady. Prolonged exemption does not indicate permanent immunity.

Symptoms.—(a) TUBERCULAR LEPROSY.—The period of incubation varies from a few months to several years. The prodromal symptoms consist of irregular fever, weakness and prostration, loss of appetite, and impaired nutrition. Repeated epistaxis is not uncommon. After a time areas of cutaneous erythema appear. These may be sharply defined and in some instances are anæsthetic. Later they undergo pigmentation. These spots vary in size and some of them may disappear. After a time pea-sized or larger nodules appear which may run together and form large tuberculous masses. These tubercles are at first soft and elastic and slightly tender upon pressure; later they become firmer and are insensitive. They may develop upon any portion of the body. The scalp is, however, usually exempt. They are most common upon the face, the dorsal surfaces of the hands and feet, upon the ankles, wrists, and forearms, and the outer aspect of the thighs.

The lesions progressively involve new areas of skin with the formation of fresh tubercular masses, and as the older ones undergo ulceration areas of cicatrization form. These changes in the skin undergo their most marked development upon the face, producing the characteristic *facies leonina*. The superficial lymphatics generally become enlarged early in the course of the disease. The eyelashes and eyebrows fall out and there is atrophy and loss of hair elsewhere upon the body. The hairy scalp is usually unaffected. The mucous membrane of the upper air passages undergoes infiltration, with the formation of tubercles. These speedily break down, giving rise to painful ulceration. Changes in the voice occur. It becomes harsh or nasal, or more or less complete aphonia may develop.

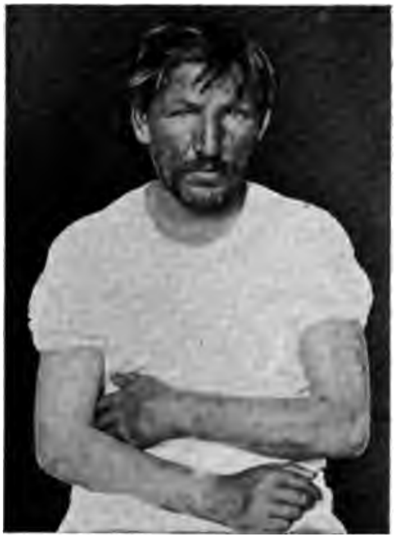


FIG. 270.—Early stage of tubercular leprosy. —German Hospital.

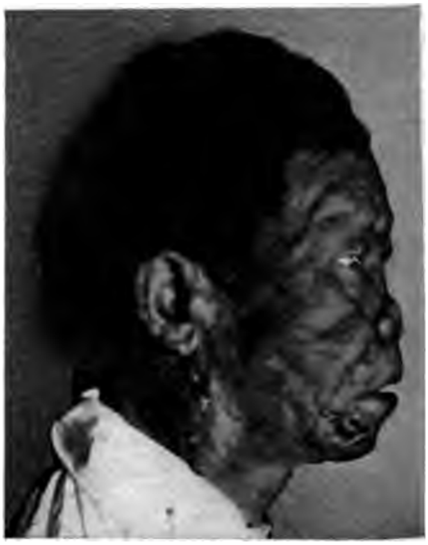


FIG. 271.—Tubercular leprosy.

As cicatrization takes place various deformities result, as stenosis of the mouth or palate, and laryngeal stenosis. The process involves the eyelids and extends to the ocular conjunctiva and the cornea, resulting in ultimate destruction of vision in from 66 to 75 per cent. of the cases.

The duration of the disease is indefinite. The patient may live for years, becoming more and more deformed and helpless. Death commonly results from exhaustion, colliquative diarrhœa, or inhalation pneumonia; sometimes from stenosis of the larynx or trachea or laryngeal œdema.

(b) ANÆSTHETIC LEPROSY.—The period of incubation is commonly more prolonged than in the tubercular form. The onset is insidious and

:erized by subjective cutaneous symptoms, as hyperæsthesia, pruritus, .in. Fever is not common. Persistent and troublesome pains in bs are frequent. Hyperidrosis may be an early symptom. Trophic ances may give rise to the formation of bullæ. Patches of erythema ; in size from one to several centimetres, usually circular or oval in , form upon the trunk and limbs. After a time these spots disappear, ; areas of anæsthesia, but anæsthetic patches may occur without velopment of macules. The erythematous spots show a variety of rom pinkish-red to a bluish- or brownish-red color, and many of them o pigmentation. As the areas become anæsthetic the pigment may lly disappear, leaving well- l white or yellowish patches ing contrast to the surround- in. The superficial nerve- are felt to be enlarged and r. The hair of the affected s may become white or fall nd as the disease develops s complete suppression of per- n. Similar lesions may upon the face. They may discrete or become confluent. disease advances they are tly the seat of bullæ, some of undergo involution, with cica- n, others break down, form- re or less superficial ulcers, on healing leave conspicuous which are at first dark but later pale, smooth, and shining.

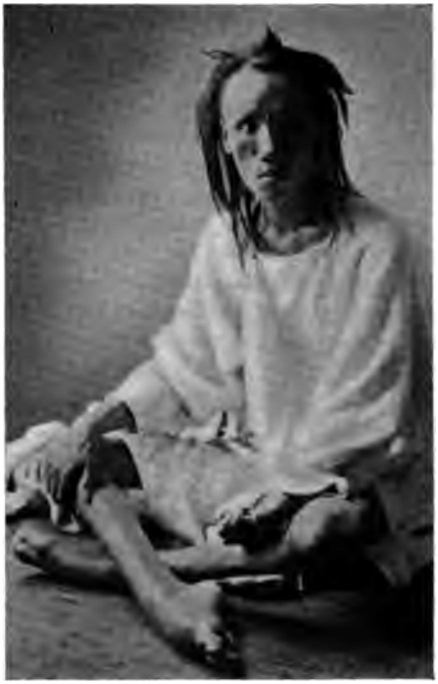


Fig. 272.—Anæsthetic leprosy.

ie modifications of sensibility first of exaltations of sensi- such as have been described; , of perversions of sensation, consist of dysæsthesias, formi- numbness, and delayed sensa- ind third, abolition of sensation, which is more or less complete. ophic changes involve the conjunctivæ and the mucous membranes nose, mouth, and throat, which may become dry and red and the areas of superficial ulceration. The ulcers which form in the anæs- patches developing in the hands and feet may be very destructive, rise to contracture and necrosis, which produce distressing deformi- e loss of fingers and toes, and the development of perforating ulcers. neous resorption of bone may take place.

. favorable cases the disease may last for a long period without velopment of marked trophoneurotic changes. The average dura- f life in this form of leprosy is about twenty years. In some es the progress of the disease is arrested and the patient may n advanced age.

(c) Mixed or Complete Leprosy.—The lesions peculiar to the tubercular and the anæsthetic forms develop simultaneously, or in succession. While the distinction between the two main forms is in well-marked cases sharply defined, there are many cases which must be referred to the mixed form.

Diagnosis.—The direct diagnosis of leprosy in the early stage may be difficult. The erythematous macules with hyperæsthesia, pain, and pigmentation, and the subsequent development of tuberculous nodules are characteristic. In the nervous form the areas of persistent anæsthesia, with bullæ, ulceration, deformities, and necrosis of the hands and feet, are important. A history of residence in a country in which leprosy prevails, even without actual association with known lepers, justifies the suspicion of contagion. The bacteriological examination of the nasal discharge, the serum of a blister, or of an excised nodule may settle a doubtful case.

The differential diagnosis involves the consideration of a great number of chronic affections which present resemblances to leprosy, among which may be named especially syphilis, lupus, multiple neuritis, syringomyelia, and Morvan's disease.

Prognosis.—The experience of history shows leprosy to be an incurable disease. Abortive cases occur but they are extremely rare. The prognosis as regards recovery or even as regards the arrest of the process is highly unfavorable; that as regards the expectancy of life must be guarded. The miserable life of the leper may be prolonged for twenty years or more. The outlook in the tuberculous form is less favorable than in the anæsthetic form.

XXXII. TUBERCULOSIS.

Definition.—An infectious disease caused by the Bacillus tuberculosis, and characterized histologically by the formation of tubercles and infiltrations of tuberculous tissue, which undergo caseation and necrosis or sclerotic changes; anatomically, by alteration and destruction of the parts immediately affected, and clinically, by local and constitutional symptoms which vary according to the structures involved and the extent of the process.

Etiology.—Predisposing Influences.—Tuberculosis is a wide-spread disease affecting both human beings and animals. Among the latter the domestic animals and, in particular, the bovines chiefly suffer. Wild animals in captivity are peculiarly liable to tuberculosis. The liability of the hog is much less than that of horned cattle; of the horse and sheep very slight; of the dog and cat even less, though these animals housed as pets with tuberculous persons sometimes contract the disease. Rabbits and guinea-pigs, especially the latter, are peculiarly susceptible to tuberculous infection, and are for that reason much used for the purposes of laboratory research. Avian tuberculosis constitutes a special variety of the disease. The wide *prevalence* of tuberculosis among human beings is due to methods of living favorable to the propagation of the infecting principle. It is most prevalent in the centres of population and in densely peopled localities in which direct sunlight, fresh air, and cleanliness are little known. About one-seventh of all deaths are caused by tuberculous disease. *Climate* has little influence as a predisposing cause. Tuberculosis is more common in proportion to the population in temperate than in tropical or extreme northern regions. *Altitude* is important. The great plateaus of the United States and Mexico, the settlements of the high regions of the Alps, the Andes, and the Himalayas are remarkably free from tuberculous affections. *Soil and Locality.*—Tuberculous diseases and especially phthisis have been shown to be more prevalent in wet, badly drained districts than in dry uplands with a porous, sandy soil. The influence of soil in this respect consists in an increased liability to catarrhal affections and hence general increase in susceptibility. *Mode of Life.*—Tuberculosis is fostered by darkened houses and unventilated sleeping rooms. Habitual life in the open air is unfavorable to it alike in the individual and in the community. Like other readily transmissible infections, it spreads with great rapidity when introduced among aboriginal peoples previously free from it. *Race.*—The influence of racial susceptibility is much less than that exerted by the mode of life. It is difficult to separate these factors. The American Indian, the negro, and the immigrant peasant from Ireland and Southeastern Europe suffer in this country from an especial liability. There is an apparent relative immunity among the Jews. *Personal Predisposition.*—The phthinoid or pterygoid chest is not only seen in phthisical individuals but it is also characteristic of those who manifest a peculiar susceptibility to phthisis. On the other hand a very large proportion of those who contract the disease have well-developed chests and every evidence of perfect health. Now that tuberculosis is known to be an infection to which the liability is wide-spread, less attention than formerly is paid to the so-called diathetic states. *Age.*—Tuberculous infection may occur at any age. Early adult life, from twenty to thirty-five, is attended with a peculiar liability. The susceptibility of the various tissues and organs varies with different periods of life. In infancy the intestines, glands, and meninges, in childhood and adolescence the bones and lymph-nodes, and later the lungs, other viscera, and the skin are more commonly affected, but any form of tuberculosis may occur at any period of life. *Sex.*—Women become tuberculous in slightly higher proportion than men. *Occupation.*—Those who work in a confined and dusty atmosphere are very liable to contract pulmonary consumption. From this point of view phthisis merits a place among the occupation diseases. *Previous Disease.*—Catarrhal affections predispose to tuberculosis. It is to this fact that must be ascribed the frequency of pulmonary tuberculosis after the acute diseases in which bronchial catarrh plays a prominent part, as pertussis and measles. The marked predisposition to tuberculosis of the lymph-nodes on the part of young children is due to their liability to catarrhal processes in the upper respiratory passages and bronchi. I am not disposed to think that tuberculous infection is especially favored by enteric fever. Influenza and variola are regarded as predisposing influences. In such cases the development of the disease may be either the result of direct infection upon a soil prepared by the antecedent malady or of the lighting up of a latent tuberculous focus. Tabes mesenterica is doubtless in most cases the result of infection by way of the lesions of a catarrhal enterocolitis. Among chronic diseases syphilis and diabetes are very often followed by pulmonary tuberculosis, and the latter affection is a common terminal condition in chronic bronchitis, disease of the heart, cirrhosis of the liver, and chronic nephritis. Cases of tabes and other forms of spinal sclerosis often terminate in pulmonary tuberculosis. In cases of chronic antecedent disease, whether the tuberculous process be frank or latent, the final event is an acute miliary tuberculosis. A blow or injury is very often followed by tuberculous disease of the part. In the case of meningitis, or bone or joint disease, it is necessary to assume an antecedent latent tuberculosis; when pleural or pulmonary tuberculosis follows a contusion of the chest in the absence of fracture of a rib or laceration of the pleura or lung, we may assume that the resistance of the part has been impaired by the injury, and infection thus rendered possible. Much more commonly, careful inquiry will elicit a history of previous symptoms of tuberculosis. The danger of a surgical operation in local tuberculosis is always to be considered. Latent trouble elsewhere may be lighted up, or an acute miliary process develop.

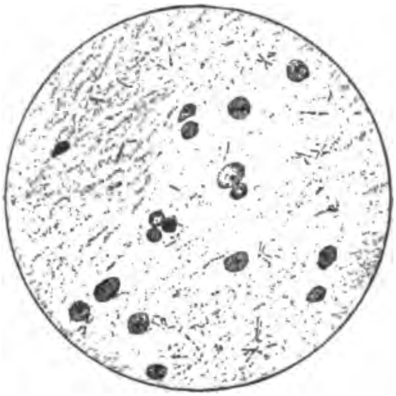


FIG. 273.—Spread of sputum showing Bacillus tuberculosis.

THE PATHOGENIC ORGANISM.—The demonstration by Koch of the tubercle bacillus and the etiological unity of the tuberculous diseases constitutes one of the most remarkable and beneficent achievements of modern medicine and ranks in importance with Jenner's great work.

The bacilli are present in varying abundance in all the lesions of tuberculosis. They are very numerous in active lesions, but scanty in the sluggish processes of chronic glandular or bone disease. When a tuberculous focus, as a softened lymph-node, discharges its contents into a vein or lymph vessel, the bacilli are swept on with the current to effect new lodgement and produce new tubercles at various points in the body. When not found in the effusions of serous tuberculous inflammation, as pleurisy, or in sections of chronic or obsolescent lesions, the nature of the process may frequently be demonstrated by culture or inoculation. There are specific varieties of tubercle bacilli. Koch (1901) stated that the bacillus of bovine tuberculosis does not cause the disease in human beings nor the bacillus of human tuberculosis the disease in cattle. Differences in the two forms are recognized; but the view that bovine tubercle bacilli may give rise to tuberculosis in man is now generally accepted. Individual strains differ greatly in virulence. The bacilli are thrown off by way of the discharges, the sputum in laryngeal and pulmonary tuberculosis, the urine, fæces, vaginal discharges and rarely the semen in the genito-urinary forms, and tuberculous sinuses, abscesses, and ulcerated surfaces in various parts of the body. They retain their vitality outside the body for an undetermined period.

The chief source of infection is the sputum. Beside this all others fall into insignificance. The principal vehicle of transmission as shown by Cornet is the atmospheric dust.

Modes of Infection. — (a) *Heredity.* — Clinical and experimental evidence are alike against transmission from the male by means of the spermatozoids. The hypothesis that transmission may occur from the tuberculous mother by way of the ovum has some experimental support. Transmission by way of the blood, the bacilli penetrating the placenta, is supported by clinical and laboratory facts. The placenta under such circumstances is usually tuberculous, but in some instances it has been apparently normal. The number of reported cases of congenital tuberculosis in man is limited.

The difficulties in determining the part played by heredity in individual cases arise from the uncertainty in regard to the transmission of individual susceptibility from the affected forebears to the offspring, the absence as yet of definite conclusions as to the prolonged latency of tubercle bacilli in the tissues of an apparently healthy child of a tuberculous parent, and the fact that tuberculosis is at the outset a distinctly local process, which may, when acquired by postnatal infection, become circumscribed or obsolescent, and remain latent in the tissues (lymph-nodes, for example) for an indefinite period, to become again active under various circumstances, as traumatism, acute disease, or softening and rupture of a gland-capsule. The mere fact that a parent, grandparent, or collateral relation in a previous generation suffered from tuberculosis no more proves the hereditary nature of the disease than the occurrence of scarlet fever in them would render it hereditary. Yet the assumption in regard to the one disease might with the same indifference to scientific accuracy be made of the other.

(b) *Inoculation.*—Tuberculosis in man has been in rare instances produced by inoculation. Those who work in the post-mortem room frequently contract local skin tuberculosis, the nature of which has been demonstrated both microscopically and by inoculation in animals—post-mortem warts. These lesions are discrete, small nodules and are almost always situated upon the backs of the hands or fingers. Inoculation may also take place in various accidental ways; among these are circumcision, cuts from the broken spit-cup of a consumptive, and the bite of a tuberculous person. There is no evidence to support the assertion of the antivaccinationist that consumption is transmitted in the vaccine virus, beyond the fact that in a very limited number of cases lupus has developed at the site of the vaccine pock. In point of fact, the lesion almost always remains local at the seat of inoculation.

(c) *Inhalation.*—The vehicle is atmospheric dust to which bacilli are attached. This dust in rooms occupied by the consumptive and around his person is made up of the dried pulverized sputum. An obvious measure of prophylaxis is to keep the sputum, discharged into suitable receptacles, moist and disinfected until it can be destroyed by fire or otherwise effectually disposed of. In some instances direct infection takes place by the inhalation of tubercle bacilli contained in the fine particles of moist sputum ejected by the patient in the act of coughing or even in conversation, and remaining for a time suspended in the air in his immediate neighborhood. Among the facts that lend support to the hypothesis of infection by inhalation are the frequency with which the early lesions involve the larynx, lungs, and bronchial glands; the prevalence of the disease in cloisters, asylums, prisons, and other similar institutions, and the remarkable investigations of Flick, who showed that not only certain localities but also particular houses become infected, and that members of different families successively occupying such houses succumb to the disease.

The degree of intimacy of association with the consumptive plays an important part in the danger of the transmission of the disease. It is no uncommon thing for a woman who has nursed a consumptive daughter or sister to contract the disease and die of it in the course of some months. This occurrence is infrequent among nurses, whose attendance is less close and hours are less prolonged. The latter have also an advantage in their technical knowledge of the dangers and in habits of prophylaxis. In the case of husband and wife this danger of the transmission of pulmonary tuberculosis is abundantly established by common observation and the results of statistical inquiries.

(d) *Infection by Food.*—The milk of tuberculous cows, and milk foods, including butter made from it, have been shown to be capable of giving rise to the disease. Tuberculous lesions of the udder are not necessary, the milk of animals healthy in this respect having been shown to be infectious. Bovine tuberculosis constitutes a positive danger to the human race. The frequency of tuberculosis of the intestines and mesenteric glands in young children finds a ready explanation in infected milk.

The meat of tuberculous animals is not without danger. It has been shown experimentally to be infective to guinea-pigs. Thorough cooking probably destroys the infecting principle; uncooked meats, smoked beef and similar articles may convey the disease. Legal enactments against the exposure for sale of the flesh of tuberculous animals are well founded.

A. Acute Miliary Tuberculosis.

Tubercle bacilli find their way into the blood from a focus of tuberculous endangitis or the perforation of the vessel wall by a softening caseous mass.

VARIETIES.—Three clinical forms occur: (a) the general or so-called typhoid, characterized by the symptoms of an acute general infection; (b) the pulmonary, in which the symptoms are chiefly referable to the lungs, and (c) tuberculous meningitis.

(a) **Generalized Miliary Tuberculosis.**—Miliary nodules are thickly disseminated throughout the various organs of the body.

Symptoms.—The symptoms are those of an acute general infection, and the condition presents many of the features of enteric fever, for which it is often mistaken. The signs of local disease are rarely marked. The onset is gradual after a period of rapidly failing health. In some of the cases the onset is abrupt. Nose-bleeding is not common. The pulse is rapid in proportion to the fever, not often dicrotic, and shows remarkable variations in frequency within short periods of time. The fever increases, the temperature being very irregular, usually much higher in the evening than in the morning and often reaching 104° F. (40° C.) or more with remissions of three or four degrees. In some of the cases the range is distinctly intermittent. Inverse temperatures are occasionally observed. There is profound asthenia with rapid loss of flesh. Pulmonary symptoms increased respiration frequency, especially early in the attack, and dyspnœa with faint cyanosis, are common. Diffuse râles, the signs of a bronchitis not unlike that of enteric fever, are heard, and in some of the cases there may be dulness at an apex, or patches of subcrepitant and crepitant râles at various parts of the chest. In a group of cases there is no fever or the temperature does not exceed subfebrile ranges, and the true nature of the disease is revealed only upon post-mortem examination. Delirium is less common than somnolence and stupor, which deepen to coma, terminating in death. Toward the last there may be an intensification of the lung symptoms, or the evidences of involvement of the meninges. Cheyne-Stokes respiration is common.

Diagnosis.—The DIRECT DIAGNOSIS of this form of acute miliary tuberculosis rests upon the signs of a profound toxæmia, irregularity of the temperature, the occurrence in some cases of an inverse range, the rapidity and irregularity of the pulse, and the absence of the signs which characterize the other specific infections and septic processes. If there be localized pulmonary signs, the history of glandular or bone tuberculosis, or meningeal symptoms, the diagnosis becomes more probable. Choroid tubercles, or tubercle bacilli in the fluid obtained by lumbar puncture, or in the blood, render it positive and final.

The DIFFERENTIAL DIAGNOSIS between general miliary tuberculosis and enteric fever very often taxes the art of medicine to its utmost. This is particularly the case when the onset is marked by bronchitis of some severity, attended by dyspnœa and faint cyanosis, with little cough. Under these circumstances great rapidity and irregularity of respiration and pulse, and in general terms a correspondence in their frequency, irregularity of the temperature and conformity to the remittent or intermittent rather than the subcontinuous type, signs of deficient oxygenation of the blood, constipation, only slight enlargement of the spleen, herpes, the absence of rose spots, and a leucocytosis are in favor of miliary tuberculosis. The following facts must receive due consideration. There are cases common in childhood and not extremely rare in adult life in which the fever curve of enteric fever is remittent throughout. Constipation is by no means rare in enteric fever, and diarrhœa may occur in miliary tuberculosis. Enlargement of the spleen occurs in both diseases, but is neither so early nor, except occasionally in children, so decided in tuberculosis. Herpes, though rare, has been observed in enteric fever. Rose spots occur in tuberculosis, but they are rare and appear singly rather than in crops, and do not present the appearance nor run the course of the eruption which characterizes enteric fever. Leucocytosis is common in miliary tuberculosis; leucopenia the rule in enteric fever. But leucocytosis occurs in enteric fever complicated by inflammatory and suppurative processes. Albuminuria and the diazo reaction may occur in both diseases. The Widal reaction may not be conclusive, since it may occur in an individual who has passed through an attack of enteric fever and subsequently become tuberculous. Nor, for the same reason, is the presence of Bacillus typhosus in the urine conclusive. If that organism is, however, found in blood cultures the diagnosis of enteric fever may be made. The two conditions may coexist and the lesions of both diseases have been present upon post-mortem examination.

(b) **The Pulmonary Form.**—The acute phenomena develop in persons who suffer from persistent cough or are known to have chronic pulmonary tuberculosis, or, especially in children, may follow an infectious disease, as measles or whooping-cough.

Symptoms.—The onset is like that of an acute bronchitis or bronchopneumonia. Troublesome cough, mucopurulent expectoration, sometimes rusty sputum or blood spitting, dyspnœa, slight cyanosis, and a dusky flushing of the cheeks are among the symptoms that attract attention. In children especially, but sometimes also in adults, there are patchy areas of dulness at the bases posteriorly, with areas of tympanitic resonance, the sign of collateral emphysema. Râles of larger size, both sibilant and sonorous, may be heard; but much more commonly they are fine or coarse crepitant. A grazing friction scarcely to be distinguished from the finest subcrepitant or crepitant râles is the sign, as established by post-mortem observations, of a miliary tuberculosis of the pleura. Bronchial breathing of high pitch is, in children, often heard at the bases and opposite the root of the lung. The temperature is not usually very high, 102°–103° F. (38.9°–39.5° C.), often irregular, and may be of inverse type. The pulse is irregular. The spleen is usually enlarged. Toward the end cerebral symptoms sometimes develop, the pulse grows more feeble and rapid, coarse râles obscure the finer respiratory signs, and Cheyne-Stokes breathing occurs. The duration of the disease varies from a fortnight or more in the acute to several months in the chronic cases.

Diagnosis.—The DIRECT DIAGNOSIS is not usually attended with difficulty. In children this form of miliary tuberculosis very often follows measles and whooping-cough and constitutes the principal danger in those diseases. The history of chronic tuberculosis, pulmonary, glandular, or joint, is of diagnostic importance. Tubercle in the choroid, when found, establishes the diagnosis, but the proportion of cases in which it occurs is limited. The occurrence of meningeal symptoms is important. Tubercle bacilli are often absent from the sputum upon repeated examination; when found their significance is positive.

DIFFERENTIAL DIAGNOSIS.—This relates to non-tuberculous bronchopneumonia. The anamnesis is very important. A history of tuberculous disease in any of its forms, even a chronic cough without impairment of general health, merits careful consideration. The degree of dyspnœa and cyanosis, tubercle bacilli, hæmoptysis, and, above all, the persistence of the fever and other symptoms are of diagnostic value.

(c) **The Meningeal Form.**—*Acute Tuberculous Meningitis.*—An acute miliary tuberculosis in which the membranes of the brain, less commonly also those of the cord, are chiefly implicated. As the membranes of the base are the common seat of the process, the affection is sometimes spoken of as basilar meningitis. This form is especially common between the second and fifth years of life. It is rare during the first year and relatively infrequent in adult life. A primary tuberculous depot may usually be discovered, most frequently in the bronchial or mesenteric glands. It may be in the lungs, the middle ear, the bones, or genito-urinary organs. In a small proportion of the cases the most careful autopsy fails to reveal the local source of the infection.

Symptoms.—There are frequently prodromal symptoms. These consist of gradual loss of appetite and weight, irritability, fretfulness, and change of disposition. There may be a history of recent measles or whooping-cough, or of a fall.

STAGE OF IRRITATION.—The onset may be attended with a severe general convulsion, or with the usual triad of meningeal symptoms—headache, vomiting, and retraction of the muscles of the back of the neck, the last being less marked than in cerebrospinal fever. Fever is present, usually moderate but gradually increasing to 102°-103° F. (39°-39.5° C.). The pain is intense and paroxysmal, and the exacerbations are accompanied by a short sudden scream—*hydrocephalic cry*. The child holds its hand to its head, and sometimes screams continuously for hours at a time. The vomiting is without apparent cause and is repeated from time to time. The bowels are usually constipated. The retraction of the neck may be slight at first and only manifest when the head is bent forward or rotated. The respiration frequency remains normal, but the pulse, at first rapid, becomes irregular and slow. Sleep is restless and accompanied by muscular twitchings and sudden starts and cries. The pupils are as a rule contracted, and very often to a greater degree upon one side than upon the other. Kernig's sign is present.

TRANSITIONAL STAGE.—The signs of irritation gradually and irregularly subside. The vomiting ceases, the belly becomes retracted and scaphoid, and constipation is stubborn. Headache is replaced by dulness, stupor, and occasional delirium. The retraction of the neck continues, the pupils are dilated and irregular, and strabismus is common. There may be convulsions, or rigidity of various muscle groups. The respiration is at times sighing and irregular, and when disturbed the child utters the sudden, shrill cry so often heard in the disease. The temperature is irregular and atypical. Irregular patches of erythema are noted, and if the skin is tapped with the finger-tip, or the nail drawn across it, a vivid red spot or line shortly appears and only slowly fades—*tache cérébrale*—a sign of little diagnostic value since it occurs in enteric fever, hysteria, and other conditions in which there is relaxation of the peripheral vessels.

STAGE OF PARALYSIS.—The stupor deepens to coma and the patient cannot be aroused. Muscular spasms occur and there may be general convulsions. The pupils are dilated and irresponsive to light; there is paralysis of ocular muscles, and ophthalmoscopic examination reveals optic neuritis. Tubercles in the choroid are by no means always seen. Tetanoid contractions, cataleptic states, tremor, and athetoid movements occur, and in some cases there are hemiplegias or monoplegias. Aphasia may occur. The pulse now becomes rapid and feeble, and the symptom-complex known as the typhoid state, with dry tongue, muttering delirium, involuntary discharges, and subnormal temperature, develops. The duration of the attack varies from two to four or five weeks. There is sometimes a pre-agonistic rise of temperature.

A moderate leucocytosis is usually present during the whole course of the attack. There are cases which begin with great abruptness and intensity, in persons apparently in good health, and run their course in a few days; on the other hand there are cases which run a chronic course, with anomalous symptoms suggestive of tumor of the brain.

Diagnosis.—The DIRECT DIAGNOSIS of tuberculous meningitis rests upon the presence of the signs of a local tuberculous process, the mode of onset, which differs from that of cerebrospinal fever, the course of the disease, and the results of lumbar puncture. The fluid withdrawn is usually turbid and often contains tubercle bacilli. It is sometimes sterile.

DIFFERENTIAL DIAGNOSIS.—For the details of the differential diagnosis between tuberculous meningitis and pneumococcus and streptococcus meningitis and cerebrospinal fever see Cerebrospinal Fever.

B. Tuberculosis of the Lymph-nodes—Scrofula.

Tuberculosis of the lymph-nodes is more common in children than in adults, but it occasionally occurs in middle life and infrequently in persons of advanced age. Catarrhal inflammation of mucous membranes, by which their resistance is impaired, is probably the most important predisposing factor in this form of tuberculous disease. Tonsillitis and nasopharyngeal catarrh doubtless stand in a causal relation to the cervical adenitis so common in childhood; measles, pertussis, and recurrent attacks of catarrhal bronchitis, to tuberculosis of the bronchial glands; and the intestinal diseases to which infants are prone afford the gateway of infection of the mesenteric glands.

Glandular tuberculosis is very commonly a local form of the disease. There is a remarkable tendency to encapsulation and latency. The deposition of lime salts is common. A long quiescent bronchial gland may become the source of a local or general tuberculous process.

VARIETIES.—(a) Generalized, and (b) local tuberculosis of the lymph-nodes are to be considered.

(a) **The Generalized Form.**—A form of the disease in children characterized by the successive implication of groups of glands, and terminating in a general tuberculous cachexia or in meningitis has been described.

(b) **Local Tuberculous Adenitis.**—The groups usually affected are, in the order of their frequency, the cervical, the tracheobronchial, and the mesenteric. The cervical glands are very frequently involved. The children of the poor, and especially the negro, mostly suffer, but those living in affluence do not wholly escape. Chronic rhinitis, tonsillitis, otitis media, eczema capitis vel faciei, conjunctivitis, or keratitis may afford the port of entry for the infection.

Cervical Glands.— SYMPTOMS.—The submaxillary glands are most frequently and usually first involved. The posterior cervical chain, the glands above the clavicle, and the axillary glands are also affected in many cases. The disease may affect one or both sides; when both, to a much greater extent on one than the other. The bronchial glands may also be tuberculous. Infection of the pleuræ or lungs may subsequently take place. The enlarged glands may at first be felt as discrete, smooth, firm, and somewhat elastic tumors, over which the skin is freely movable. They rapidly enlarge and coalesce, forming large disfiguring masses to which the overlying skin becomes adherent, with subsequent inflammation and suppuration. If the resulting abscess be not opened, it breaks, leaving a sinus which heals slowly, followed by a characteristic, retracted, ightly scar. Fever is usually present during the active stage of the cess and the patient is anæmic. The process is slow, but many of the ›s, especially in children, ultimately recover.

Diagnosis.—*Direct.*—Indolent glandular enlargements in the neck axillary region, more marked on one side than the other, becoming erent and slowly softening with abscess formation, are usually tuber-)us, especially in children who suffer from local catarrhal or inflam- ;ory diseases of the upper air-passages, otitis media, eczema of the d or face, chronic conjunctivitis or keratitis, or tuberculous disease in ər parts of the body.

Differential.—Slight cervical adenitis occurs in connection with various ırrhal processes and the exanthemata. As a rule these enlargements lually undergo resolution as the nary disease subsides. Sometimes ŗ suppurate. This shorter course of process, the association with acute ase, and the slighter degree of en- ement are of diagnostic importance. Hodgkin's disease may at first be ŗ difficult to recognize. The greater luency of tuberculous adenitis in dren, the early implication of the ıds in the submaxillary region, the ŗ development, the tendency to ımmatory adhesions among the ıds themselves and to the skin, and suppuration and abscess formation, in favor of the tuberculous nature he process. Limitation to a group glands, as the cervical or axillary, or to one side, is much more ımon in tuberculous adenitis than in Hodgkin's disease.

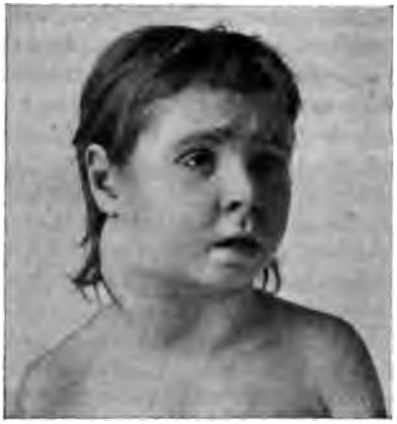


Fig. 274.—Chronic cervical adenitis.—Rotch.

Tracheobronchial Glands.—This form of tuberculosis is very common 'oung children, and particularly so in the inmates of foundling asylums, hanages, and similar institutions. The glands may attain large size. trachea and bronchi may be flattened, and pressure may be exerted n the superior cava, the pulmonary artery, and the azygos vein. softening caseous contents of the glands may perforate into the ıchi or trachea and cause asphyxia; into the great vessels with gen- infection of the blood stream, or very rarely into the œsophagus. monary infection very often occurs either by contiguity of tissue or ıg the root of the lung. Pericardial tuberculosis may occur.

Symptoms.—Pressure symptoms occur, but they are less common and urgent than the anatomical conditions suggest. The enlarged mass stitutes one of the forms of mediastinal tumor. Dyspnœa, paroxysmal, ısy cough from pressure on the recurrent laryngeal nerves, cyanosis and iness of the face from pressure on the superior cava, dysphagia from com- sion of the œsophagus, are occasional symptoms. In the majority of s the mechanical disturbance is slight or absent altogether. Nor are defi- physical signs common. Impaired resonance upon light percussion over the manubrium sterni may be noted, and slight relative dulness along the spine in the upper dorsal region. These physical signs are, however, neither so constant nor so well marked as to serve a useful purpose in the diagnosis.

Mesenteric Glands—Tabes Mesenterica.—A slight enlargement is common and may not give rise to special symptoms. As a rule the enlargement is general and attains considerable size. The retroperitoneal glands are often coincidently involved. Caseation and softening occur. Resorption of the fluid portions and the deposition of lime salts sometimes take place. The tuberculosis may be primary, infection having arisen by way of the lesions of intestinal catarrh, or it may be secondary to tuberculous lesions of the bowel.

SYMPTOMS.—Tabes mesenterica is common in very young children. The belly is enlarged and tympanitic; the enlarged glands cannot always be felt; there is diarrhœa with thin, watery, and offensive stools. The nutrition is deranged and the little patients are anæmic, puny, and wasted. The superficial abdominal veins are often enlarged and conspicuous. There is fever of hectic type. In a group of cases there is an associated tuberculous peritonitis, the belly is distended, firm, or doughy, and nodular tumors may be felt. Massive tuberculous enlargement of the mesenteric and retroperitoneal glands occasionally occurs in adults.

DIAGNOSIS.—*Direct.*—In young children the diagnosis is commonly attended with no great difficulty. The appearance of the child is suggestive. Sometimes the enlarged glands are palpable. Tuberculous adenitis elsewhere, or the evidence of tuberculous disease of the lungs, is of diagnostic importance.

Differential.—The diagnosis of the essential character of circumscribed tuberculous glandular masses in the abdomen, especially when they are of considerable size, and in the adult without tuberculous disease of the intestines, peritoneum, or lungs, is often attended with difficulty. The differential diagnosis can in many of the cases only be reached by exclusion and may even then remain in doubt. Tuberculin may be used.

C. Tuberculosis of the Serous Membranes.

General Tuberculosis of Serous Membranes.—The process may be general, the pleuræ, pericardium, and peritoneum being involved simultaneously or in rapid succession. There may be an acute miliary tuberculosis; a more chronic form with agglomeration of tuberculous material with caseation and inflammatory and suppurative lesions; and finally a chronic proliferative process with firm tuberculous nodules, fibroid lesions, great thickening of the membranes, and the absence of exudate. The pericardium is less frequently involved than the pleuræ and peritoneum.

Tuberculous Pleurisy.—The pleurisy may be acute, with fibrinous, serofibrinous, purulent, or hemorrhagic exudate; or it may be chronic. It is very often latent. Secondary and terminal forms occur. A rare form of acute tuberculous pleurisy with ulceration and necrosis of the pleura has been described. Subacute cases with a serofibrinous exudate are very common. They are almost constantly associated with circumscribed tuberculous disease of the lungs, or with tracheobronchial adenitis. The exudate may become purulent. There are cases in which no signs of tuberculous disease can be found, in which after a period of latency varying from a few weeks to many years, with excellent health, pulmonary or acute miliary tuberculosis supervenes. The visceral pleura is always involved in pulmonary tuberculosis extending to the periphery of the lung. Adhesions with more or less thickening result. In the absence of protecting adhesions a caseating mass in the lung may perforate the visceral pleura and cause pyopneumothorax. Finally there is a chronic adhesive pleurisy with great thickening and involvement of the interlobar pleura and the lung itself. For the symptomatology and diagnosis of tuberculous pleurisy see Pleurisy.

Tuberculosis of the Pericardium.—The process may be part of a general miliary tuberculosis, or latent in cases of chronic tuberculosis or other chronic disease, or it may cause a chronic adhesive pericarditis analogous to the more common chronic adhesive pleurisy. There are acute cases with fibrinous or plastic, serofibrinous, hemorrhagic, or purulent exudate, and the ordinary symptoms of pericarditis, in which, in the absence of tuberculous disease elsewhere, the true nature of the process cannot be recognized *intra vitam*. (See Pericarditis.)

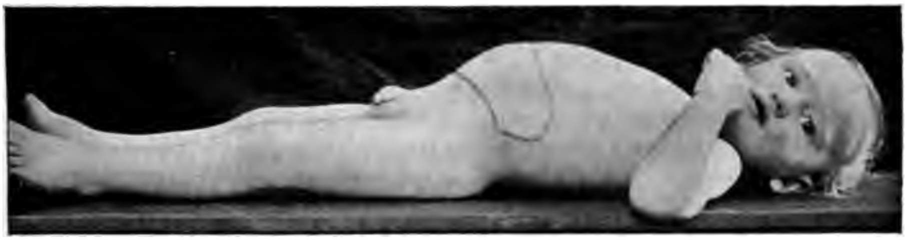


FIG. 275.—Tuberculous peritonitis. Outline indicates a hard mass which was found on operation to consist of areas of nodules matting together the intestines.—Rotch.

Tuberculosis of the Peritoneum. — There may be diffuse miliary tuberculosis or circumscribed areas corresponding to tuberculous ulceration of the intestine, acute miliary tuberculosis with serofibrinous or bloody exudate, chronic tuberculosis with agglomerations of tuberculous tissue undergoing caseation and necrosis, chronic proliferative or fibroid peritonitis with extensive adhesions and thickening of the capsule of the liver and spleen. The infection takes place by way of the intestines, especially in children, and in adults is propagated from the Fallopian tubes or the seminal vesicles, prostate, or testicle. In by far the largest proportion of cases infection of the peritoneum is secondary to tuberculosis of the lungs or pleura. Tuberculous peritonitis has been known to follow contusion of the abdomen; it sometimes has its starting point in the hernial sac and often constitutes a terminal condition in chronic visceral disease, especially cirrhosis of the liver.

Symptoms.—Tuberculosis of the peritoneum presents peculiar clinical phenomena which serve to distinguish it from peritonitis due to other causes.

The disease may be latent and discovered only upon operation, or post mortem. In other cases the onset may be sudden with urgent symptoms, as fever, vomiting, pain, and tenderness. There are cases in which the onset and early symptoms suggest enteric fever.

Fever occurs in the acute cases and the temperature often reaches 104° F. (40° C.) or more; in many of the cases the rise is only to subfebrile ranges—100° F. (37.8° C.). In the chronic cases fever is absent and subnormal temperatures often occur. The pulse is variable and, in the absence of fever, of moderate frequency. Tympanitis is common in the acute cases, but in the chronic form the belly may be small and doughy. Ascites of small amount is common; it is usually serous, but may be purulent or hemorrhagic, and is sacculated from the beginning, or soon becomes so. A diffuse pigmentation of the skin may suggest Addison's disease. Irregular attacks of pain associated with fever and digestive disturbance occur, and tenderness upon pressure is a more or less continuous symptom. Dense infiltration of the omentum with tubercles and fibrinous exudation may convert that structure into a thick, cord-like mass adherent to the transverse colon and extending across the abdomen. This tumor-like omental thickening may be recognized upon palpation. Similar masses may be felt in other parts of the abdomen. Sacculated fluid exudates confined by adhesions among the abdominal or pelvic organs, the mesentery, and the walls, form cyst-like tumors which suggest ovarian or other cysts and often lead to errors in diagnosis.

Less frequently the mesentery of the small intestine, the root of which extends in an oblique direction from the lumbar vertebræ to the right sacro-iliac symphysis, undergoes thickening and retraction, which is associated with great shortening of the intestine and thickening of its walls in such a manner that the bowel is drawn together into a tumor-like mass occupying the right side of the abdomen. These changes may be so extensive as to involve the bowel in its entire length. Massive enlargement of the mesenteric glands is very often present in tuberculous peritonitis, especially in children.

Diagnosis.—The DIRECT DIAGNOSIS of tuberculosis of the peritoneum is attended with much difficulty in the acute cases with sudden onset, great pain and tenderness, rigidity of the abdominal muscles, vomiting, and fever. The presence of tuberculous lesions elsewhere, and in particular at the apex of one lung, in the pleuræ, bones, or genito-urinary tract, is of great diagnostic importance. In this group of cases prompt operative measures are indicated, not only in order to clear up the uncertainty as to the causal conditions—*surgical diagnosis*—but also as the only curative measure which yields promise of relief in several of the conditions, as perforation of the bowel or other hollow viscus, or the rupture of an abscess or cyst, which cause acute peritonitis with precisely the same symptoms. In the subacute forms the direct diagnosis cannot always be made. Persistent abdominal symptoms in a tuberculous individual constitute sufficient ground for a provisional diagnosis, which the subsequent course of the case will frequently confirm. Many of the subacute cases are latent.

The diagnosis in the chronic forms depends upon the presence of the general symptoms of chronic peritonitis, the recognition of tuberculous foci in other parts of the body, encysted fluid exudate, or irregular tumor-like masses within the abdomen, the tuberculin test, which may be used in any doubtful case unattended by fever or with fever of only moderate range, and the finding of tubercle bacilli in the fluid obtained by paracentesis, or a positive reaction to the injection of such fluid into guinea-pigs.

DIFFERENTIAL.—The points of discrimination between peritonitis e to other causes and tuberculous peritonitis have been indicated the foregoing paragraphs. Non-tuberculous neoplasms are usually ore local, circumscribed, and definite in their relation to the viscera, as e kidneys, spleen, or liver. Fever is less apt to occur. But more impornt still are the absence of prior or concurrent evidences of tuberculosis ewhere, and negative results of laboratory investigations.

The omental and intestinal tumors which occur in the chronic forms e fairly distinctive and only lead to uncertainty in the case of malignant ease. The differential diagnosis rests upon the anamnesis, the more rapid sting in cancer, and the differences in the cachexia of the two conditions.

Ovarian Cysts.—Errors of diagnosis are common. In some of the es of tuberculous peritonitis with encysted exudate the general health fairly well preserved. The physical signs may be similar in both condins. The contour in tuberculous pseudocysts is less regular; areas of lness upon percussion, or palpable nodular masses may be demonstrable, d changes in form or position may arise with variations in the amount of s in the coils of intestines. Depression of the vault of the vagina occurs both conditions. Tubal disease and nodular masses in one or both arian regions are suggestive. Febrile outbreaks are common in tuberlosis, but rarely occur in non-inflammatory ovarian cysts.

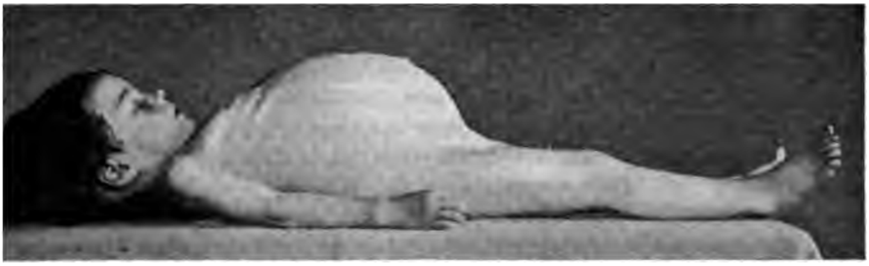


FIG. 276.—Tympany due to tuberculous peritonitis.—German Hospital.

Cirrhosis of the Liver.—If the ascites is so great as to interfere with e palpation of the liver in a doubtful case, paracentesis is necessary for ignostic purposes. A hemorrhagic fluid may be present in tuberculosis or rcinoma. This occurrence, together with thickening of the peritoneum or monstrable tumors, or the evidence of tuberculosis or carcinoma in distant gans is diagnostic. In a considerable proportion of the cases of hepatic rhosis, tuberculosis of the peritoneum occurs as a terminal condition.

D. Tuberculosis of the Alimentary Canal.

Tuberculous lesions of the structures forming the digestive tract, th the exception of the liver and intestines, are rare.

Lips, Tongue, and Mouth.—Tuberculous ulcers of these organs occur rare instances, mostly in association with laryngeal or pulmonary disease. on the lips they are liable to be mistaken for chancre or epithelioma. berculous ulcers upon the tongue occur in the form of deep circumscribed lesions, with well-defined but irregular borders and a caseous base. They resist treatment, not being influenced by the iodides, and tend to spread. The glands at the angle of the jaws are not enlarged. The salivary glands are very rarely the seat of tuberculous infection. Tuberculosis of the hard and soft palate in rare instances is the result of the invasion of these structures from adjacent parts. The tonsils are frequently infected. There may be superficial ulceration or diffuse infiltration with miliary tubercle. Caseous depots may be present. Infection may take place by means of tuberculous milk or other food, dust, or by the sputum in pulmonary disease. The frequency of tuberculous cervical adenitis, especially in children, finds an explanation in tonsillar disease. In ulcers of doubtful character upon the lips and tongue, or elsewhere in the mouth, a portion of the tissue may be excised for examination, or inoculations may be made. In a suspicious ulcer of the tongue failure of the iodides and absence of glandular involvement are against a diagnosis of syphilis.

Pharynx and Œsophagus.—In laryngeal and chronic pulmonary tuberculosis miliary tubercles and superficial ulceration frequently invade the oropharynx. The latter condition, when associated with ulceration of the epiglottis, is attended with great pain upon deglutition, and constitutes a most distressing condition in laryngeal phthisis. Adenoid vegetations of the nasopharynx are in some instances infected. An extension from the larynx may invade the upper part of the œsophagus. Rare cases of tuberculous ulceration have been reported.

Stomach and Intestines.—Ulceration of the wall of the stomach is a recognized pathological condition but the diagnosis cannot be made during life, since the symptoms are the same as in ordinary peptic ulcer. It has occasionally been observed post mortem in tuberculous subjects, but non-tuberculous peptic ulcer is more liable to occur in those debilitated and rendered anæmic by tuberculous disease. The probability that a peptic ulcer may become tuberculous is to be considered. Intestinal tuberculosis may be primary, especially in children, and is then usually followed by infection of the mesenteric glands or peritoneum. Primary tuberculosis of the intestine in the adult is exceedingly rare.

Symptoms.—Irregular diarrhœa, colicky pains, and moderate fever occur. Intestinal hemorrhage may be the initial symptom. Emaciation and signs of tuberculosis of the lungs or elsewhere suggest the actual pathological condition. There are cases in which the tuberculosis begins in the cæcal region, and the symptoms are circumscribed tenderness, slight irregular fever, and diarrhœa alternating with constipation. When these symptoms subside and recur after quiet intervals of varying duration, the condition simulates a chronic appendicitis. Hemorrhage occurs and necrosis may take place, causing peri-appendicular abscess or perforation into the peritoneum. Thickening of the intestinal wall forms part of the process.

SECONDARY LESIONS are much more common. The lower portion of the ileum and the large bowel are usually involved. Infection occurs by means of the swallowed sputum, and the intestinal disease gives rise to troublesome and distressing symptoms in the later stages of many cases of phthisis. The lymphatic glands are early involved and there is frequently extensive ulceration of the mucous membrane of the small and large bowel. There may be ovoid ulcers in the ileum, corresponding to Peyer's patches, but as a rule the tuberculous ulcer is transverse and in many cases annular. Its borders and floor are thickened from the infiltration of tubercle, which shows caseation at various points. The muscular coat is often involved, patches of recent tubercles are seen upon the corresponding serosa, local adhesions occur, forming knot-like masses among the intestinal coils, and in rare instances perforation takes place. Sclerotic changes often proceed side by side with caseation and necrosis, and lead to cicatrization, irregular puckering, and stenosis. These lesions are sometimes localized in the cæcum and appendix, and form dense sausage-shaped tumors in the right lower quadrant of the abdomen, slightly or not at all movable, painful upon palpation, and suggestive of carcinoma. More extensive adhesions and infiltration in this region sometimes occur, and in rare instances a fecal fistula. *Fistula in Ano—Anal Fistula.*—This condition is in a large proportion of the cases tuberculous and associated with pulmonary tuberculosis which is sometimes latent or obsolescent. Operation is occasionally followed by a flaring of the lung trouble into activity, whether *post hoc* or *propter hoc* cannot always be determined. This fact does not militate against effectual operation by excision, since it is better to suffer from one focus of tuberculosis than from two.

Secondary tuberculous ulceration of the intestine manifests itself by a group of abdominal symptoms superadded to those of the pre-existing disease, usually pulmonary. Less frequently the intestinal lesions are secondary to tuberculosis of the peritoneum, the primary infection being in the lymph-nodes in children, or the genito-urinary tract in adults of either sex. Abdominal pain, tenderness, loss of elasticity with local doughiness or tumor formation, particularly in the right iliac region, diarrhœa often alternating with constipation, and later the signs of stenosis of the bowel, namely, local bloating, smooth sausage-shaped tumors indicating the contour of the distended gut, and stormy peristalsis, make up the clinical picture. In the rare cases in which the obstruction becomes complete the ominous characteristic symptoms of occlusion of the bowel (q.v.) appear.

Diagnosis.—The DIRECT DIAGNOSIS of primary tuberculosis of the intestine cannot always be made even in children. It depends upon hereditary predisposition, the possibility of feeding upon the milk of tuberculous cows, irregular high fever, rapid emaciation and loss of strength, and the presence in the stools of many tubercle bacilli upon repeated examination. Secondary lesions may be diagnosticated when persistent abdominal symptoms, not yielding to treatment, come on in the course of pulmonary consumption or local tuberculosis in other parts of the body, and in particular when there are also localized physical signs indicative of intestinal thickening, kinking, or obstruction. If fecal fistula develops and tubercle bacilli are found in the discharge as well as in the stools, the diagnosis is positive.

DIFFERENTIAL. — The discrimination between intestinal tuberculosis and the conditions which resemble it cannot in all cases be made. Two topics, however, demand especial mention—carcinoma and appendicitis.

Cachexia and pain occur as in carcinoma elsewhere. Fever is not a prominent symptom. The temperature is on the contrary often subnormal.

Ribbon-shaped stools, foul-smelling stools in which blood, pus, and necrotic fragments of the neoplasm are found, and the general symptoms of stenosis are suggestive of cancer. Absence of tuberculosis elsewhere, negative findings as to bacilli, and failure of the temperature rise after the injection of tuberculin are of great diagnostic importance.

There are rare cases of tuberculosis of the cæcum in which the process invades the lymphoid tissue of the appendix—tuberculous appendicitis. Primary attacks of appendicitis are so well characterized that the question of tuberculosis does not enter into their consideration. The acute or subacute character of the early symptoms even in the chronic cases would appear in the anamnesis. Of diagnostic importance are other diffuse abdominal symptoms, pain, tenderness, diarrhœa preceding the local phenomena, and the coincidence of tuberculosis in other organs. It is not to be forgotten that an attack of non-specific appendicitis may develop in a tuberculous individual.

E. Tuberculosis of the Brain and Spinal Cord.

Tuberculosis occurs as an acute meningitis which, while chiefly basilar, is almost always also spinal, and constitutes one of the manifestations of the acute form of general or disseminated infection—acute miliary tuberculosis (q.v.); as a chronic meningo-encephalitis due to the development of multiple tubercles, usually within circumscribed limits; and finally as solitary tubercles (see p. 1264).

F. Tuberculosis of the Genito-urinary Organs.

Tuberculosis of the genito-urinary tract is frequent and important. Lesions have been observed in the fœtus, and the occurrence of tuberculous orchitis in very young children suggests the possibility of hereditary transmission. In the preponderating majority of instances the disease is secondary to disease of some distant organ, especially the lungs, and the infection must be ascribed to transmission by way of the blood. In a considerable proportion infection takes place from the peritoneum. Tubal and vesical tuberculosis have, however, been observed in cases of intestinal tuberculosis in which no evidence of the implication of the peritoneum could be found. Less frequently the disease arises by direct infection from the rectum to the bladder, or to the uterus or vagina, in consequence of adhesions and fistula formation. Tuberculous abscesses in the pelvis may be the source of infection of any of the genito-urinary organs. Vertebral tuberculosis may implicate the kidney by direct extension. The possibility of primary tuberculosis as the result of direct infection in sexual intercourse appears very great. Whether or not accidental infection by way of the vagina or urethra may take place from other sources, as infected instruments or syringes, suppositories, or in digital examination, or by transmission from the rectum by way of the clothing has not been fully established. The infection may involve any of the tissues of the genito-urinary system. It often extends rapidly, and in some cases there are manifestations of the disease at different points at the same time.

Tuberculosis of the Kidneys.—The disease may be secondary. In acute general tuberculosis scattered tubercles are present in the substance and upon the surface of the kidneys. In pulmonary tuberculosis there may be scattered nodules, or pyelitis. Primary tuberculosis of the kidney also occurs. In many of the cases the lesions are at the same time present in the kidneys, extending to the pelvis and uterus, and in the bladder, prostate, and seminal vesicles, and the seat of primary invasion is uncertain. Renal tuberculosis is most frequent in middle life but may be met with at any age. Males suffer much more frequently than females.

Symptoms.—The urine contains pus in varying amounts. There is increased frequency of micturition. These symptoms often go on for years without abnormal subjective sensations and with maintenance of the general health. There may be tenderness upon firm pressure. In exceptional cases the kidney may be greatly enlarged, or there may be a pyonephrosis. Under such circumstances there may be a palpable abdominal tumor. The urine is albuminous, and in addition to pus-cells contains epithelium and granular débris. Tube-casts are not very common. Tubercle bacilli are present. Hemorrhage may occur. As the disease advances the other kidney becomes involved, and a tuberculous cachexia with chills, irregular fever, sweating, and emaciation and progressive asthenia ensues. The lungs are implicated and an acute disseminated miliary tuberculosis occurs as a terminal event. Encysted caseous or calcareous masses in the kidney are occasionally found in the post-mortem room and point to the possibility of spontaneous cure.

Diagnosis.—DIRECT.—The above symptoms, associated with the evidence of tuberculosis in the testicle or prostate, or in the tubes or ovaries, the presence of tubercle bacilli in the urine, and a positive reaction to the tuberculin test, justify a positive diagnosis. The differentiation of the urine by catheterization of the ureters renders possible a diagnosis of the kidney affected. The urine may contain bacilli from tuberculous lesions in the bladder or elsewhere in the genito-urinary tract, and the fact that the morphological and tinctorial characters of the smegma bacillus are practically the same as those of the tubercle bacillus is to be borne in mind. The specimen for examination in a doubtful case must be obtained by catheterization under the strictest precautions against contamination, and the possibility that even then smegma bacilli may be accidentally present must not be forgotten. Inoculation tuberculosis caused by the urinary sediment is proof positive of genito-urinary tuberculosis, but not necessarily of tuberculosis of the kidney.

DIFFERENTIAL.—It may be difficult to differentiate tuberculous pyelonephritis from calculous pyelitis. A history of attacks of renal colic, various forms of crystalline sediment and blood-cells in the urine, or actual hemorrhage, are in favor of the latter. Hemorrhage is much less common in tuberculosis of the kidneys.

Suprarenal Capsules.—Tuberculosis of the adrenals with fibrocaseous lesions is the most common anatomical change found in Addison's disease, and may manifest itself by the symptoms of that disease (q.v.).

Tuberculosis of the Ureters and Bladder.—The symptoms of renal tuberculosis are those of cystitis, and infection of the bladder is usually secondary to infection of the kidneys on the one hand, or of the testes, prostate, or seminal vesicles on the other. The process very often invades the ureters from the pelvis of the kidney. Primary tuberculosis of the bladder is a rare affection.

Tuberculosis of the Prostate and Seminal Vesicles.—These organs are frequently the seat of tuberculous growths and caseous nodules. The prostate is often found upon digital examination to be enlarged and nodular. It is sometimes tender. There is great irritability of the bladder, vesical tenesmus, frequent micturition or retention of urine, in which case the use of the catheter is attended with great pain. Tuberculosis of the urethra is rare. It may present the symptoms of stricture.

Tuberculosis of the Testes.—The diagnosis is usually unattended with difficulty because the organ is accessible and the changes are somewhat characteristic. The disease occurs in infants as well as in adults. One or both testicles may be involved. It may be primary, but in most cases is secondary to pulmonary or other visceral or bone tuberculosis. It is frequently associated with tuberculous peritonitis. The tuberculous testicle may be recognized by the enlargement which principally affects the epididymis, pain, tenderness, and only a moderately uneven surface.

The DIFFERENTIAL DIAGNOSIS between tuberculous and syphilitic disease of the testicle may be attended with uncertainty. In the latter, pain and tenderness may be absent, the testicle itself rather small, the epididymis involved, and the surface, owing to the agglomeration and various size of the gummata, is more nodular and uneven.

Tuberculosis of the Fallopian Tubes and Ovaries.—The tubes are very frequently affected. The disease is often primary. There is enlargement with great thickening and infiltration of the walls, upon which, in some cases, irregularities of the surface may be felt. It may occur in children and young girls, and is usually bilateral. The ovaries are secondarily involved. Abscess formation and the extension to the peritoneum are common. Implication of the uterus is extremely rare.

Diagnosis.—DIRECT.—This rests upon the local findings, such as enlargement and irregular thickening of the tubes, evidences of adhesions, signs of peritoneal tuberculosis or pulmonary phthisis, anæmia, loss of weight, fever in the evening upon moderate exertion and at the menstrual period.

DIFFERENTIAL.—Gonorrhœal salpingitis may be present without serious derangement of the general health. The enlargement of the tubes is not attended with the same degree of infiltration or irregularity of the surface, the anamnesis is suggestive, and the presence of gonococci in the discharges conclusive.

Tuberculosis of the Liver, Spleen, Myocardium, Endocardium, and Arteries cannot be recognized with certainty during life. These forms of visceral tuberculosis are therefore rather of anatomical than of clinical interest.

G. Tuberculosis of the Lungs.

Pulmonary Tuberculosis; Phthisis; Consumption.

VARIETIES.—(a) Acute pneumonic phthisis; (b) chronic ulcerative phthisis, and (c) fibroid phthisis.

(a) ACUTE PNEUMONIC PHTHISIS.

According to the distribution of the lesions two types are recognized, the pneumonic and the bronchopneumonic.

The Pneumonic Form.—A single lobe or an entire lung may be involved. This form is much more common in adults than children, and in males than females.

Symptoms.—The onset is usually abrupt, with a chill followed by high fever, pain in the side, cough, and expectoration, at first scanty and mucoid, later more abundant, often frothy and blood-stained. The attack frequently occurs in the midst of apparent health; occasionally during the course of an apparently ordinary mild influenza or "cold," and sometimes in an individual who has a tuberculous lesion regarded as obsolescent. The respiration is rapid and dyspnœa may be urgent; the pulse is frequent and variable. The physical signs are those of croupous pneumonia, feeble vesicular murmur, with crepitus, later dulness, increased vocal fremitus, and bronchial breathing. They correspond to the limits of a lobe or to a whole lung, and when, as is often the case, they are also, in the course of some days, found upon the opposite side the clinical picture is that of a double pneumonia.

Diagnosis.—The DIRECT DIAGNOSIS rests upon the course of the attack and the finding of tubercle bacilli in the sputa. The latter have been observed as early as the fourth day. As a rule their presence is not noted until later. Fibres of elastic tissue constitute the signs of necrosis of the pulmonary structure. The following clinical manifestations are suggestive and should arouse a suspicion as to the character of the process: hereditary predisposition to tuberculous infection; individual history of tuberculous infection which may have remained subacute or become quiescent; physical depression preceding the outbreak, especially if accompanied by cough and expectoration; an irregular temperature range conforming rather to the remittent than the continuous type; recurrent chills; circumscribed patches of high-pitched, ringing, coarse crepitant râles persisting for several days with but little change; a mucopurulent greenish expectoration, and extremely feeble breath sounds over the affected region.

The DIFFERENTIAL DIAGNOSIS between acute pneumonic tuberculosis and croupous pneumonia cannot be made in a large proportion of the cases during the first week. There is, in truth, usually no suspicion that the case is not one of ordinary pneumonia until even a longer time has elapsed.

Prognosis.—The outlook is in the highest degree unfavorable. Death has occurred as early as the sixth day, more commonly after three or four weeks, or as late as the second or third month. In a limited number of cases the acute symptoms gradually subside and the case becomes one of chronic phthisis.

The Bronchopneumonic Form.—The lesions are those of an acute caseous bronchopneumonia. Groups of lobules are affected, with crepitant tissue intervening, but extensive areas or even an entire lobe may be involved.

A second form is due to the aspiration of blood and the contents of tuberculous cavities into the finer bronchial tubes during hæmoptysis—tuberculous aspiration pneumonia. This condition may follow early hæmoptysis, which has not been preceded by marked symptoms, or occur after hæmoptysis in the course of a chronic tuberculosis.

In a third form the caseous bronchopneumonia involves lobules at both apices and in other parts of the lungs, causing patches of consolidation varying in diameter from 1 to 3 or 4 cm. and sometimes scattered uniformly throughout both lungs.

Symptoms.—These are the forms which constitute a majority of the cases of acute pulmonary tuberculosis—*phthisis florida* or *galloping consumption.* They are common in adults but far more common in children. The clinical picture varies greatly. In adults the disease may develop in persons apparently well or in those who have been failing in weight and strength. The onset is rapid but not abrupt. There are irregular chills, fever of hectic type, sweating, loss of appetite, cough, and expectoration which is usually slight. Blood spitting is sometimes the first event to attract attention. The pulse and respiration frequency are high, and loss of weight and strength is rapid and progressive. The physical signs may be at first obscure, but presently diminished expansion, patchy dulness, especially at an apex, vesiculobronchial respiration, and moist crepitant and small mucous râles occur. The disease affects one, more commonly both lungs. Tubercle bacilli and fibres of elastic tissue are present in the sputa, often at an early date. The symptoms become more intense, and in the course of two or three weeks in the more acute cases the patient falls into the so-called typhoid state, with stupor, delirium, dry tongue, and high fever. Meanwhile the physical signs, more extensive dulness, bronchial breathing, high-pitched and coarser râles, indicate the extension and progress of the lesions. In some cases, however, the signs are obscured by the development of areas of collateral emphysema. Softening may occur with cavity formation and corresponding changes in the physical signs. In children this form of pulmonary tuberculosis may arise as an independent disease. Much more commonly it follows an acute infection, especially measles and pertussis. In a majority of the cases the bronchopneumonia which occurs as a sequel to diseases of this group is tuberculous.

Diagnosis.—The DIRECT DIAGNOSIS of acute bronchopneumonic phthisis rests upon the association of the foregoing symptoms and physical signs with the presence of elastic fibres and tubercle bacilli in the sputa.

The DIFFERENTIAL DIAGNOSIS between tuberculous and non-tuberculous bronchopneumonia is, in the early stages of the disease, and especially in children, often impossible. Later the presence of elastic-tissue elements and tubercle bacilli are decisive. Meanwhile the anamnesis is important. A hereditary predisposition, association with individuals who are tuberculous, or dwelling in an infected house, or enlarged superficial lymphatics, or a history of symptoms or signs indicative of enlarged bronchial glands is important. The signs of marked apical lesions are highly suggestive of tuberculous disease, but diffuse tuberculous bronchopneumonia may occur without marked apex consolidation.

Prognosis.—The outlook is in the highest degree unfavorable. In adults death may occur in the course of three or four weeks; in children within a few days. There are cases, however, in which the disease runs a somewhat more protracted course, and a limited number, both in adults and children, in which after the gravest symptoms the condition of the patient undergoes some improvement and the case gradually passes into one of chronic phthisis.

(b) CHRONIC ULCERATIVE PHTHISIS.

This is the common form of chronic pulmonary tuberculosis.

The lesions vary greatly in kind, distribution, and extent. They comprise nodular and miliary tubercles, tuberculous bronchopneumonia, pneumonic inflammation of the vesicular structure surrounding the tubercles, frequently presenting the appearance of ordinary red hepatization, sometimes the more uniform diffuse tuberculous infiltration, cavities of various size, together with collateral emphysema and changes in the bronchi and bronchial glands and in the pleura, with firm, thick adhesions or effusion, which may be serofibrinous, purulent, or hemorrhagic. The tendency is, (a) to caseation, softening, ulceration, necrosis, and cavity formation, and (b) to sclerosis. The latter process may result in the formation of a limiting membrane, by which the lesion is encapsulated, or, when extensive, in traction deformities of the chest, and bronchiectasis.

The fact that the primary lesion or lesions in pulmonary tuberculosis of the chronic ulcerative type are local and circumscribed is of the greatest practical importance both in diagnosis and prognosis; first, because it underlies the clinical division of the cases into incipient and advanced, and second, because cases referable to the first group are mostly amenable to treatment.

The distribution of the lesions is in a majority of the cases as follows: The earliest lesions are situated, not at the extreme apex of the lung, but 2 to 4 cm. below it and nearer the posterior and lateral surfaces than the anterior surfaces. Extension from this point is downward and forward, the upper lobe being progressively involved in regions corresponding to the first, second, and third interspaces, and spreading upon both sides of the midclavicular line. Less commonly the primary lesion is found in the upper lobe at a point corresponding to the first and second interspaces below the outer third of the clavicle. As the process extends downward, the anterolateral region of the lobe is involved. Invasion of the middle lobe of the right lung is usually by extension from the upper lobe. Secondary implication of the lower lobe begins at a point 2 to 4 cm. below its apex at the level of the fifth dorsal spine, and extends downward and outward in a line roughly corresponding to the inner border of the scapula when the patient's hand is placed upon the opposite shoulder and the elbow raised as high as possible. In the course of time the upper lobe of the opposite lung usually becomes involved, the earliest lesions appearing a short distance below the actual apex and rapidly becoming diffused. The right upper lobe is first involved somewhat more commonly than the left. Primary implication of the base is rare.

In advanced cases miliary tuberculosis, visceral tuberculosis involving various organs, amyloid disease, and fatty liver occur.

The extension of the lesions is, (a) peripherally by the direct invasion of contiguous tissue; (b) radially by means of the lymph current; (c) by conveyance along the bronchial system, (i) in the direction of adjacent or distant vesicular structures—inhalation, insufflation; (ii) in the direction of the upper air-passages—laryngeal ulceration; (d) by transference, *e.g.*, to the digestive tract—secondary pharyngeal, lingual, or intestinal tuberculosis; by dissemination, as in the case of the rupture of a gland or other encapsulated focus into a serous cavity, or a blood-vessel—acute miliary tuberculosis.

The progress of the lesions is variable. On the one hand, infiltration, caseation, softening, ulceration, necrosis, proceeding at different rates in different foci; while, on the other hand, sclerotic changes encapsulate and limit the advance of the disease and tend to circumscribe the process.

Symptoms.—As the primary infection is local, there is usually a period of latency. *The patient is tuberculous before he is consumptive, and, in the fortunate cases, he may be tuberculous without ever becoming consumptive.*

The MODE OF ONSET is determined by the degree of activity of the tuberculous process and the nature of the reaction of the infected individual. It may be characterized by latency, with indefinite symptoms not suggestive of pulmonary disease, or masked by the symptoms of grave disease in other organs, tuberculous or non-tuberculous.

Gastro-intestinal Symptoms.—Loss of appetite, gastric irritability and vomiting, acid eructations frequently precede the pulmonary symptoms for a considerable time. The cough is regarded as "a stomach cough."

Anæmia.—In children and adolescents, especially young girls, there is early chloro-anæmia with pallor, progressive weakness, palpitation and headache upon exertion, and slight afternoon fever. Menstrual irregularities, especially amenorrhœa, are suggestive.

Ague-like Fever.—Constitutional symptoms, recurring chills, fever, and sweating characterize the onset in a considerable group of cases. When such paroxysms recur with regularity and with only slight cough and expectoration, especially in a malarious region or in an individual who has previously suffered from ague, a false diagnosis may readily be made.

Pleurisy.—The early phenomena may be those of a persistent dry pleurisy, the signs of which are sometimes restricted to the apex, sometimes more extended. In other cases the impairment of health begins with pleural effusion. The resorption or aspiration of the fluid is sooner or later followed by the signs of consolidation in an upper lobe, and the symptoms of phthisis. Many of these cases are, in fact, pleurogenous, with secondary pulmonary infection, and the early dry cough is that of pleural irritation. In some cases the lung lesions rapidly develop; in others an interval of weeks, months, or even years may occur.

Hæmoptysis.—Blood spitting may be the first indication of the disease. An abundant hemorrhage is sometimes followed by the rapid development of the signs of a diffuse tuberculosis. In other cases hæmoptysis recurs from time to time before the positive physical signs of pulmonary disease can be recognized. It is probable that the local lesions almost always antedate the pulmonary hemorrhage.

Bronchitis.—The great majority of cases begin with the signs of a catarrhal bronchitis. The patients often suffer from nasopharyngeal catarrh and manifest an especial tendency to "catch cold." At length the cough becomes persistent, there is habitual expectoration, especially in the morning, and upon examination the râles, which are heard widely er the chest, are found to be more abundant and moist and of higher tch over the upper part of one lung, where there is also relative dulness d deficient expansion.

Chronic Bronchitis and Emphysema.—The terminal tuberculosis so mmon in these conditions is usually masked for a considerable time by e symptoms and signs of the primary condition. This is especially the se when there are asthmatic symptoms.

Laryngitis.—The symptoms of pulmonary phthisis are frequently eceded by hoarseness, occasional aphonia, and a laryngeal cough. It is obable that, in the majority of these cases, tuberculous lesions already ist in the lungs.

Tuberculosis of the cervical and axillary lymph-glands may precede the velopment of pulmonary tuberculosis for a long time, or coexist with iescent lesions in the lungs.

Stages.—The attempt to divide the course of the attack into a stage the growth and development of tubercles, a stage of caseation and ftening, and a stage of cavity formation has fortunately been abandoned. the first place, as new foci of disease are constantly forming in advancing ses, all three of these anatomical conditions are frequently present at the me time; secondly, they do not correspond with definite clinical periods, d, finally, a patient in the so-called third stage, with signs of cavity rmation, is often in a more favorable condition, with better prospect r the arrest of his disease, than another in the first stage, with extensive d rapidly advancing infiltration or diffuse foci.

The following schema was adopted by the National Association for e Study and Prevention of Tuberculosis in 1905:

Incipient (favorable)	Slight initial lesion in the form of infiltration limited to the apex or a small part of one lobe. No tuberculous complications. Slight or no constitutional symptoms (particularly including gastric or intestinal disturbance or rapid loss of weight). Slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours, especially after rest. Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.
Moderately advanced	No marked impairment of function either local or constitutional. Localized consolidation moderate in extent with little or no evidence of destruction of tissue; Or disseminated fibroid deposits. No serious complications.
Far advanced	Marked impairment of function, local and constitutional. Localized consolidation intense; Or disseminated areas of softening; Or serious complications.
Acute miliary tuberculosis	
Unimproved	All essential symptoms and signs unabated or increased.
Improved	Constitutional symptoms lessened or entirely absent; physical signs improved or unchanged; cough and expectoration with bacilli usually present.
Arrested	Absence of all constitutional symptoms; expectoration and bacilli may or may not be present; physical signs stationary or retrogressive; the foregoing conditions to have existed for at least two months.
Apparently cured	All constitutional symptoms and expectoration with bacilli absent for a period of three months; the physical signs to be those of a healed lesion.
Cured	All constitutional symptoms and expectoration with bacilli absent for a period of two years under ordinary conditions of life.

Trudeau's classification is as follows:

1. *Incipient.*—Cases in which both the physical and rational signs point to but slight local and constitutional involvement.
2. *Advanced.*—Cases in which the localized disease process is either extensive or in an advanced stage, or where, with a comparatively slight amount of pulmonary involvement, the rational signs point to grave constitutional impairment or to some complication.
3. *Far Advanced.*—Cases in which both the rational and physical signs warrant the term.
4. *Apparently Cured.*—Cases in which the rational signs of phthisis and the bacilli in the expectoration have been absent for at least three months or who have no expectoration at all; any abnormal physical signs remaining being interpreted as indicative of a healed lesion.
5. *Arrested.*—Cases in which cough, expectoration, and bacilli are still present, but in which all constitutional disturbance has disappeared for several months; the physical signs being interpreted as indicative of a retrogressive or arrested process.

"Closed" and "Open" Pulmonary Tuberculosis.—Too much stress has been laid upon the importance of tubercle bacilli in the sputa in the early diagnosis. These organisms do not appear until after the caseation and softening of a tuberculous lesion situated near a bronchus or bronchiolus, into which tuberculous material finds its way by the necrosis of the intervening tissue. The period prior to this event, which may extend over weeks or months, or, in extreme cases, over years, is known as the "closed period"; that which follows as the "open period." The general recognition of this distinction is desirable.

Symptoms of Incipient Pulmonary Tuberculosis.—The greater number of these have already been described under the heading "mode of onset." The association of hæmic, circulatory, digestive, and nervous derangements is especially important.

The *pulse* is either persistently frequent but regular, or subject to abnormal acceleration upon physical effort or mental excitement. The *temperature* shows slight elevation upon exertion, after meals, and before and during menstruation. The observations must be taken every two hours during the day, while the patient is in repose. Subfebrile ranges—99.5° F.—are significant. *Chest pains* are common. They are of two kinds, pleural pain over the seat of a lesion, and a dull shoulder pain extending down the arm and sometimes mistaken for rheumatism. The *cough* is frequently short and dry, a troublesome hacking brought on by exertion or excitement or changes of external temperature. Very often it occurs only on rising in the morning and persists in paroxysms until a small, tough mucoid mass is expectorated, after which it is absent for the rest of the day. *The Sputum.*—There is little characteristic in the expectorated material. It is usually at this stage of the disease merely a grayish sago-like mucus, containing alveolar cells which have undergone the myelin degeneration. In the closed stage tubercle bacilli are absent, though one or two may, in rare instances, be found as the result of inhalation. Repeated examinations are necessary. Their continuous presence in the sputum is the positive sign of tuberculosis in the open stage. The examination may yield negative results for long periods in cases of quiescent limited upper lobe lesions, and then, after an attack of some acute affection, as influenza, or, in the midst of apparent health, bacilli may appear suddenly and last a short time—transient open tuberculosis. Elastic fibres are not often encountered in the incipient stages.

Hæmoptysis.—This accident occurs in about 70 per cent. of all cases of phthisis at some period in the course of the disease. The hæmoptysis which occurs in the incipient stage differs from that in the advanced stages in being, as a rule, slight, recurrent, and due to oozing from patches of acute congestion surrounding closed tuberculous foci, or to superficial erosions of bronchial mucosa. In advanced phthisis the bleeding is due to the erosion of a vessel in the wall of a cavity, or the rupture of an aneurism of a branch of the pulmonary artery. It is usually profuse and not rarely fatal. The expectorated blood in early hæmoptysis—closed tuberculosis—does not usually contain tubercle bacilli; that in the advanced stages is often followed by expectoration containing those organisms. Large initial blood spittings may, in rare instances, usher in the open stage and be associated with bacillary sputum.

General Nutrition and Weight.—The toxins which give rise to anæmia, vasomotor derangements, pseudodyspepsia, fever, and nervous erethism interfere with nutritive processes and cause loss of weight which is often rapid and striking.

Associated Diseases.—The patients are especially prone to catarrhal and other inflammatory outbreaks. Coryza, laryngitis, bronchitis, pneumonia, and pleurisy are common and may recur repeatedly in the same patient.

Hoarseness, due to subacute laryngeal catarrh with slight abductor paresis, may be an early symptom. Actual paralysis of the recurrent is less common. It may be due to pleural adhesions or to pressure upon the recurrent by tuberculous lymph-glands. It much more frequently occurs upon the left side. Phenomena of inferior importance are unequal dilatation of the pupils, a reddish or bluish gingival line, slight or transient enlargement of the thyroid gland, and albuminuria.

The Physical Signs in Incipient Pulmonary Tuberculosis.—*Inspection* reveals very early a retarded and slightly diminished respiratory excursus in the infraclavicular region of the affected side. This sign may, in some cases, be earlier detected by palpation. The vocal fremitus may also be slightly increased. *Percussion* may show quite early relative dulness, often slight but recognizable by the higher pitch and shorter duration of the sound, and a slightly tympanitic quality. *Auscultation* yields even more suggestive signs. There is an early deviation from the normal type of breathing. The first change consists in the development of the quality described as rough. The inspiratory murmur is enfeebled. Cog-wheel or interrupted breathing is occasionally heard in the region immediately adjacent to and below the portion of the lung involved. This may, however, occur in other conditions. Moist crepitant and small mucous râles are early signs, but in many cases they remain long absent. Râles, not heard upon full inspiration, even full inspiration after coughing, may in some cases be elicited by a full inspiration followed by forced expiration, with cough at the end of the latter. The rough breathing is presently replaced by vesiculobronchial respiration which, as the lesion progresses, becomes bronchovesicular and, later, as consolidation becomes complete—advanced stage—bronchial. Pleural friction sounds of varying quality and intensity may often be heard over the affected region. They are sometimes transient, sometimes persistent. Basal friction sounds are also occasionally heard in the incipient stage. Less frequent but very suggestive when present, is a subclavian systolic murmur, more common, as a rule, upon inspiration, though occasionally heard with expiration. This loud systolic whiff, due to traction upon the vessel wall by pleural adhesions, is a very striking phenomenon. Extension of the absolute cardiac dulness to the right or left, as the case may be, constitutes an important sign.

The Diagnosis of Incipient Pulmonary Tuberculosis. — DIRECT DIAGNOSIS. —In the absence of cough and expectoration a positive diagnosis can rarely be made. When these phenomena are present, and especially when tubercle bacilli are found, the question as to the nature of the process is at once settled. From the standpoint of therapeutics the recognition of phthisis in the closed stage is of such importance that, in a suspected case, a provisional diagnosis constitutes a motive for immediate and systematic treatment. This provisional diagnosis rests not upon any single rational symptom or physical sign of the stage of incipiency, but upon the association of several of them in an individual in whom no other pathological process by which to explain them can be demonstrated. The anamnesis is important. Family predisposition, close habitual association with tuberculous persons, an unfavorable occupation, an unhygienic life may appear as etiological factors, but their absence has only a negative value.

The bodily conformation may be misleading. The classical *habitus phthisicus*—the phthinoid or paralytic chest—is symptomatic of advanced, not of incipient phthisis. Recurrent hoarseness, bronchitis, anæmia, dyspepsia, loss of weight, fever, and hæmoptysis are symptoms of great moment. Among the physical signs, diminished and retarded respiratory excursus of that part of the chest corresponding to the limited lesions, diminished resonance with a faint tympanitic quality, rough or vesiculobronchial respiration, and a few moist, clicking râles or a prolonged whizzing râle at the end of the first two or three inspirations are highly significant. It is to be remembered that in slowly advancing lesions the signs may be obscured by collateral emphysema. Not less significant is the localization of those signs in an infraclavicular or axillary region. The variation of auscultatory signs must be remembered—the fact that râles are sometimes heard only after cough, the frequent temporary disappearance of crepitus after several deep inspirations, the tendency of râles to disappear late in the day and in dry weather, and the intensification of auscultatory phenomena during menstruation. Rosenberger claims that tubercle bacilli can be found in the blood even when absent in the sputum. In any case in which the symptoms and signs warrant a provisional diagnosis, the patient must be carefully instructed as to his mode of life, the necessity of keeping himself under systematic observation, and the importance of the repetition of the examination at stated intervals.

DIFFERENTIAL DIAGNOSIS.—The mode of onset is to be considered. The patient is in failing health; has he the symptoms and signs of phthisis, latent or marked? Are his symptoms those which the toxins of tuberculosis cause? Superficial examination and hasty observation will not discover the answer to these questions. Above all, the practitioner must avoid the delusion that every paroxysmal fever is malarial and every cough in a dyspeptic a "stomach cough." *Pleurisy* is highly suspicious. Probably two-thirds of the cases of persistent fibrinous pleurisy or pleural effusion ultimately become tuberculous. *Malaria* may be recognized by the blood examination. *Hæmoptysis* is common in mitral disease, especially stenosis, and many cases of valvular disease of the heart are diagnosticated phthisis. This error may be avoided by a routine physical examination, which must in every case include the heart. *Cervico-axillary adenitis* may long coexist with a fair degree of health. It is well, however, to watch the infraclavicular regions, especially upon the side in which the tuberculous glands are situated. *Laryngitis.*—Persistent hoarseness with swelling of the arytenoids and slight adductor paresis is frequently the phenomenon of pulmonary phthisis. *Recurrent catarrhal and inflammatory affections* in the respiratory tract are ominous, especially when there are hereditary or personal predisposing factors to tuberculous infection. Infraclavicular or axillary localizations are highly suggestive. The patient should be forewarned and placed under treatment. Such reassuring phrases as "local bronchitis" and "spot on the lung" are worse than obsolete; they are dishonest.

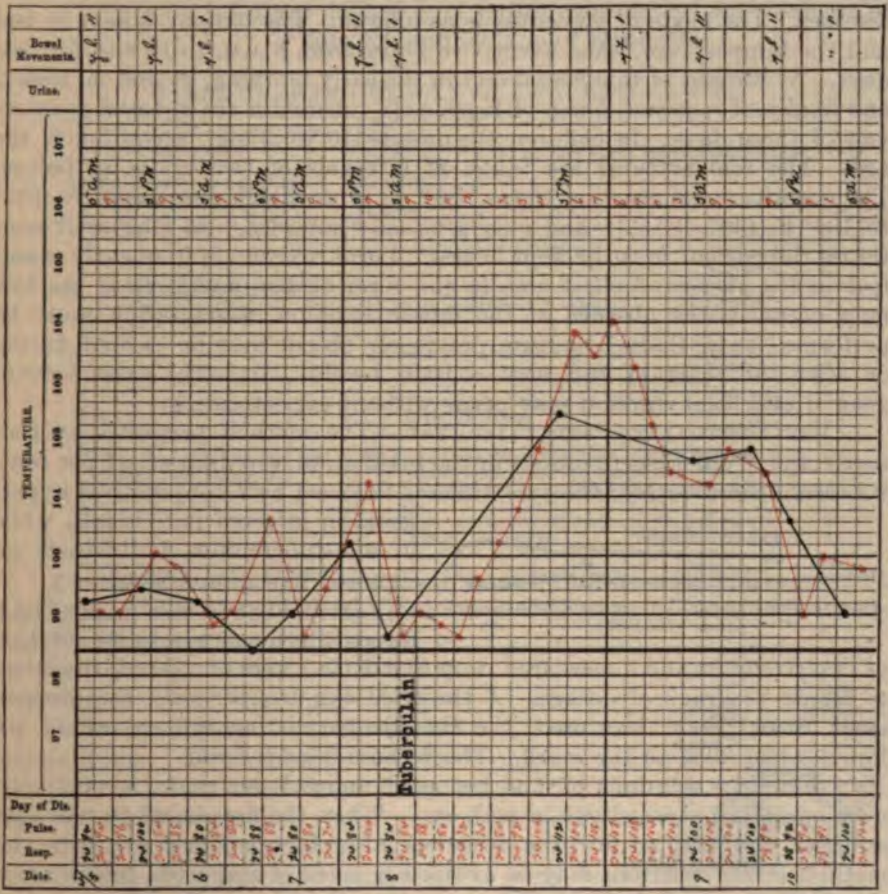


FIG. 277.—Subcutaneous tuberculin test. Positive reaction.

THE TUBERCULIN TEST.—If a case remains doubtful, or if, for personal reasons, delay must be avoided, tuberculin may be employed. To the objection as to danger, it may be affirmed that in the hands of innumerable careful observers its cautious use has not been followed by untoward effects. To the objection as to results, it is to be admitted that it has sometimes failed in cases of demonstrated tuberculosis, and that some degree of reaction has occurred in non-tuberculous cases. No one, however, has reported an intense reaction to tuberculin in a healthy person or in other diseases. The mode of procedure is as follows: The patient is kept in bed and the temperature taken every two hours from 8 A.M. to 10 P.M. for two days. At the end of this period and in the early morning, at first .5, then 2 and finally, if required, 5 mg. of Koch's old tuberculin are injected at intervals of three days. In children the dose is .10 to .5 mg., according to the age. The temperatures are taken at intervals of two hours as before. The constitutional reaction shows itself in fever which rises rapidly—102° to 104° F. (39°–40° C.)—and only gradually subsides. So long as it continues the patient must be kept in bed. Local reaction is frequently manifest in the suspected chest area in the form of fine moist râles. As this may occur in the absence of the febrile reaction, auscultation must be performed twice daily. Sputum previously absent may be ejected during or after the reaction and may contain bacilli. In other cases intense general and local reaction may occur without expectoration.

THE OPHTHALMOTUBERCULIN TEST.—The method proposed by Calmette and Wolff-Eisner consists in the instillation of a drop of a 1 per cent. tuberculin solution into the eye, which is followed by a conjunctival hyperæmia in infected individuals, while in non-tuberculous individuals no signs of irritation follow.

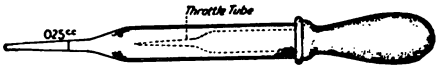


FIG. 278.—Eye-dropper with throttle for measured drop.—Baldwin.

In Baldwin's clinical studies a filtered sterile normal saline solution of two strengths and a measured drop (0.025 c.c.) were employed, the latter to insure accuracy of dosage. A throttled eye-dropper with a calibrated mark to 0.025 c.c. was used, the throttle permitting the control of the liquid when filled to the mark. The technic is as follows:

"The two solutions of 0.33 per cent. and 0.5 per cent., respectively, were employed successively in each eye. When the first failed to react the stronger was instilled into the other eye after forty-eight hours. By this method severe reactions may be avoided in cases of suspected tuberculosis, though if time failed the stronger solution might be used without serious discomfort being anticipated should a marked reaction follow. In order to avoid any danger of contamination the solutions were sealed in glass tubes containing three or four drops and then boiled. These can easily be broken in a piece of gauze or cotton at a file mark. The eye-dropper is then inserted, after being cleansed with alcohol and sterile saline solution. Care is used to prevent the introduction of spicules of glass. The solution should be warmed in cold weather.

"The lid of one eye is pulled down and the measured drop instilled as with any other fluid by holding the eye-dropper parallel to the eye, but care should be observed that it does not overflow on the cheek; this is easily accomplished by holding the lid down until the drop is distributed about the sac."

Baldwin's Scheme for Recording Reactions.

Negative.—No difference in color when lower eyelids are pulled down and compared.

Doubtful. Slight difference with redness of caruncle.

Positive. + = Distinct palpebral redness with secretion.
" ++ = Ocular and palpebral redness with secretion well marked.
" +++ = Deep injection of entire conjunctiva with œdema of lids, photophobia, and secretion.

TABLE SHOWING RESULT IN 137 CASES—Baldwin.

I. Pulmonary tuberculosis. No. cases, 45.		Reacted....42 Doubtful... 1 Negative... 2	(1 miliary and 1 tuberculin-treated).
II. Pulmonary or other tuberculosis; healed from 1 to 17 years. No. cases, 9.		Reacted.... 8 Doubtful... 1	(17 years).
III. Pulmonary or other tuberculosis suspected. No. cases, 26.	(a) From history (14 cases).........	Reacted.... 4 Doubtful... 4 Negative... 6	
	(b) From symptoms (8 cases).......	Reacted.... 2 Negative... 6	
	(c) From physical signs (4 cases)....	Reacted.... 2 Negative... 2	
IV. Apparently healthy persons. No. cases, 57.	(d) With family history of tuberculosis (18 cases).	Reacted.... 8 Doubtful... 1 Negative... 9	
	(e) Constantly associated with tuberculous doctors, nurses, husbands and wives of invalids (18 cases).	Reacted.... 6 Negative...12	
	(f) With no family history or exposure (21 cases).	Reacted.... 2 Negative...19	

Contraindications.—These include diseases of the conjunctiva, eyelids, and cornea, and of the internal structures of the eye. Undue exposure to dust, smoke, or strong light should be avoided during the test. The test is superfluous when the diagnosis can be made by physical or microscopic examination. The repetition of the test is not unattended with danger due to sensitization.

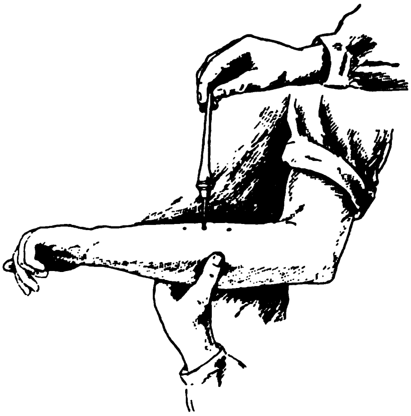


Fig. 279.—V Pirquet's method.

Cutaneous Tuberculin Reaction.—Von Pirquet, who suggested this test, employed at first a 25 per cent. solution of old tuberculin, but subsequently used the undiluted old tuberculin. The inoculation is performed on the forearm with a chisel-shaped instrument, the skin having been previously thoroughly cleansed with ether. Any suitable instrument may, however, be employed, and any convenient part of the body selected.

Positive Reaction.—If a papule of at least 5 millimetres in diameter develops at the point of vaccination in twenty-four hours, the patient may be regarded as infected with tuberculosis. As a general rule the more active the tuberculous process the more intense and extensive the cutaneous reaction. Very intense reactions occur in glandular tuberculosis and in these cases inflammatory changes in the skin beyond the borders of the papule often occur—scrofulous reaction. The signs of reaction occur in periods varying from two or three hours to several days and are later in proportion to the degree of dilution of the tuberculin. No direct information as to the period at which the infection has taken place, namely, as to whether it is old or recent, its situation in the body or the extent of the lesions, is afforded by the cutaneous test.

Negative Reaction.—Failure may be due to absence of tuberculous infection or to various causes, among which von Pirquet especially enumerates the following: (1) relative insusceptibility, which is rare in early childhood but not infrequent in older persons having circumscribed lesions which are encapsulated; (2) failure of the capacity for reaction in the last stages of tuberculosis; (3) loss of the capacity to react in immunity produced by tuberculization, either by progressively increasing doses or by a recent single dose of larger amount; (4) temporary disappearance of the capacity of reaction during an attack of measles.

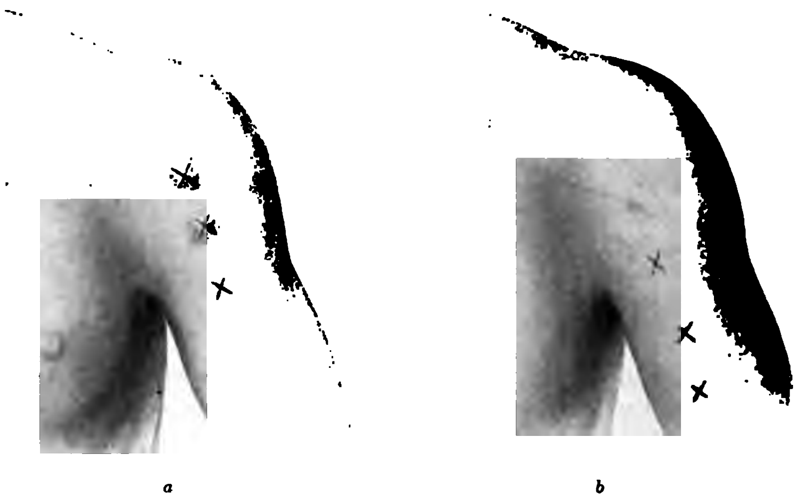


FIG. 280.—Petruschky's method: *a*, lower vaccination 1:10 negative, upper two 1:0 positive; *b*, lower vaccination 1:10 and upper two 1:0 negative.

In a considerable proportion of cases the failure of the test is, therefore, due to relative insusceptibility, and it has been shown that well-marked "secondary positive reaction" may be obtained upon the repetition of the test in these cases. A primary reaction is, however, the sign of an active tuberculous process. Petruschky makes, on the upper arm with the point of a cannula, cross-lined vaccinations with old tuberculin in dilutions of 1 to 10, 1 to 5, or 1 to 0. He finds no reaction in fully cured cases, and that the test is without danger and does not give rise to any important symptoms in any case. Petruschky regards this procedure as an important means of early diagnosis.

TUBERCULIN INUNCTION—MORO'S TEST.—This diagnostic procedure, described as "a diagnostic measure without rupture of continuity of the skin," consists in the inunction into the skin of the chest or abdomen, over an area of 4 square inches, of a mass about the size of a pea of an ointment composed of 5 c.c. of Koch's old tuberculin rubbed up with 5 grammes of anhydrous wool fat. Absorption takes place slowly, and on the following day, or, more commonly, not until the second day, in cases of previous or present tuberculous infection, a positive reaction shows itself in the appearance of small papules in the area of inunction or its immediate vicinity. These lesions vary in number and color from a few pale papules to a numerous, thick-set crop that are very red. The skin may be reddened and the seat of some itching. These cutaneous lesions wholly disappear in the course of a week. They are not attended by other local or constitutional phenomena. The test is negative when the skin shows no changes of any kind.

THE OPSONIC METHOD.—A. E. Wright and S. T. Reid,[1] in a communication "On the Possibility of Determining the Presence or Absence of Tuberculous Infection by the Examination of a Patient's Blood and Tissue Fluids," have arrived at the following conclusions:

"(1) Conclusions which can be arrived at when we have at disposal the results of a series of measurements: (a) Where a series of measurements of the opsonic power of the blood reveals a persistently low opsonic power with respect to the tubercle bacillus, it may be inferred, in the case where there is evidence of a localized bacterial infection which suggests tuberculosis, that the infection in question is tubercular in character. (b) Where repeated examination reveals a persistently normal opsonic power with respect to the tubercle bacillus, the diagnosis of tubercle may, with probability, be excluded."

"(2) Conclusions which may be arrived at where we have at disposal the result of an isolated blood examination: (a) Where an isolated blood examination reveals that the tuberculo-opsonic power of the blood is low, we may—according as we have evidence of a localized bacterial infection or of constitutional disturbance—infer with probability that we are dealing with tuberculosis—in the former case with a localized tubercular infection, in the latter with an active systemic infection. (b) Where an isolated blood examination reveals that the tuberculo-opsonic power of the blood is high, we may infer that we have to deal with a systemic tuberculous infection which is active, or has recently been active. (c) Where the tuberculo-opsonic power is found normal, or nearly normal, while there are symptoms which suggest tuberculosis, we are not warranted, apart from the further test described below, in arriving at a positive or a negative diagnosis."

The further criterion to which reference was made in the preceding paragraph is the following: When a serum, after it has been heated to 60° C. for ten minutes, is found to retain, in any considerable measure, its power of inciting phagocytosis, we may conclude that "incitor elements" have been elaborated in the organism, either in response to autoinoculations occurring spontaneously in the course of tubercular infection, or, as the case may be, under the artificial stimulus supplied by the inoculation of tubercle vaccine.

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[1] Proceedings of the Royal Society, B Vol. LXXVII, 1906.

Iodine Test.—The iodine salts, and especially potassium iodide in moderate doses, may produce the physical signs of a local catarrh over a suspected area (Striker). The signs of consolidation may also become more definite. In the expectoration, which almost always follows, tubercle bacilli are frequently found.

X-ray Examination.—The results obtained are less definite than in the advanced stages of the disease. The complicated apparatus and great technical skill required also stand in the way of the general employment of this method in the diagnosis of pulmonary tuberculosis in the incipient stage.

The pulmonary tuberculosis which so often occurs as a terminal condition in pneumonoconiosis, chronic bronchitis, and emphysema is not usually recognizable in the incipient stage—masked tuberculosis. The symptoms of the primary disease are not so much altered as intensified. As the process advances tubercle bacilli previously absent may be found in the sputa.

Symptoms of Moderately and Far Advanced Pulmonary Tuberculosis.—There is no definite border-line between the stages. The difficulties in diagnosis disappear. The symptoms and signs become definite and characteristic. Any tyro can interpret the clinical picture.

1. Pulmonary Symptoms.—*Cough* may be slight but is usually prominent and annoying. After cavity formation it is commonly paroxysmal; with laryngeal involvement, husky and brassy. *Sputum.*—The expectoration is variable. There may be little or none, even when cough, fever, and rapid wasting are associated with the physical signs of extensive consolidation. The mucoid expectoration of the early period presently shows scattered grayish or grayish-green purulent masses in which tubercle bacilli and elastic fibres may be found. With softening the expectoration becomes more profuse and distinctly purulent. Nummular sputa are often present after cavities have formed. The sputa sometimes contain calcareous masses varying in diameter from 1 mm. to 1 or 2 cm., often of irregular shape. They are the result of the deposition of lime salts in circumscribed caseous masses. They find their way into a bronchus by the ulceration and necrosis of the intervening tissue. *Hæmoptysis.*—The amount varies from a trace to 500–750 c.c. In a majority of the attacks it does not exceed 15 c.c. It is in many cases repeated, and a hemorrhagic form of phthisis has been described. Hemorrhage into a large cavity may prove fatal without any blood being expectorated. Bacilli and elastic fibres may sometimes be discovered in the clots. After a day or two small black clots and blood-casts of the smaller bronchi are often coughed up and an access of fever may occur. *Dyspnœa.*—The respiration is not usually increased except upon exertion.

2. Constitutional Symptoms.—*Fever* is an important initial symptom. It may be remittent in type, or intermittent and paroxysmal, with ague-like periodicity. The temperature is an important indication of the progress of the disease. The periods of quiescence are afebrile and marked by gain in weight, while those of activity are accompanied by fever and loss of flesh. There are, however, rare cases in which, with advancing lesions, pyrexia is absent. The fever of the incipient stage is sometimes continuous, with slight daily remissions and exacerbations; that of the

moderately advanced stage corresponds to the activity of the process, and when present is of remittent or intermittent type, tending to subside altogether when the patient is kept at rest; while that of the far advanced period of the disease, with ulceration, necrosis, and the formation of cavities, is septic in character,—so-called hectic,—the range of temperature being subnormal—95°-96° F. (35°-35.5° C.)—in the morning between 10 A.M. and noon, and steadily rising to a maximum of 104°-105° F. (40°-40.5° C.) between 6 and 11 P.M. Colliquative sweating frequently attends the morning fall of temperature. The measurements should be made at two-hourly periods in order to ascertain the actual minima and maxima. Inverse temperatures are sometimes observed. *Sweating.*—Profuse sweats may occur, not only toward morning, but at any time at which the patient sleeps. They may occur early but are much more common in the far advanced cases. *Circulation.*—The pulse-frequency is increased and variable. It usually rises with the fever. The pulse is often large, soft, and compressible. As the sign of vasomotor paresis, capillary and venous pulsation may sometimes be seen. *Loss of weight* is a marked symptom. It is often rapid and extreme—*consumption; phthisis.* During periods of quiescence it is often arrested, and in favorable cases weight is regained. In rare cases increase of weight occurs in the absence of other signs of improvement. *Anorexia, vomiting, intractable diarrhœa, œdema* of the legs and feet with or without albuminuria. are common in the stadium ultimum. *Peripheral neuritis,* showing itself in extensor palsy of the wrists, more commonly the feet, sometimes occurs. The mental condition in the terminal dyscrasia is often remarkable—*spes phthisica.* The patients up to the very last busy themselves with plans for the future, new methods of treatment, different climates, business schemes, and the confident expectation of recovery.

Physical Signs in Advanced Pulmonary Tuberculosis.—*Inspection.*—The thorax undergoes deformities corresponding to the progressive diminution in the volume of the lungs. It tends to assume permanently the expiratory form.

Palpation.—Diminished expansion at the apex may be determined in the following manner: The examiner stands behind the patient, who is seated, and gently grasps the shoulders with his hands, the tips of the fingers being in the infraclavicular spaces, the thumbs resting upon the upper part of the scapulæ. The patient then slowly draws a deep breath; to study the expansion at the bases the hands grasp the two sides of the chest in a similar manner in the lower axillary regions. Lagging and limitation of the excursus are signs of great significance. The vocal fremitus is increased over areas of consolidation; over vomicæ it may be increased or diminished. It is usually enfeebled over thickened pleuræ.

Percussion.—In doubtful cases percussion should be performed during quiet breathing and upon full-held inspiration, as minor differences in the sounds upon the two sides then become more marked. Slight relative dulness may be recognized by the elevation of pitch which accompanies it. Light percussion above, beneath, and over the clavicle should be practised. The supraspinous fossæ and the points corresponding to the apices of the lower lobes are important regions. Dulness over consolidation and tympany over vomicæ is the rule; but scattered small tuberculous foci with intervening air-containing vesicular structure, especially when there is collateral emphysema, yields resonance with a tympanitic quality, and a cavity filled with fluid will often yield a dull or, upon very nice percussion, a flat percussion sign. Over large cavities, situated near the periphery of the lung, the cracked-pot sound may be obtained.

Auscultation.—In the incipient stage the vesicular murmur is usually enfeebled, sometimes scarcely audible. Rough breathing is an early sign. Cog-wheel inspiration is often present, but it occurs in other conditions. With advancing lesions vesiculobronchial, bronchovesicular, and bronchial respiration succeed each other, to be finally replaced, as softening occurs and vomicæ form, by cavernous or amphoric breathing. Râles of all kinds, from the crepitus of the beginning lesion to the gurgling of cavities, attend the process. Râles are due largely to the accompanying bronchitis, and vary in kind and number according to the character of the secretion and the activity of the process in different parts of the lung. Puerile respiration may be heard over the adjacent unaffected lobes or over the opposite lung. The vocal resonance is increased and bronchophony and pectoriloquy may be elicited over areas of dense consolidation and cavities. Whispering pectoriloquy is an important sign of large superficial cavities. Pleural friction sounds are an important early sign and occur from time to time during the progress of the case. At first usually near the apex, they occur over advancing lesions in all points of the chest. Pleural friction at the left anterior border of the lung, and especially over the lingula, frequently has the cardiac rhythm—pleuropericardial friction. The signs of cavities are very variable. Situated superficially, a cavity may cause a distinct shallow depression in one or two intercostal spaces. A rapidly formed cavity, or several small cavities without much surrounding condensation or pleural thickening, may yield a full, clear resonance in which the only modification is a slightly tympanitic quality—vesiculotympanitic resonance. Tympanitic and amphoric resonance are usual. The pitch may be modified by opening and closing the mouth—Wintrich's sign—or upon change of posture—Gerhardt's sign. The cracked-pot sound can be brought out only over fairly large cavities with thin walls and superficially situated, and disappears for a time after it has once or twice been heard. Cavernous respiration is usually soft and low pitched. It may, however, be tubular or amphoric. Gurgling râles are common over large cavities and sometimes have a ringing or metallic quality, especially during coughing. Increased vocal resonance and whispering pectoriloquy are valuable signs. Over large cavities in the upper lobes the heart sounds and sometimes a transmitted systolic murmur are occasionally heard, and in rare cases sharp, splashing râles having the cardiac rhythm. Over a layer of dense, consolidated lung extending to a large bronchus there may be signs—circumscribed tympanitic percussion note, amphoric respiration, and coarse moist râles—which closely simulate those of a cavity. Light percussion, the pitch of the sound not being raised by opening the mouth or change of posture, and the absence of the cracked-pot sound may be of use in the recognition of the actual condition.

Diagnosis of Advanced Pulmonary Tuberculosis.—The DIRECT DIAGNOSIS rests upon the physical signs and the presence of tubercle bacilli in the sputa. The symptoms are variable and acquire diagnostic value only in proportion as they correspond with the signs of the lesions and their distribution and evolution.

The spirometer has deservedly fallen into disuse in the diagnosis of incipient tuberculosis of the lungs, and its use is attended with danger in the advanced cases. The expectations raised by the publications of Arloing and Courmont in regard to the use of tuberculous serum—agglutination—in the diagnosis of tuberculous disease have not been realized.

Prognosis in Chronic Ulcerative Phthisis.—When proper treatment is instituted in the stage of incipiency and rigorously carried out, the prospect of a cure is good. The frequency with which limited obsolescent, even healed tuberculous lesions are found post mortem in the lungs of individuals dead of other diseases or by accident, bears ample testimony to the intrinsic tendency to recovery. It has been said that more people recover from pulmonary tuberculosis without being aware that they have had it than die of it.

The prognosis in the advanced cases is unfavorable and in the far advanced cases hopeless. In individual cases, the early recognition of the disease, a good family history, limited local lesions, slight constitutional reaction, and the disposition and means to make a business of getting well are favorable conditions in the prognosis. The pleurogenous cases often run a slow and relatively favorable course. The cases characterized by recurrent blood spitting are unfavorable. Only in the worst cases is the course of the disease relentlessly progressive. As a rule there are periods varying from weeks to months in which the lesions remain quiescent, the symptoms subside, the general health improves, and there is a gain in weight. The duration varies from some months to several years, the average being about two and a half years. A remarkable decrease in the death-rate from consumption has taken place in recent years. This is to be ascribed to the discovery of the tubercle bacillus, the recognition of the fact that tuberculosis is an acquired rather than an inherited disease, and the dissemination among the people of the knowledge by which its spread can be restricted.

(c) FIBROID PHTHISIS.

This term is used to designate a very chronic tuberculous process in the lungs, with relatively slight ulceration and much development of fibrous tissue. It may begin as a tuberculous bronchopneumonia or follow an ordinary ulcerative phthisis. In a large proportion of the cases it begins as a chronic tuberculous pleurisy. The anatomical and clinical condition is practically that of pulmonary cirrhosis. One or both lungs may be affected; if both, one to a much greater extent than the other. There are vomicæ at the apex, surrounded by dense fibroid tissue, and bronchiectatic cavities elsewhere. The pleura is greatly thickened, and encapsulated cheesy masses, with patches of recent tubercle, and enlarged bronchial glands are present. Amyloid disease of the liver, spleen, and intestines develops in the advanced cases.

Symptoms.—Cough, often paroxysmal and more common in the morning, purulent expectoration, sometimes fetid, and dyspnœa upon exertion constitute the symptom-complex. Blood spitting occurs. The patients are thin, but frequently have fair health. Fever is not a common symptom.

Physical Signs.—The chest is flat, the shoulders lower, and the clavicles prominent. The vocal fremitus is diminished. Resonance is greatly impaired and has the tympanitic quality. At the apex cavernous, at the base bronchial, respirations are heard. Râles are not common, but coarse moist and gurgling râles may be present when fluid collects in the cavities or dilated bronchi. The superficial area of cardiac dulness is increased, the impulse may be seen and felt in two or more interspaces, and the heart is displaced toward the affected side.

Diagnosis.—DIRECT.—This rests upon the symptoms and physical signs as detailed above.

DIFFERENTIAL.—The distinction between tuberculous and non-tuberculous pulmonary cirrhosis cannot in all cases be made *intra vitam.* The presence of tubercle bacilli in the sputum is positive. When not found during life they are often present in the lesions after death. Atrophic emphysema presents some points of resemblance to fibroid phthisis, but differs from it in being a symmetrical affection and occurring only in aged persons.

Prognosis.—The outlook is favorable as to life, unfavorable as to recovery. The disease is chronic, lasting from ten to twenty or twenty-five years. The patient is usually able to attend to his affairs. There are cases characterized by recurrent hemorrhages, and death sometimes occurs from hæmoptysis.

Turban has suggested the following scheme for uniform records for comparative statistics in tuberculosis of the lungs:

1. Stadium = extent and severity of disease in the lung.	I. Disease of slight severity, affecting at most one lobe or two half lobes. II. Disease of slight severity, more extensive than I, but affecting at most two lobes; or severe, and affecting at most one lobe. III. All cases of greater extent and severity than II.
2. Disease quiescent or progressing.	
3. Length of time since onset.	To date from first occurrence of symptoms, such as persistent cough, hæmoptysis, or pleurisy.
4. General condition	Satisfactory. Unsatisfactory.
5. Digestion	Normal. Abnormal.
6. Pulse-frequency.........	To be taken in morning during repose.
7. Temperature	Daily maxima over 101.3°. Daily maxima between 99.7° and 101.3°. Temperature normal with two-hourly rectal temperature (mouth temperature 0.4°–0.5° lower).
8. Tubercle bacilli and mixed infection.	Tubercle bacilli present. Tubercle bacilli absent. Mixed infection.
9. Tubercular complications.	Name of affected organ.
10. Other complications	Name of disease: serious complications such as heart disease, nephritis, or diabetes are to be noted.
11. Capacity for work	Full, undiminished. Slightly reduced. Much reduced or lost.
12. Result of treatment	Improved. Not improved. Died.

Nos. 1–10 are filled up on admission or commencement of treatment.
Nos. 11 and 12 on discharge.

XXXIII. SYPHILIS.

Definition.—A chronic specific infection, propagated by inoculation and characterized by, (a) a peculiar initial lesion—the chancre; (b) constitutional symptoms with mucous and cutaneous lesions and enlargement of the superficial lymph-nodes; and (c) the development of granulomatous lesions in the various tissues of the body. These effects of the infection are consecutive, and constitute (a) the primary stage, (b) the secondary stage, and (c) the tertiary stage. Syphilis is frequently transmitted from the parent to the child—hereditary syphilis.

Etiology.—PREDISPOSING INFLUENCES.—Syphilis is a venereal disease and is usually acquired by illicit sexual intercourse. When acquired by an innocent person in the marital relation or by accidental means, it is described as *syphilis insontium.* Individual susceptibility is universal and affects all periods of life. Accidental infection is common among medical men. The fingers are usually the site of the primary lesion. Chancres upon the lip or tongue may result from the conveyance of the virus by kissing, the use of drinking utensils, the pipe, and other indirect methods. The infection is active in the oral and pharyngeal lesions—mucous patches. Unnatural vices are responsible for a certain proportion of the primary sores about the mouth. A nurse suckling a syphilitic infant may be inoculated upon the nipple and is also liable to accidental inoculation upon the lip, finger, or elsewhere. Local epidemics of syphilis among infants from arm-to-arm vaccination have been reported. Such accidents are no longer possible. Syphilis has been transmitted in tattooing. Hereditary transmission may take place from the father, the mother presenting no evidence of infection—*sperm inheritance, paternal heredity.* In rare instances a child begotten by a father in the active—secondary—stage has shown no evidence of syphilis. In equally rare cases the child of a father who, after thorough treatment, has shown no signs of the disease has developed congenital syphilis. There are usually unknown factors in problems of th's nature. After vigorous systematic treatment, and the lapse of three years after the entire disappearance of symptoms, a man may be allowed to marry and is not likely either to infect his wife or to beget infected children.

Transmission from the mother is called germ inheritance—*maternal heredity.* A woman suffering from syphilis in the active stage is liable, when conception occurs, to bear a syphilitic child. As a rule both parents are syphilitic, the one having infected the other. A very remarkable fact is set forth in Colles's law, which, briefly stated, is this: A child that is affected with hereditary syphilis, its mother showing no signs of the disease, will not infect the mother. Such a child will infect its nurse or others, but the mother appears to have acquired an immunity without manifesting any of the usual phenomena of the disease. In the case of the mother becoming infected after conception, the child may show the signs of congenital syphilis or, less frequently, it may escape. A parent or parents in the stage of tertiary syphilis may have non-syphilitic children.

EXCITING CAUSE.—Many organisms have been described in the course of the last twenty-five years. Recent observations have established the fact that the spirochæta described by Schaudinn in 1905, and named by him *Spirochæta pallida*, is the cause of the disease. This organism is very delicate, closely coiled, having pointed ends, and motile. A larger spiral organism found in association with it upon the surface of syphilitic sores, and also upon the ulcerated surfaces of non-syphilitic lesions, and in smegma from healthy men and women, he named Spirochæta refringens. S. pallida—*Treponema pallidum*—has been found with great frequency in syphilitic lesions at various stages of the disease, as well as in the organs of congenital syphilis and the placenta, and in greater numbers in the active lesions. It has been found in the syphilitic lesions of inoculated monkeys. It has been grown in culture by Noguchi.

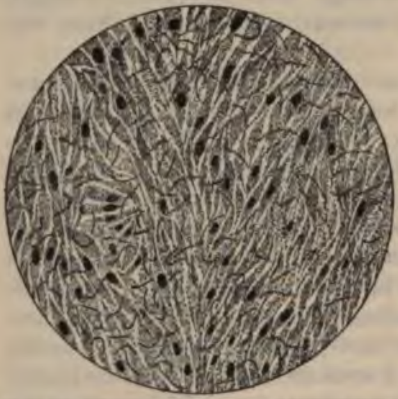


FIG. 281.—Treponema pallidum in a chancre.

(a) ACQUIRED SYPHILIS.

The Primary Stage.—A period of incubation varying from two to four weeks, exceptionally longer, elapses between the inoculation and the appearance of the initial sore. This lesion consists of a small red papule, which gradually enlarges and breaks down in the centre, forming a circumscribed, superficial ulcer, with a peculiar hard, gristly, or cartilage-like movable base, which still further increases in size and is known as the indurated or hard chancre. This initial lesion may remain small and readily elude observation when just within the urethra or in the female genitalia. In the other localities it usually appears as a conspicuous and characteristic sore. In the course of a week or two the associated lymph-nodes undergo a painless and indolent enlargement.

The Secondary Stage.—The earliest indications of constitutional infection are usually manifest within a period varying from six to twelve weeks. They consist of the following phenomena: *Fever*, usually so mild as to attract little attention, 101° F. (38.5° C.), sometimes marked, less frequently severe. In type it may be subcontinuous, remittent, or intermittent; in duration indefinite, sometimes only subsiding upon the vigorous use of antisyphilitic treatment. It may not appear until late in the course of the disease. The recognition of syphilitic fever is of great diagnostic importance. It may simulate malaria or the symptomatic fever of advanced pulmonary tuberculosis, or hectic fever due to other causes.

Anæmia.—The erythrocytes often fall rapidly to 3,000,000 and occasionally lower. There is pallor with a sallow or muddy tinge of the skin. The *superficial lymph-nodes*, especially the suboccipital and epitrochlear glands, become enlarged and tender. Lassitude, headache, rhachialgia, and the vague pains of a general infection are common. The designation syphilitic cachexia has been applied to cases in which this symptom-group is pronounced.

Cutaneous Lesions—Syphilodermata.—The earliest eruption is usually macular or roseolar. The individual spots are irregularly oval, of large size and often run together. They are symmetrically distributed upon the trunk and anterior surfaces of the arms and thighs. In color they are reddish-brown, often so faint as to be scarcely observed, sometimes vivid or coppery. This exanthem usually fades in the course of some weeks, but sometimes recurs at subsequent periods in the course of the disease. Later a papular eruption may appear upon the face and trunk, not unlike acne. This syphilide occasionally appears upon the forehead just below the edge of the hair—*corona veneris*. Frequently associated with it is a pustular eruption, suggestive of the variolous rashes. This combination of papules and pustules appearing upon the head and trunk, especially when syphilitic fever is present, may give rise to an erroneous diagnosis of smallpox. Still later squamous rashes appear, much like psoriasis but less scaly, coppery in color, and often confined to the palms and soles. Papulosquamous lesions are by no means rare. These eruptions frequently appear in the above order, but sometimes in a different succession, and two or more are often present at the same time. Symmetry and polymorphism are characteristic of the syphilodermata in the secondary stage. *Flat Condylomata.*—About the vulva and anus, upon the perineum, at the corners of the mouth, occasionally at the umbilicus, and in the folds of the armpits and groins, and elsewhere where the skin is constantly moist or there are opposing cutaneous folds, there sometimes appear, but not in all cases, flat warty growths, slightly elevated, with distinct borders and a moist, grayish surface. The secretion causing these lesions is inoculable and they are in the highest degree characteristic of syphilis. *Alopecia Syphilitica.*—Not rarely the hair, and often the eyebrows and lashes, fall out during the secondary stage. The loss of hair may be in patches, like those of alopecia areata, or there may be a general thinning. *Onychia Syphilitica.*—The nails are, in some cases, affected by a syphilitic inflammation involving the matrix, and are lost or become deformed.

Lesions of the Mucous Membranes.—The oral mucosa is chiefly affected. About the time of the appearance of the rash the throat and mouth become sore. There is a general erythematous angina, more intense than elsewhere, upon the tonsils and pharynx, where are frequently visible small, superficial ulcers with well-defined, scalloped borders and grayish-white surfaces—*mucous patches*. These patches are also common on the tongue, the lips, and the buccal mucosa. They are characteristic of syphilis and the secretion from their surface is highly inoculable. Whitish patches upon the tongue—*leucomata*—sometimes occur, especially in smokers. Papillary hypertrophy of the mucosa about the vulva or at the verge of the anus may give rise to warty excrescences of considerable size—*condylomata*.

Other lesions of diagnostic importance are iritis, which is often encountered early in the secondary stage and tends to recur; much less frequently choroiditis and retinitis, and deafness from otitis media or labyrinthine disease. Abortion and miscarriage are common and repeated interrupted pregnancies are very suggestive. Periostitis is a border-line lesion marking the late secondary or early tertiary stage. It especially involves the tibiæ, clavicles, cranial bones, and less frequently the sternum. It is usually circumscribed and often associated with nodes. Upon palpation the surface of the long bones is rough, and nodular bosses may be felt upon the bones of the skull. There is tenderness upon pressure, and pain, which is usually worse at night.

The Tertiary Stage.—There is no distinct time between the secondary and tertiary stages. Tertiary lesions are sometimes present shortly after infection; sometimes they make their appearance along with the phenomena which are characteristic of the secondary stage; more commonly they do not appear until a longer or shorter, often a remote, period after the lesions of that stage have subsided. The third stage of syphilis is characterized by certain lesions of the skin, the development of gummata, disease of the bones, and amyloid degenerations. *Cutaneous Lesions.*—Circumscribed nodular lesions are common. They appear in groups, which are irregular, asymmetrical, and characterized by the formation of deep, rounded ulcers which involve the deeper layers of the skin and tend to coalesce, healing at one point and spreading at another, and leaving deep scars as they heal. Rupia, a deep ulcerating tertiary lesion covered by stratified, oyster-shell-like crusts, is much less common than formerly.

Gummata.—These lesions are circumscribed and vary in size from minute bodies to tumors sometimes reaching five centimetres in diameter. They develop in the skin, subcutaneous tissue, mucous membranes, internal organs, muscles, and bones. In the bones they form dense, hard, hemispherical, subperiosteal masses called nodes. Gummata of the skin and subcutaneous tissue tend to break down and form deep ulcers, which heal slowly and leave deep, disfiguring scars. Under treatment they are frequently absorbed. Gummata of the mucous membranes are especially common in the mouth, nose, and pharynx. They involve underlying structures and often give rise to extensive and deep ulceration and necrosis of cartilage and bone. Perforation of the nasal septum, destruction of the nasal bones, perforation and more or less extensive destruction of the hard and soft palate, and adhesions of the uvula or soft palate to the pharyngeal wall are common effects. Ulceration and necrosis of the cartilages of the larynx also occur. Stricture of the rectum is one of the results of gummatous infiltration and ulceration. Syphilomata are common in the internal organs. They sometimes form agglomerations of large size. Their usual course is to undergo fibroid metamorphosis with puckering and deformity. Syphilitic nodes and periostitis have already been described. Further lesions are extensive and deep necrosis, which may become perforating, as in the bones of the cranium, the formation of exostoses which may cause serious and obscure pressure symptoms, as in the brain or spinal cord or the articulations. Syphilitic dactylitis, often followed by permanent deformity, is the manifestation of gummatous infiltration and periostitis of the bones of the fingers and toes. Much less common are gummata of the muscles and myositis syphilitica. Amyloid degeneration is common in syphilis even in the absence of chronic suppuration. It occurs especially in neglected cases of the acquired disease and is rare in the congenital form.

(b) HEREDITARY SYPHILIS.

The infant may show the characteristic symptoms at birth or may present the appearance of health. In the latter case the evidences of infection appear in the course of one or two months.

1. **Symptoms at Birth.**—The child is undersized and usually wasted and wrinkled. Bullæ may be present on the wrists and ankles and scaly patches upon the palmar and plantar surfaces. Mucous patches upon the nasal and oral mucous membrane, rhagades at the angles of the mouth and at the anus are characteristic. There is enlargement of the liver and spleen. Nodular thickening of the bones and, in some cases, separation of the epiphyses occur. Such children snuffle, are extremely feeble, are difficult to feed, and usually perish within a short time. Hemorrhage is occasionally encountered. It is more common at the navel but may be subcutaneous, or there may be bleeding from the mucous surfaces.

2. **Early Symptoms.** — When born without symptoms, syphilitic children are often plump and well nourished and remain so until some time between the third and eighth weeks. The earliest symptoms are usually those of a syphilitic endorhinitis, namely, impeded nasal respiration, difficulty in nursing, snuffling, and a mucopurulent, sometimes bloody discharge. In severe cases necrosis of the nasal bones may occur, followed by characteristic deformity of the face. Involvement of the Eustachian tubes and middle ear results in deafness. Such cases constitute one of the groups of deaf-mutes. Cutaneous lesions appear about the same time. They consist of a certain general muddy sallowness, in sharp contrast to the fresh rosy skin of a healthy infant, patchy erythema or eczema, or large, irregular coppery patches with well-defined borders. These eruptions are frequently first seen upon the buttocks, but may invade other regions. A papular syphilide is common. Rhagades upon the lips and especially at the corners of the mouth and at the anus are of diagnostic importance. Mucous patches develop and are highly contagious. The secretion from these lesions usually constitutes the means of infection in wet-nurses and others. Alopecia, onychia, dactylitis also frequently occur as the case goes on. A general adenopathy is not common, but the lymph-nodes in relation to local lesions of the skin often undergo an indolent enlargement. The spleen is enlarged; the liver less constantly and to a less extent. The large relative size of the liver in the new-born is to be kept in mind.

3. **Later Symptoms.** — Children suffering from congenital syphilis may regain the appearance of health under judicious management. Very frequently they remain undersized and badly nourished and look prematurely old. The facies and cranial development are very often characteristic. The skull is frequently asymmetrical, the forehead prominent, the bridge of the nose in some cases depressed, the lips pouting, with radiating linear scars, especially at the corners of the mouth. At the second dentition and at puberty the symptoms of hereditary syphilis frequently reappear.

Hutchinson Teeth—Notched Teeth.—The upper central incisors are peg-shaped, shorter, and narrower than normal, and especially narrower at the cutting edge than at the neck. The enamel is usually well formed, not pitted and thinned as after prolonged non-specific sickness in infancy, and there is at the cutting edge a single notch of varying depth in which the enamel is deficient and the dentin exposed.

Other symptoms are interstitial keratitis, iritis, deafness of labyrinthine origin, bone lesions, and, in particular, a gummatous periostitis which gradually causes marked thickening and deformity and which shows an especial tendency to affect the tibiæ. The nodes of late hereditary syphilis are usually symmetrical and are sometimes mistaken for rickets. They may first appear in adolescence. There may be enlargement of the spleen and visceral gummata.

The question of the transmission of syphilis to the third generation remains undecided.

(c) VISCERAL SYPHILIS.

1. **Syphilis of the Central Nervous System** (see Diseases of the Nervous System).

Para- or Metasyphilitic Diseases.—The term parasyphilitic was suggested by Fournier to designate a group of affections not directly due to syphilis, but much more common in those who have had that disease than in others. This category includes tabes, paresis, certain types of epilepsy, and forms of arteriosclerosis. They are not curable by mercury and the iodides.

2. **Syphilis of the Lungs.**—Pulmonary syphilis is a very rare condition. The following forms are described: (a) *White Pneumonia of the Fœtus.*—The process may involve extensive portions of a lobe or an entire lung. The affected tissue is heavy, airless, and of a grayish-white color. The alveoli are filled with desquamated epithelium and their walls are thickened and infiltrated. (b) *Gummata* irregularly scattered throughout the lung, especially in connection with the bronchi and more abundantly about the root than elsewhere. There is an associated bronchopneumonia. (c) *Fibrous interstitial pneumonia* beginning at the root of the lung and extending along the bronchi and vessels. This sclerotic process may begin in the pleura and involve the connective-tissue framework, especially in the interlobar tissue. It principally affects the portions of the lung adjacent to the root. It is encountered in individuals with a syphilitic history or in whom there are other forms of visceral syphilis, and is sometimes associated with gummata. As in other forms of pulmonary sclerosis, bronchiectasis is often present.

Symptoms. — The clinical manifestations are those of pulmonary tuberculosis or pulmonary sclerosis. In the former case the absence of tubercle bacilli upon repeated examination, and the absence of signs of destructive lesions, as elastic tissue, are suggestive; in the latter the signs of chronic interstitial pneumonia and of bronchiectasis are present. The acute syphilitic pneumonia and chronic syphilitic phthisis of French authors are not generally recognized as clinical entities.

3. **Syphilis of the Liver.**—The following forms are described: (a) *Diffuse Syphilitic Hepatitis.*—This is common in congenital syphilis. The organ is large and firm and shows the presence of minute and larger gummata and extensive connective-tissue hyperplasia. (b) *Gummata.*—In congenital syphilis gummata of various sizes may occur at any period. In the acquired disease they are usually among the later manifestations of the acute process. They are commonly multiple and may attain the size of an orange. They undergo fibroid changes with contraction and cause remarkable deformities of the organ; in rare cases softening takes place with the formation

of one or more fluctuating tumors. (c) *Syphilitic Perihepatitis.*—Glisson's capsule and the connective tissue along the portal canals are thickened. Great vein obstruction may occur when the connective-tissue proliferation extends along the large venous trunks. (d) *Amyloid Liver.*—This change is very common in syphilis. Gummata may be present or a consecutive diffuse hepatitis may occur.

Symptoms.—The clinical phenomena are by no means constant. Congenital syphilitic hepatitis can scarcely be diagnosticated with precision even when suspected. The organ is enlarged and firm. There may be jaundice. In the adult the symptom-complex of atrophic cirrhosis is frequently present. The symptoms are sallowness or slight jaundice, digestive disturbances, loss of weight, and ascites.

Irregularity in the outline of the liver dulness occurs in many of the cases. The evolution and involution of gummata cause progressive and retrogressive deformities of the liver which are of great importance in diagnosis. These syphilitic tumors are less dense in consistence than the surrounding tissue in hepatitic or amyloid disease, and can in some cases be differentiated from it upon palpation. In syphilitic perihepatitis an audible and palpable friction rub may sometimes be recognized; jaundice is present in one-third of the cases and may be intense. Pains in the hepatic region occur and the signs of ascites and of splenic enlargement are by no means rare. In amyloid liver the symptoms of amyloid disease in other organs are usually present. The liver is enlarged, smooth, and firm, Its outline may be irregular. There is commonly also enlargement of the spleen. Anæmia, polyuria with albumin and casts, and a tendency to dropsy are present.

4. **Syphilis of the Digestive Tract.**—The œsophagus and stomach are very rarely involved. Ulceration of the small intestine is likewise uncommon. The rectum is far more often affected. Rectal syphilis is more common in women. The lesions are due to gummata in the submucous tissue above the internal sphincter, which undergo ulcerative changes which become chronic and on healing cause stenosis. There may be tenesmus, discharge of bloody pus with the stools, and pain on defecation. Later the symptoms are those of stenosis.

5. **Syphilis of the Circulatory System.**—**The Heart.**—Valvular lesions are exceedingly infrequent. Both vegetations and gummata have, however, been observed. Mural lesions are common. They comprise gummata, fibroid induration, amyloid degeneration, and endarteritis obliterans. Changes in the blood-vessels of the heart occur both in the congenital and the acquired disease. Valvular lesions give rise to definite murmurs. Syphilis of the myocardium may be present without symptoms; those characteristic of myocarditis are usual. Sudden death may occur.

The Arteries.—There are two forms of syphilitic arteritis, an obliterating endarteritis which is not distinctive, and a gummatous periarteritis which involves especially the smaller arteries of the brain and the branches of the coronary arteries, and is specific. Syphilitic changes in the arteries are etiologically related to arteriosclerosis and aneurism.

6. **Syphilis of the Kidneys.**—Gummata and amyloid degeneration constitute the common changes. The former cannot be recognized *intra*

vitam; the latter presents the usual symptoms. An acute syphilitic nephritis, without specific characters, has been described.

7. **Syphilis of the Testicles.**—Gummata in the substance of the testis is not uncommon. It may be mistaken for tuberculosis. It is usually painless and does not tend to invade the skin or to undergo softening or suppuration. An interstitial orchitis may develop as a slowly progressive affection unattended with pain and resulting in induration and atrophy. One testis is usually affected to a greater extent than its fellow.

Diagnosis.—1. **General Diagnosis of Syphilis.**—(a) THE PRIMARY LESION.—The surgeon is more frequently consulted than the medical man. In a suspicious sore the following points are of importance: a history of exposure within a month or six weeks; induration; movability; sluggish ulceration; scanty secretion; slight painless enlargement of the inguinal glands. These traits belong equally to the chancre upon the genitalia and elsewhere. The history of exposure may, however, be in default. The patient may prefer to conceal the actual fact at the risk of his future health as well as his character for veracity; or the inoculation may have occurred in marital intercourse or otherwise by non-genital infection. The initial lesion may not have attracted the patient's attention. This is especially liable to occur in women. In man the lesion is sometimes inconspicuous and may be mistaken for preputial herpes or an abrasion; or it may be masked by coincident chancroids, or, when at the meatus or in the urethra, by a gonorrhœa, or finally an extragenital chancre, even when well characterized, may fail to arouse the suspicion of the practitioner as to its true nature. There is only one diagnostic rule, namely, to preserve a guarded and discreet openness of mind in all doubtful cases and carefully watch for subsequent developments.

(b) THE SECONDARY STAGE.—Consecutive events are important. The history of exposure, especially when doubtful, and the history of a subsequent sore, however doubtful, are of great diagnostic value. Sore throat and roseola are usually the first symptoms which attract the patient's attention. A painful erythematous angina, with tonsillar ulceration and mucous patches, with a symmetrical, faint, brownish-red macular rash upon the trunk, and painless enlargement of the inguinal, suboccipital, and epitrochleal lymph-nodes, especially when associated with fever, constitutes a symptom-complex upon which a direct diagnosis may be made. Later polymorphous rashes, corona veneris, alopecia, irregular fever, and anæmia are confirmatory.

The Wassermann Test.—This serum test requires a very careful technic and is exposed to many sources of error. It therefore should only be undertaken by trained laboratory workers. The reliability of this diagnostic method has been fully established. Wassermann has given the results of 3000 tests, of which 1010 were upon cases surely non-syphilitic and used as controls. In 1982 syphilitic cases about 90 per cent. gave positive results. In cases without manifest symptoms at the time, "latent syphilitis," about 50 per cent. gave positive reactions. It is, however, not only a means of diagnosis but it is also a therapeutic guide. In the course of treatment a positive reaction may be absent with entire latency as to symptoms, only to reappear again, either with or without symptoms. The intensity corresponds to some extent to the activity of the process. A positive result is common in cases of tabes and paresis. Cholesterinized antigen appears to be preferable to the alcoholic extract of syphilitic liver.

Provocative Test.—When the Wassermann reaction is negative in a suspected case a small dose of salvarsan is given and in a short time the Wassermann is repeated. The theory is that the salvarsan acting upon the lesions causes the spirochætes to enter the circulation.

Luetin Test (Noguchi).—This procedure consists in the superficial injection into the skin of one arm of a suspension of *Spirochæta pallida* in pure culture and devitalized by heat, the other arm being similarly treated by an uninoculated control medium. Separate syringes are used for the luetin and the control. The amount of luetin employed for a single test is 0.07 c.c. A positive reaction consists in the appearance, some hours later or upon the following day, of an inflammatory nodule surrounded by a bright red areola which is fully developed in 48 to 72 hours. Mild constitutional symptoms may occur. Pustulation sometimes appears. This test is of use in tertiary cases, but rarely yields a positive reaction in primary or untreated secondary syphilis.

(c) THE TERTIARY STAGE.—The anamnesis is here also of great importance. It is often defective. Sometimes discretion suggests a very guarded investigation of the past history of the patient. Inquiry should be made concerning persistent rashes and falling of the hair. Careful inspection of the throat and skin should be made for the signs of past lesions. Scars in the groins are insufficient evidence. Suppurating buboes are usually due to chancroids, not syphilis. Slowly progressive ulcerating lesions of the skin, advancing in one direction and healing in another,—serpiginous,—gumma and gummatous ulceration, perforation of the nasal septum, of the hard or soft palate, necrosis of the nasal or cranial bones, the signs of iritis, the presence of nodes, irregular periosteal thickening or exostosis, especially upon the clavicles, tibiæ, or bones of the skull, particularly when two or more of them are associated, constitute diagnostic data of final importance in the direct diagnosis. But these lesions are often wholly absent and the sufferer from tertiary syphilis, and particularly the sufferer from nervous syphilis, may be entirely free from the gross or visible external manifestations of the disease. In many cases of nervous syphilis it is impossible to elicit a history of marked secondary signs.

2. **Diagnosis of Hereditary Syphilis.**—Repeated miscarriages are, in connection with any of these phenomena, of diagnostic value. The efflorescence of the characteristic rash associated with snuffles, mucous patches, and rhagades within the first three months justifies the direct diagnosis. At subsequent periods of life the characteristic facies, infantile development, symmetrical nodes, notched teeth, and interstitial keratitis tell their own story and may solve the problem of diagnosis in obscure nervous or visceral disease.

3. **Diagnosis of Visceral Syphilis.**—The anamnesis and the presence of the signs of former lesions are of primary importance. In the male careful search should always be made for vestiges of the primary sore. It is to be remembered that the manifestations of visceral syphilis are usually not in themselves different from those of lesions due to other pathological processes, and that their true nature can be recognized only

by the history of infection, the presence and association of characteristic external phenomena, or the therapeutic test.

1. *Syphilis of the Brain and Cord* (see Diseases of the Nervous System).

2. *Syphilis of the Lung.*—The clinical diagnosis of this rare condition in any of its forms can seldom be made with precision. Chronic interstitial pneumonia with the signs of bronchiectasis, or chronic bronchopneumonia in a person with a history of syphilis or presenting well-characterized lesions in other parts of his body, may be of syphilitic origin. Tuberculosis of the lungs and gummata may coexist.

3. *Syphilis of the Liver.*—The diagnosis is most important, as it is essential to the choice of treatment. The irregularly enlarged liver, with soft circumscribed gummata, may suggest cyst, abscess, or malignant tumor. Under such circumstances an unnecessary surgical operation might be performed. A history of infection, collateral lesions, and fair general health suggests syphilis. The diagnosis in gummata forming large conglomerate tumor masses in the right or left lobe in absence of collateral evidence must remain obscure. In cirrhosis and perihepatitis recovery under specific treatment is often the only sign. Irregularity of outline, which, when there is ascites, can only be determined after paracentesis, is very suggestive. Amyloid disease of the liver is commonly associated with similar visceral changes elsewhere.

4. *Syphilis of the Digestive Organs.*—The history affords presumptive evidence in disease of the œsophagus. Syphilis of the stomach cannot be positively diagnosticated. The chronic course of syphilis of the rectum, the symptoms of gradual stenosis of the gut, and the results of digital examination by which a firm fibrous annular contraction is usually felt, quite unlike the irregular, ragged surface of ulcerating cancer, are essential diagnostic criteria.

5. The clinical diagnosis of *syphilitic disease of the heart and arteries* must be a provisional one. Cardiovascular changes, in no respect differing in symptomatology from those occurring in syphilitic subjects, are often due to other causes.

6. The diagnosis of *renal syphilis* cannot be made during life.

7. *Syphilis of the Testes.*—The recognition of syphilis in these organs may be of great importance in obscure visceral disease. Syphilis, tuberculosis, and cancer are to be differentiated. Gummata involve the body of the testicle and give rise to irregular conglomerated masses, unattended with pain and showing no tendency to invade the skin or undergo softening. Tubercle more commonly affects the epididymis and is often associated with the signs of tuberculous disease elsewhere. Malignant disease runs a more rapid course, attains a larger size, is attended with pain, and tends to involve the skin and undergo ulceration.

Therapeutic Diagnosis.—Treatment by salvarsan reinforced by mercury and the iodides under suitable conditions will frequently cause the disappearance of symptoms. Intermittent courses of treatment, repeated during long periods of time, usually prevent the recurrence of symptoms, arrest the tendency to abortion, and may be followed by the birth of healthy children in whom neither the early nor the late manifestations of syphilis occur. Obscure skin eruptions fade if of syphilitic origin.

With reference to visceral syphilis the following facts are important: The symptoms of nervous syphilis may, in early cases, disappear after the use of antisyphilitic remedies; in pulmonary syphilis the results are inconclusive; syphilis of the liver in certain of its forms is amenable to treatment and in some cases the improvement is rapid and permanent; syphilis of the heart and arteries shows retardation rather than cure and here the therapeutic test is useless. The symptoms of the secondary stage yield promptly, while in the tertiary stage the gummatous lesions yield more or less gradually and the sclerotic lesions are but slightly if at all influenced. The so-called parasyphilitic diseases are not cured by antisyphilitic treatment. Finally there are rare cases of acute malignant syphilis which run a rapidly fatal course wholly uninfluenced by treatment.

Prognosis.—Under early, systematic treatment, repeated from time to time in courses of proper duration for a period of three or four years, an apparent cure is, in the majority of cases, established and maintained.

XXXIV. GONORRHŒA.

Definition. — A contagious catarrhal inflammation of the genital mucous membrane, chiefly propagated by impure sexual intercourse, and due to the gonococcus of Neisser.

This wide-spread venereal infection is scarcely inferior in importance to syphilis. In truth, when we take into consideration the facts that syphilis is much less virulent than formerly in its early and late constitutional effects, and that it gives rise to symptoms which compel the most ignorant and inexperienced to seek professional advice at a period when it is still amenable to treatment, and that gonorrhœa, while retaining all its capacity for immediate and late harmfulness, is too often regarded as a trifling local disorder, the very existence of which may be unsuspected by the female patient, we may even question whether gonorrhœa is not the more serious disease of the two.

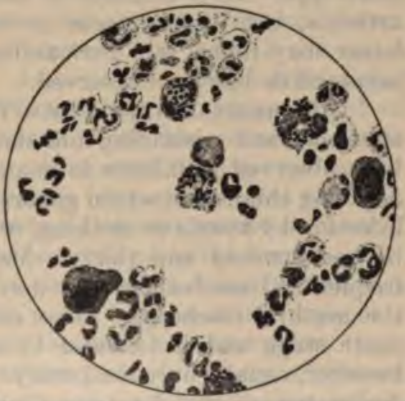


FIG. 282.—Spread of pus containing gonococci.

The gonorrhœal infection may limit itself to the mucous membrane of the genitalia—(1) the primary local infection; it may invade the genito-urinary organs by direct continuity of structure—(2) secondary local infection; or finally, it may be swept into the blood stream and give rise to (3) constitutional infection.

With reference to the spread of the infection in the genito-urinary tract, it is a question of extent. Every case in the male is at first an infection of the anterior urethra. With reference to systemic effects, it is a question of degree; any case is liable to systemic disturbance, and malaise, feverishness, head and back pains, and other symptoms of constitutional infection are often present at the onset or later in the course of the attack. The fever associated with the initial symptoms is due to the absorption of toxins.

The Primary Local Infection.—The gonococcus may persist in a slight urethral discharge capable of giving rise to the disease for long periods after the patient has thought himself cured; that innocent women frequently become infected upon marriage. It is also to be remembered that the existence of a urethral stricture bears a causal relation to cystitis, pyelitis, persistent rhachialgia, lassitude, general disability, and neurasthenia, and in acute disease it may be the cause of urinary retention. The ocular infections—*ophthalmia neonatorum* and *gonorrhœal conjunctivitis*—are extremely dangerous and if neglected frequently lead to loss of sight.

Secondary Local Infection.—The gradual extension of the specific inflammation from the mucous membrane primarily involved gives rise, in the male, to posterior urethritis, epididymitis, prostatitis, periurethral abscess; in the female to abscess of the vulvovaginal glands, metritis, salpingitis, inflammation of the ovary, and, in rare cases, to acute peritonitis; in both sexes to cystitis and pyelitis, usually mixed infections.

Constitutional Gonorrhœal Infection.—GONORRHŒAL SEPSIS—SEPTICOPYÆMIA.—The presence of the gonococcus in the blood has been demonstrated. There are cases of rapidly fatal general infection, usually associated with suppurative lesions in the urinary tract. The symptoms are chill, high temperature of irregular range, profuse sweats, muttering delirium, and stupor deepening to coma.

CARDIAC AND ARTICULAR LOCALIZATIONS—GONORRHŒAL ENDOCARDITIS.—This localization is of frequent occurrence in gonococcus septicæmia. Gonococci have been isolated from the blood during life, and the vegetations upon the valves after death. The endocarditis is often of the malignant type. In a majority of the cases the endocarditis has followed an arthritis, but it may occur in the absence of the joint affection or the latter may follow it. Pericarditis may occur, and an acute gonorrhœal myocarditis has been observed.

GONORRHŒAL ARTHRITIS.—The designation gonorrhœal rheumatism is erroneous and misleading and should be abandoned. This localization has been observed in infants in connection with ophthalmia neonatorum and in young children in whom gonorrhœa is common as the result of accidental infection by towels or clothing, or of vicious practices. It is most common between twenty and thirty. Males suffer from the joint affection more frequently than females. It may occur at any time during the course of the urethral discharge. Most commonly the arthritis begins during the acute stage and is followed by a subsidence of the discharge, which is, however, usually only temporary. It may occur after discharge has greatly diminished or not for several weeks after the beginning of the attack. A single joint may be affected; more commonly two or even three are involved. Polyarthritis is rare and the migratory form characteristic of acute rheumatism does not occur. The joints remain inflamed and only slowly get well. The ankles, knees, and wrists are especially liable to gonorrhœal inflammation and the temporomaxillary, sternoclavicular, vertebral, and sacro-iliac articulations are frequently attacked. The inflammation is endo- and periarticular. In the latter case the exudate sometimes extends along the sheaths of the tendons. The effusion into the joints is that of a serous synovitis. It may be seropurulent. The gonococcus may be isolated from the endo- and the periarticular exudates. In some cases the results are negative. Mixed infections—staphylococci, streptococci—occur.

Gonorrhœal arthritis tends to become chronic. Relapses are frequent; disorganization or disabling ankylosis may result. Among the complications are iritis, endocarditis, pericarditis, and pleurisy.

Diagnosis.—DIRECT.—The anamnesis is most important. The existence of a mucopurulent urethral discharge is not positively conclusive. A young person suffering from gonorrhœa may also contract rheumatic fever. In doubtful or obscure cases the complement-fixation test must be employed. In the female a vaginal discharge should be examined for gonococci. Clinically, a limited number of joints involved, the persistence of the inflammation without migration, the association of endocarditis of the malignant form, and implication of the temporomaxillary, sternoclavicular, or vertebral articulations are of diagnostic value.

DIFFERENTIAL.—*Rheumatic Fever.*—The evanescent—*migratory*—character of the arthritis, the number of joints involved,—*polyarthritis,*—the frequent rapid and complete resolution of the inflammation, a history of exposure to cold or wet, of previous attacks, of heredity, and the commonly lower intensity of an endocarditis, when present, are in favor of rheumatic fever. *Pyæmic or Septic Arthritis.*—The presence of a suppurative focus, caries, osteomyelitis with toxæmic phenomena, as irregular chills, high fever of irregular remittent or intermittent type, profuse sweating, rapidly developing anæmia, the implication of a single joint or, at most, two or three, and the evidences of large intra-articular effusion, are in favor of septic arthritis. *Gout.*—If the patient be a middle-aged male, energetic and self-indulgent, the attack be sudden, the great toe, ankle, knee, or wrist involved, the swelling tense, the surface livid and glossy, and tophi be present around the small joints or in the helix of the ear, a diagnosis of gout may safely be made. I have seen a calculus impacted in the urethra cause a free purulent discharge, followed by an attack of gout. *Arthritis Deformans.*—There are cases in which, for a time, the joint affection, whether arthralgic, polyarthritic, or monarthritic, cannot be distinguished at the time of acute exacerbations from gonorrhœal arthritis. No question arises in regard to the differential diagnosis except in cases of recent or chronic urethritis in the male or leucorrhœa in the female.

Prognosis.—Gonorrhœal infection in its systemic form is always a serious matter. The virulent septic cases prove fatal in a few days or a week or two; the endocarditis frequently assumes the malignant type; the arthritis is commonly rebellious to treatment and not rarely is followed by lasting, even permanent disability. Salicin and the salicylates are useless.

XXXV. EPHEMERAL FEVER—FEBRICULA—SIMPLE CONTINUED FEVER.

Definition.—Fever of short duration, occurring in the absence of definite lesions or known specific cause, and characterized by elevation of temperature and the derangements of function which commonly attend it. Fever lasting twenty-four or forty-eight hours and ceasing completely is designated *ephemeral fever;* an attack of three or four days' duration, *febricula;* and one lasting a week or more, *simple continued fever.*

Etiology.—Children and neurotic individuals are more liable to transient febrile attacks than others—a fact due to the instability of the heat-regulating mechanism. Several groups of cases are described: (a) symptomatic, (b) toxic, and (c) infectious.

(a) In many cases the fever is doubtless due to unrecognized local lesions, as in angina tonsillaris or catarrhal bronchitis of the larger tubes, with little or no cough and no râles; or to slight injury such as results from a fall upon the head.

(b) Among the toxic cases are those which arise from indigestion or gastro-intestinal catarrh with the absorption of fever-producing substances. The fever sometimes follows prolonged mental or physical effort, exposure to damp and cold without definite lesions, exposure to the sun but not to a degree sufficient to cause thermic fever, and the inhalation of the concentrated emanations from decomposing organic matter. There are instances in which a number of persons have been at the same time taken ill, with nausea, vomiting, fever, and, in some instances, collapse symptoms after being present at a very offensive post-mortem examination or the opening of an obstructed sewer or of a mortuary vault.

(c) Mild or abortive cases of the infectious diseases have been regarded as febricula. Stillé called attention to the occurrence of cases of cerebrospinal fever so mild that they can "only be recognized by the lurid light of the epidemic." The true nature of the mildest form of enteric fever—typhus levissimus—may be readily overlooked, and there are cases of scarlet fever, measles, and rheumatic fever without distinctive symptoms beyond a transient fever for two or three days—larval cases of the infectious diseases. In view of these facts, it is evident that the more closely the cases of transient fever without obvious symptoms are studied, the fewer will be encountered that are really neither symptomatic nor specific, and there is a tendency to do away with this group of fevers altogether as a nosological entity. On the other hand, every practitioner occasionally encounters cases for which no other place in the classification of diseases can be found.

Symptoms.—The onset is usually abrupt; exceptionally gradual with lassitude and languor. The usual symptoms of febrile infection are present. In rare instances, especially in children, there may be chilliness or a convulsion. Defervescence takes place by crisis between the second and the fourth days; if later, usually by rapid lysis.

A direct **diagnosis** may be made from the abrupt onset of the fever, its short course and the critical termination in the absence of local lesions and cutaneous rashes. The **prognosis** is invariably and essentially favorable.

XXXVI. ROCKY MOUNTAIN SPOTTED FEVER.

Tick Fever.

Definition.—An acute infectious endemic disease, prevalent in the northwestern mountainous regions of the United States, and due to the bite of a tick. It is characterized by the gradual onset of symptoms common in the acute infections, a papular or petechial eruption, and an irregular fever of variable duration terminating by lysis.

This disease, long known under designations such as "mountain fever," "black fever," and "spotted fever," has recently been made the subject of scientific investigation under the auspices of the Montana State Board of Health (1902), the Public Health and Marine Hospital Service (1903), and the American Medical Association (1906–1907), and is now recognized as a nosological entity.

Etiology.—PREDISPOSING INFLUENCES.—Certain districts in the Rocky Mountains are the exclusive regions in which the disease occurs. The great majority of the reported cases have been observed in April, May, and June. A few have occurred in March and July. No period of life affords exemption. The disease has been noted as early as the second and as late as the seventy-fourth year. Ranchmen, lumbermen, engineers, and prospectors have supplied the greater number of cases.

EXCITING CAUSE.—Despite various hypotheses, the infecting organism has not yet been demonstrated. It is inoculated by the bite of a tick—*Dermacentor andersoni.* Tick fever may be caused in monkeys and guinea-pigs by the inoculation of the defibrinated blood of human beings suffering from the disease. Some observers have attributed the disease to the inoculation of the infecting principle by mosquitoes.

Symptoms.—The average duration of the period of incubation appears to be about seven days. The onset is attended by nausea and vomiting, muscular pains, malaise, headache, and epistaxis, chilliness or a distinct chill. The temperature rises abruptly to 103°–104° F. (39.5°–40° C.) and its range may be subcontinuous or intermittent. At the expiration of from sixteen to twenty days defervescence takes place, followed by subnormal temperatures for a few days. In fatal cases the temperature usually remains high, but a preagonistic fall sometimes occurs. The eruption shows itself about the third or fifth day, first appearing upon the ankles, wrists, and forehead, and gradually spreading over the whole body. It remains, however, more abundant upon the extremities than upon the trunk. It is at first maculopapular and disappears upon pressure. In the course of a few days it becomes more distinctly papular and does not disappear upon pressure or when the skin is made tense. Many of the spots become petechial and in the severer cases more or less extensive ecchymosis occurs. The intervening skin is congested, slightly cyanotic, and jaundiced. In favorable cases the eruption fades with the defervescence and its disappearance is followed by desquamation. Gangrene of the penis or scrotum or of the toes has been reported. In mild cases the rash may not appear. Gastro-intestinal symptoms are prominent. The liver and spleen are enlarged. The urine is diminished in volume, of high color and specific gravity, and contains albumin and casts of all kinds. The blood shows a moderate secondary anæmia and in some cases a moderate leucocytosis—13,000.

Diagnosis.—The direct diagnosis rests upon the endemic occurrence of a disease having the above symptom-complex in particular localities and at certain seasons of the year, a history of tick or mosquito bites, sudden onset, chilliness or high continued or intermittent fever, the appearance of the maculopapular eruption upon the third, fourth, or fifth day, petechiæ, ecchymosis, and defervescence followed by desquamation.

Prognosis.—Rocky Mountain spotted fever is a very fatal disease. According to several observers the mortality varies from 70 to 90 per cent. In some seasons the cases are mild and most of them have terminated in recovery. Among the phenomena which in individual cases render the outlook especially unfavorable are deep jaundice, extensive hemorrhage into the skin, delirium, and exhaustion.

XXXVII. ICTERUS INFECTIOSUS.

Weil's Disease; Acute Febrile Icterus.

Definition.—An acute infectious disease characterized by sudden onset with chill, followed by high fever and jaundice.

Etiology.—PREDISPOSING INFLUENCES.—The clinical picture suggests the severer cases of catarrhal jaundice and various febrile forms of gastro-intestinal disease that may run their course with or without jaundice. It corresponds very closely to the disease observed by Griesinger in Cairo, and Kartulis in Alexandria, and described under the designation *bilious typhoid* or *typhus biliosus.* Many cases reported as Weil's disease clearly do not belong to that category, such as santonin poisoning, septicæmia, abortive enteric fever, and the so-called hepatic form of enteric fever. The view that Weil's disease is a form of rheumatic fever complicated by a resorption icterus is not generally accepted. Some etiological considerations support the assumption that the disease is the manifestation of a specific infectious process. Among these are the following: The cases which correspond to Weil's description usually occur sporadically, but not rarely they appear in groups in circumscribed localities, and during the hot season. Males are more affected than females—90 per cent. Certain occupations exert a predisposing influence, butchers, tanners, and laborers in sewers being especially liable to the disease. It has been attributed to the drinking of contaminated water, and epidemics, especially among soldiers, have been ascribed to the swallowing of such water during bathing. The disease is most frequent between the twenty-fifth and the fortieth years of life. It is uncommon in childhood, and rare after fifty.

EXCITING CAUSE.—The researches of Jäger render it probable that an organism cultivated from the urine of living cases and from the organs of a case dead of the disease—*Proteus fluorescens*—is the infecting agent.

Symptoms.—The attack begins abruptly, usually without prodromes and often with a chill. Headache, vertigo, pain in the back and limbs occur. There is great lassitude. The temperature rises rapidly to 104° F. (40° C.) or higher, and is remittent in type. It lasts from eight to fourteen days. There are recurrences of the fever, and in a considerable proportion of the cases relapses occur. Stupor and delirium occur, and the resemblance of some of the cases to enteric fever may be striking—a resemblance increased by the early development of splenic enlargement. Jaundice makes its appearance between the third and fifth days and is of variable intensity, being in a considerable proportion of the cases deep and attended by clay-colored stools. The liver is increased in size and tender upon pressure. The urine is commonly albuminous, with hyaline and epithelial casts, and sometimes contains red blood-corpuscles. Hæmaturia is not very uncommon. In the fatal cases deep stupor, delirium, and coma precede death. There is rapid wasting during the attack. Muscular pains persist after the defervescence and are among the last symptoms to disappear. Angina tonsillaris is occasionally an early complication. Herpes facialis and other cutaneous lesions, as erythema and hemorrhage into the skin, have been observed. A group of the graver cases are hemorrhagic. Parotid bubo is a rare complication. The duration of the attack varies from two to four weeks, and the convalescence is slow.

Diagnosis.—The direct diagnosis rests upon the occurrence of jaundice with the symptoms of an acute severe infection, quite unlike ordinary catarrhal icterus on the one hand, and without the phenomena of the specific infections on the other. The age, sex, and occupation of the patient are to be considered. It is probable that some of the local epidemics reported as catarrhal jaundice of severe type have been outbreaks of Weil's disease.

Prognosis.—The death-rate is low, most of the cases terminating in recovery.

XXXVIII. GLANDULAR FEVER.

Definition.—An infectious disease of children, characterized by sudden onset, erythematous angina, enlargement of the tonsils, high fever of short duration, and swelling and tenderness of the lymph-nodes of the neck, particularly those along the posterior border of the sternocleidomastoid muscles.

Etiology.—PREDISPOSING INFLUENCES.—Certain individuals, particularly in childhood, exhibit a peculiar irritability of the superficial lymphatic glands, some of which become enlarged and painful in almost any disease, however trifling. Enlargement of the lymph-nodes is characteristic of measles, rötheln, tuberculosis, syphilis, and many other diseases, and, when acute, is commonly attended with some degree of fever. Glandular fever—*Drusenfieber of Pfeiffer*—is a definite nosological entity, the predisposing influences to which, except that it is almost exclusively a disease of childhood, are wholly unknown. It occurs sporadically and in epidemics.

The EXCITING CAUSE has not yet been determined.

Symptoms.—The onset is sudden, with a rise of temperature to 101°-103° F. (38.5°-39.5° C.). There is pain on moving the head and neck, together with nausea, vomiting, and abdominal pain. The angina is not as a rule intense nor of long duration. The adenopathy shows itself upon the second or third day, the glands becoming enlarged in varying degree to the size of a walnut, painful and tender to the touch. There is slight periglandular œdema, but no general swelling or redness, and but little difficulty in swallowing. In a limited proportion of the cases substernal uneasiness and an unproductive, paroxysmal cough are the signs of implication of the tracheal and peribronchial glands. The axillary, inguinal, and mesenteric glands are sometimes involved. The fever is of short duration but the enlarged glands only slowly undergo involution. Suppuration has been noted.

Diagnosis.—The direct diagnosis rests upon the presence of the foregoing symptom-complex. The differential diagnosis must be made by exclusion.

Prognosis.—Recovery is the rule. The occurrence of suppuration in some of the affected glands, otitis media, retropharyngeal abscess, and severe, even hemorrhagic nephritis may protract the illness or cause it to terminate in death; but these accidents are infrequent.

XXXIX. MILIARY FEVER.

Sweating Sickness.

Definition.—An acute infectious disease characterized by fever, profuse sweating, and an eruption of miliary vesicles.

Miliary fever, or the sweating sickness, prevailed extensively in England in the fifteenth and sixteenth centuries. Outbreaks have occurred within recent years in France, Italy, and Austria. They have sometimes extended over wide areas; more frequently they have been limited to districts or villages. These epidemics have lasted in some instances three or four weeks; in others they have run their course in a week or ten days. When the disease shows itself it attacks, like influenza and dengue, a large number of persons in a very short time.

Etiology.—Nothing is known of the pathogenic principle which gives rise to miliary fever.

Symptoms.—After prodromal symptoms of short duration, the attack sets in suddenly with moderate fever, profuse sweating, and epigastric distress. These symptoms are followed by an erythematous rash and the copious eruption of miliary vesicles, most abundant upon the neck and trunk. In the more severe cases the symptoms of an intense infection—high fever, profound depression, hemorrhage, and delirium—are present. Desiccation and desquamation occur. The duration of the disease varies from one to four weeks.

Diagnosis.—Direct.—This rests upon the development of an acute illness, characterized by the above symptoms, in a large proportion of the inhabitants of a locality in rapid succession, and the absence of the symptoms of influenza, dengue, or other infectious maladies.

Differential.—The sweating might suggest malaria, but the orderly succession of chill, fever, and sweat which characterize the ague fit in the regularly recurring forms, the well-defined periodicity, the effects of quinine, and the presence of Laveran's parasite in the blood would settle any doubt. Influenza bears a strong resemblance to miliary fever. Its pandemic prevalence, the prominence of catarrhal symptoms, the frequency of high fever, and the absence of the vesicular rash determine the question of diagnosis. Dengue differs from miliary fever in its geographical distribution, being a disease of tropical and subtropical climates; in its mode of onset and course; in its early arthropathy; in its eruptions; and in the fact that profuse sweating and a copious vesicular eruption are uncommon. Rheumatic fever might suggest miliary fever by the abundant perspirations which are common; but the prominence and migratory character of the arthritis, the higher fever, and the sporadic or endemic occurrence would at once dispel any uncertainty in regard to the diagnosis.

Prognosis.—In the more malignant forms death occurs in the course of several hours. The mortality in the early course of epidemics is high. In some of the recent outbreaks the death-rate has been low.

XL. FOOT-AND-MOUTH DISEASE.

Aphthous Fever.

Definition.—An acute infectious disease of cattle, sheep, and pigs, but also met with less frequently in other domestic animals, characterized by fever, salivation, and a vesicular eruption upon the mucous membrane of the mouth, nose, and conjunctiva, less frequently also upon that of the vulva and upon the udder and teats. In the sheep, goat, and pig the affection manifests itself particularly about the hoof, while in the horse only the oral mucous membrane is involved. This disease, which occurs in widespread epizootics, is readily transmissible to man, and numerous epidemics have been described. It is more common as a sporadic affection.

Etiology.—PREDISPOSING INFLUENCES.—All those occupations which involve contact with animals suffering from the disease, their fodder or manure, or the stables in which they are housed, predispose to the disease.

EXCITING CAUSE.—The infecting principle has not been isolated. Its presence in the contents of the vesicles and in the saliva, milk, and urine, has been demonstrated by intravenous and intraperitoneal injection. It retains its virulence in stalls, fodder, and dung heaps for a period of several months, and is capable of transportation to indefinite distances by fomites. The most common source of infection in man is by the raw milk of infected animals. Herbwig's experiments upon himself and his assistants demonstrated this mode of transmission beyond doubt. The cream also contains the virus, as well as butter, curds, and cheese made from the infected milk. The possibility of acquiring the disease by handling the meat of infected animals is to be considered, though prolonged cooking may render it innoxious as an article of food.

Symptoms.—In man, after an incubation period of from three to five or eight days, the disease begins with lassitude and pains in the head, back, and limbs. These symptoms are associated with dryness of the mouth, difficulty in swallowing, and nervous symptoms, such as vertigo and insomnia. Complete loss of appetite, vomiting, and fever ensue. The last is, however, by no means a constant symptom. In some cases there is nose-bleeding. If the infection has occurred by way of the oral mucosa there are seen, in the course of a day or more, vesicles which come out in successive crops and are preceded by a more or less diffuse and very painful inflammation. In the course of a short time the vesicles increase in size and then rupture, leaving superficial ulcers which gradually heal with scars. There is salivation, great pain in eating, and a fetid breath. Urticaria and roseolous and scarlatiniform rashes appear, and in some cases crops of vesicles resembling the vesicles upon the mucous membrane of the mouth. The last gradually undergo desiccation and healing without ulceration. When infection takes place by way of lesions upon the skin, constitutional symptoms develop first, vesicles appear near the seat of infection, the above-described rashes follow, and sometimes, but by no means invariably, the vesicles subsequently develop in the mouth.

Diagnosis.—DIRECT.—This rests upon the presence of the foregoing symptoms in an individual who has been exposed to the danger of contact with infected animals or their surroundings, or of eating infected food products. In a doubtful case, experimental inoculation should be practised and for this purpose the calf, on account of its greater susceptibility, should be selected.

DIFFERENTIAL.—The ordinary form of aphthous stomatitis is not attended by the constitutional symptoms of a severe infection, nor accompanied by cutaneous rashes. The vesicles run their course so rapidly that they are seldom seen prior to the formation of the circumscribed painful ulcers, with sharp edges and yellow bases, to which they give rise. The acute exanthemata may be suggested by the constitutional symptoms and the buccal and cutaneous eruptions. Varicella, however, usually lacks the fever and lassitude .of foot-and-mouth disease; measles is characterized by catarrhal symptoms not present in the affection, and scarlet fever by a diffuse non-vesicular angina and generalized erythema developing within forty-eight hours after an abrupt onset. Diphtheria, which may be suggested by the appearance of the ulceration in the mouth in certain cases, reveals its pathological identity by the Klebs-Löffler bacillus. In any suspicious case the etiological factor in diagnosis is important.

Prognosis.—In a majority of the cases in adults recovery takes place in two or three weeks. Owing to the difficulty in taking food, and the serious nature of the gastro-intestinal derangements in children, the outlook is far more serious, and progressive emaciation and debility are frequently followed by death. Septic infection by way of the mucous and cutaneous lesions may prolong the illness.

XLI. ERYSIPELOID OF ROSENBACH.

Erythema Migrans; Erythema Serpens.

Definition.—An erythematous inflammation of the fingers, due to the inoculation of an, as yet, undetermined pathogenic principle, associated with putrescent animal matter, and characterized by swelling, tension, dark red or purplish discoloration, well-defined edges, and a tendency to advance from the point of origin, with moderate pain, some itching and burning, and the absence of constitutional symptoms.

Etiology.—PREDISPOSING INFLUENCES.—Erysipeloid is a comparatively rare disease. It has been especially studied by Rosenbach in Germany (1887), and Gilchrist (1904) and Jopson (1908) in this country. Occupation constitutes the most important predisposing influence. The affection occurs almost without exception among those who are engaged in handling dead animal matter under conditions which render it liable to putrefaction, as dealers in fish (especially shell-fish), game, and poultry, and butchers and cooks. Medical students, laboratory workers, and taxidermists are also liable to the infection. Those who handle crabs are especially exposed to the danger of contracting erysipeloid, which is sometimes spoken of as "crab cellulitis."

EXCITING CAUSE.—Rosenbach and others have described a cladothrix, but subsequent investigators have failed to obtain such an organism. The manner of inoculation, the nature of the lesions, the constant clinical course of the disease, and the pathological findings, which are those of an inflammation of the entire corium, and, to some extent also, of the subcutaneous tissue, are suggestive of a specific infection. No specific organism has, however, been demonstrated.

Symptoms.—The infection results from an injury to the skin. There may be a number of small wounds. The period of incubation varies from a few hours to several days, being commonly, however, about forty-eight hours. The inflamed skin is tense, livid, elevated above the level of the adjacent surface, and characterized by a sharply defined border which advances toward the hand and may invade the palm, the dorsum of the hand, and other fingers than the one first affected. As the border advances resolution takes place in the part first attacked. Suppuration does not occur nor do vesicles, as a rule, develop. Complete recovery takes place in a period varying from two or three to ten or fifteen days. Fever and other constitutional symptoms do not occur.

Diagnosis.—The direct diagnosis rests upon the foregoing characters and may usually be made without difficulty. The differential diagnosis from the cellulitis caused by pyogenic organisms rests upon the mode of infection in the latter, the occurrence of suppuration, and the presence of pus-producing bacteria in the exudate; and from erysipelas by the absence of constitutional symptoms and the trifling nature of the erysipeloid affection. The prognosis is uniformly favorable.

XLII. ERYTHEMA INFECTIOSUM.

In 1896 Escherich described a feebly contagious disease of childhood characterized by a rose-colored maculopapular rash with trifling subjective symptoms, among which a mild erythematous sore throat is mentioned. The incubation period varies from six to fourteen days and the rash appears first upon the face and consists of maculopapules, in some instances sharply marginate, in others gradually fading into the healthy skin. It disappears momentarily upon pressure. In the course of twenty-four hours it invades other parts of the body, sometimes presenting the appearance of urticaria. It is especially marked upon the extensor and outer surfaces of the extremities. It lasts from six to ten days, fading first upon the face, where it first appeared, and is not followed by desquamation. The superficial lymph-nodes are not enlarged. The attack does not confer immunity from scarlet fever, measles, or rubella. No outbreaks have been observed in America. The direct diagnosis rests upon the character of the rash and its distribution, the absence of constitutional symptoms, and mild transmissibility. The differential diagnosis from these diseases depends upon the absence respectively of their specific etiologic and clinical manifestations. Erythema infectiosum bears only the most remote resemblance to scarlet fever, measles, rubella, urticaria, and certain drug rashes.

II.

THE DIAGNOSIS OF DISEASES CAUSED BY ANIMAL PARASITES.

A. DISEASES DUE TO PROTOZOA.

i. Psorospermiasis.

This term is applied to the diseases produced by the sporozoa other than hæmospiridia—protozoa that only live parasitically in the cells, tumors, or organs of other animals. They are mostly cytozoa. This class includes Gregarinida and Coccidiidea. The latter is the cause of a disease common in the rabbit in which the liver is the seat of small whitish nodules, seen upon section to be dilatations of biliary ducts. Coccidia are found in the epithelial cells lining these cyst-like dilatations. Rainey's corpuscles or tubes are ovoid bodies containing sickle-shaped, unicellular organisms—Sarcocystis miescheriana—found within the sarcolemma of various animals, especially the hog.

Visceral Psorospermiasis; Coccidiosis.—Infection takes place by swallowing the spores or oöcysts containing spores. The gastric juice causes the spores to open and frees the sporocytes, which by way of the common duct reach the biliary ducts and penetrate the epithelial cells. A limited number of cases has been observed in man.

Symptoms.—Those of severe infection: fever (sometimes intermittent), dry tongue, nausea, diarrhœa, and tenderness over the liver and spleen, which are enlarged. The parasites have also been found in the ureters and kidneys. The diagnosis during life has not been made.

ii. Amœbic Dysentery.

Definition.—A colitis caused by Amœba dysenteriæ and characterized by pain, tenesmus, and frequent stools containing mucus and blood. Dysentery occurs as an acute and chronic disease. Liver abscess is common.

Etiology.—Predisposing Influences.—Dysentery is more prevalent in tropical countries. In Egypt and India it is endemic, frequently epidemic. It occurs sporadically in all temperate climates. It is a water-borne disease and infection may take place by drinking contaminated water, or eating raw vegetables washed with it.

Exciting Cause.—Amœba dysenteriæ, first described by Lambl in 1859, later by Lösch in 1875, is classed among the Rhizopoda. This organism frequently exhibits the differentiation between the hyaline ecto- and the granular endosarc very clearly, especially in the pseudopodia. It contains a vesicular nucleus and contracting vacuoles. Amœbæ are seen in great numbers in the stools, being found especially in the shreds of [illegible] or pus, in the pus of liver abscess and abscess in other positions which occasionally occur, and in the purulent expectoration in hepatic [illegible] discharging by way of a pulmonary fistula. An amœba frequently contains red blood-corpuscles. They can be grown in cultures from the stools and intestinal ulcers but not readily alone, a symbiotic organism being required. Amœba coli has been found in the stools of healthy persons. There may be different varieties, of which some are non-pathogenic.

The lesions to which this organism gives rise are situated in the large bowel, sometimes reaching as high as the lower part of the ileum. They consist of an inflammatory infiltration of the submucosa, followed by necrosis and sloughing of the mucous membrane, which results in the formation of circumscribed round, oval, or irregular ulcers with overhanging borders. The base of the individual ulcer consists, according to stage of development, of the submucous, muscular, or serous coat of the gut. There is a remarkable tendency to extend by undermining the mucous membrane with the formation of deep, serpiginous ulcerating tracts or fistula. In severe cases the entire intestine is greatly thickened and extensively ulcerated. When the process is less extensive, the rectum, the hepatic and sigmoid flexures, and the cæcum are points of selection. Healing leads to extensive new formation of fibrous tissue and constriction of the bowel.

The lesions of the liver consist of, (a) local necrosis of hepatic tissue in scattered patches, and (b) abscess formation. The abscess may be multiple and scattered throughout the organ, or single. The latter are usually situated in the right lobe and under the convex or diaphragmatic surface; less frequently near the concave intestinal surface. In the former case rupture frequently takes place into the lung or pleura. Less commonly, according to its location, the abscess may rupture into the inferior vena cava, pericardium, peritoneum, stomach, intestine, or the portal or hepatic vein.

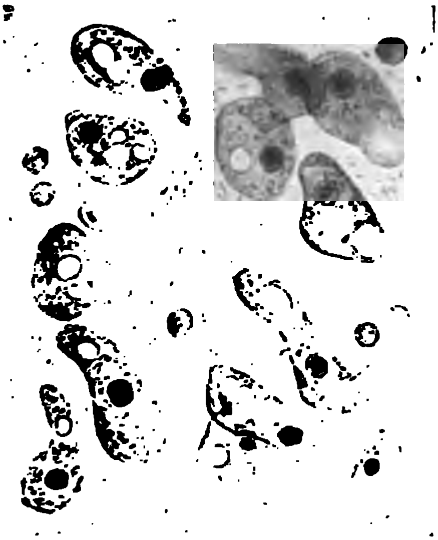


FIG. 283.—Amœba dysenteriæ.

Symptoms.—ACUTE FORM.—This form is characterized by sudden onset, pain, tenesmus, frequent stools containing blood and mucus. Large sloughs may be passed. There is fever, not often intense. Rapid emaciation occurs and the patient may die in the course of a week or two. Hemorrhage of the bowels may take place or perforation with peritonitis. Recovery is, however, the rule. In a considerable proportion of the cases the disease becomes chronic. There is a moderate leucocytosis—9000 to 16,000.

CHRONIC FORM.—The disease may be insidious in onset. There are subacute dysenteric attacks with pain, frequent stools, mucus and blood, and slight fever. These spells alternate with periods of constipation. The patients may have fairly good health, but are liable to indigestion, and the attacks are readily brought on by errors of diet, over-fatigue, sudden chilling and the like.

Diagnosis.—DIRECT.—The intestinal symptoms—pain, tenesmus, frequent stools with blood and mucus—justify a diagnosis of dysentery; the presence of amœbæ in the discharges, a diagnosis of amœbic dysentery, alike in the acute and chronic cases.

Hepatic abscess may be entirely latent. More commonly there is enlargement of the liver at its upper or lower aspect with recurrent chills, fever, sweating, local pain, and œdema. The leucocytosis is high. With the establishment of a pulmonary fistula there is dark expectoration containing amœbæ.

DIFFERENTIAL.—*Bacillary Dysentery.*—The diagnosis rests upon the absence of amœbæ and the agglutinating power of the blood-serum for the bacilli, and the more pronounced toxæmia. *Proctitis.*—Tenesmus and mucohemorrhagic stools may suggest dysentery, but the slight and transient nature of the attack and its manifest local character are of diagnostic importance.

Prognosis.—Many cases recover but the tendency to recurrence is marked. The mortality is about 25 per cent. Hepatic abscess adds greatly to the gravity of the outlook.

iii. Trypanosomiasis.

Sleeping Sickness.

Definition.—A chronic disease caused in man by Trypanosoma gambiense and characterized by undulant fever, rapid pulse, weakness, loss of flesh, and frequently a protracted lethargy.

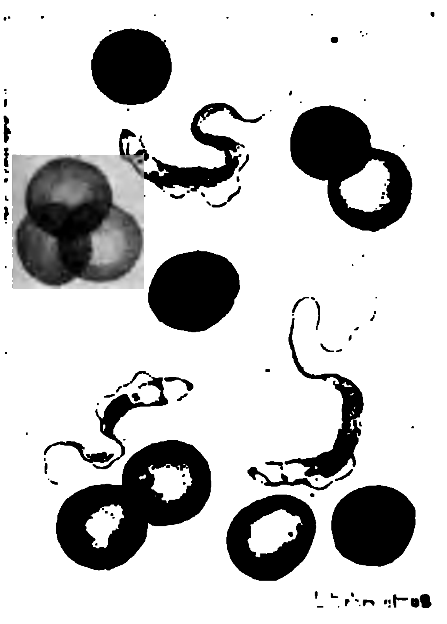


FIG. 284.—Trypanosoma gambiense.—After Bruce and Nabarro.

Etiology.—PREDISPOSING INFLUENCES.—As a disease of horses and cattle trypanosomiasis is known as *surra* in India and the Philippines and is prevalent in South Africa. Africa has, in fact, no less than six trypanosomal diseases, all of them very important: nagana, dourine, galziekte, zouspana, Gambian horse disease, and human trypanosomiasis. Human trypanosomiasis is widely distributed in Uganda and the West Coast of Africa. It is conveyed by the tsetse fly.

EXCITING CAUSE.—This flagellate protozoön was first discovered by Gruby in the blood of the frog in 1843. The organism is a unicellular, elongated body having an undulating fold or membrane upon the dorsal edge which terminates in a flagellum of varying length. Stained specimens show a large nucleus and a small chromatin mass near one pole. It has recently been grown by Novy on artificial media in the laboratory. It was later noted to be a common blood parasite in birds and fishes. It was found in 1878 by Lewis in the rat and subsequently in cattle and horses by Evans (1880) and Bruce (1895). Dutton first recognized trypanosomes in the blood of human beings in 1902. Trypanosomes have been observed with great frequency in the sleeping sickness or African lethargy and have a causal relation to that disease.

Symptoms.—As in the case of rats and other animals, trypanosome infection may be latent in human beings. Trypanosome Fever.—The symptoms consist of irregular fever, rapid pulse, weakness, swelling of the lymph-nodes and spleen, and œdema of the feet. This form of the disease has been produced in monkeys by inoculation. Sleeping Sickness.—The period of latency may be extremely prolonged, in some instances reaching five years. In a case of Manson's, symptoms developed in a fortnight. The early symptoms are dulness, apathy, headache, fever, difficulty in walking, tremor of the hands, and mumbling speech. The fever and drowsiness increase, the patient has to be aroused to take food and finally cannot be aroused at all. Death usually results from some complication, as septic meningitis.

Diagnosis.—Direct.—Exposure in an infected region, the history of an insect bite (especially by the African tsetse fly, Glossina palpalis), the varying but prolonged period of latency, the peculiar symptom-complex (especially the progressively deepening lethargy), and the presence of trypanosomes in the blood and cerebrospinal fluid are characteristic.

Prognosis.—Sleeping sickness is a very fatal disease. The duration after distinctive symptoms have occurred varies from three to twelve months.

iv. Dum-dum Fever.

Kala-azar.

Definition.—A chronic disease of the East due to a protozoön of the flagellate type and characterized by irregular fever, pulmonary congestion, anæmia, recurrent œdema of the feet, enlargement of the spleen and liver, and occasionally subcutaneous hemorrhages. This disease is identical with tropical cachexial fever and has been called Leishman-Donovan disease.

Etiology.—Predisposing Influences.—Dum-dum fever is prevalent in Oriental countries and occurs in Egypt. The European races rarely suffer.

Exciting Cause.—The bodies discovered by Leishman in 1900 and independently by Donovan in 1903, and regarded by Laveran and Mesnil as a new species of Piroplasma, have been found in the liver and spleen by puncture during life and also, in the majority of cases, in the circulating blood. They have been found in the mesenteric glands, intestinal ulcers, and bone-marrow. In stained smears of the fluid from the spleen and liver they appear as elongated oval or circular bodies with a spherical nucleus against the capsule and a rod-like body on the other side. They occur singly and in pairs and packed in phagocytic cells in the juice of the liver and spleen and in zoöglœa masses in the lung. Malarial parasites are not found.

Symptoms.—Constant enlargement of the spleen; usual but not invariable enlargement of the liver; moderate anemia; leucopenia; irregular fever prolonged for many months with occasional remissions; hemorrhages, subcutaneous and from mucous surfaces, especially the gingival and nasal mucosa; transitory œdemas, particularly of the legs and feet; and more or less marked pallor and pigmentation of the skin—these constitute the clinical picture. Dysentery and various secondary infections occur.

Prognosis.—Two forms of the disease are encountered, an intense form with recurrent attacks of high fever and rapidly developing cachexia, which terminates fatally in a few months, and a much milder form both as regards fever and cachexia, which runs a protracted course, death usually resulting from some intercurrent disease. The mortality, according to Leishman, varies from 70 to 96 per cent.

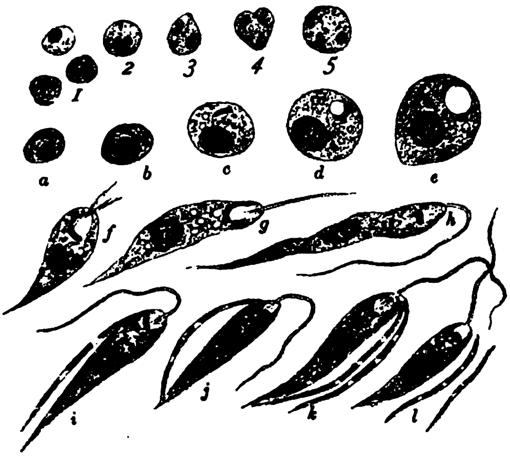


FIG. 285.—Leishman-Donovan bodies. 1, three parasites showing chromatin masses; 2, 3, 4, 5, parasites showing fission.—After Christophers. *a*, parasite from spleen; *b*, *c*, *d*, *e*, large parasites from cultures; *f*, *g*, *h*, bodies showing flagella; *i*, *j*, *k*, forms exhibiting unequal longitudinal fission; *l*, two spirillar forms separated from parasite.—After Leishman.

v. Malarial Fevers.

Definition.—A group of infectious diseases caused by the Hæmosporidia (Plasmodia) described by Laveran and transmitted to man by the bite of the similarly infected mosquito, comprising, (a) regularly intermitting periodical fever of tertian or quartan type, (b) irregular fever of remittent or continued type, and pernicious fever, often rapidly fatal, and (c) a chronic cachexia with anæmia and enlargement of the spleen.

Etiology.—PREDISPOSING INFLUENCES.—The *geographical distribution*, formerly wide, is becoming more restricted. The conditions necessary for the breeding of the mosquito, namely, heat and moisture, are everywhere predisposing factors to malaria. Regions of special prevalence are found in tropical and subtropical countries, as Panama, Central America, India, and Northern Africa, especially along the coasts and in the river basins. In Europe, Southern Russia and Lower Italy are still highly malarious. Germany and France are almost free, while the prevalence of the disease in Holland and England has practically ceased. In the United States the malarial fevers have steadily diminished since the colonial period. At the time of the first settlement of the country the prevalent fevers were malarial; as clearings were made and the soil tilled and drained, malarial fevers and enteric fever prevailed side by side until at length malarial fevers disappeared and enteric fever became predominant. Malarial fever has almost disappeared from New England and New York. It is now comparatively infrequent in Eastern Pennsylvania, New Jersey, and Maryland.

DESCRIPTION OF PLATE XIII.

The Parasite of Tertian Fever. (Drawn by Mr. Brödel for Thayer and Hewetson's paper, The Malarial Fevers of Baltimore, Johns Hopkins Hospital Reports, Volume V. We copy the original legend.)

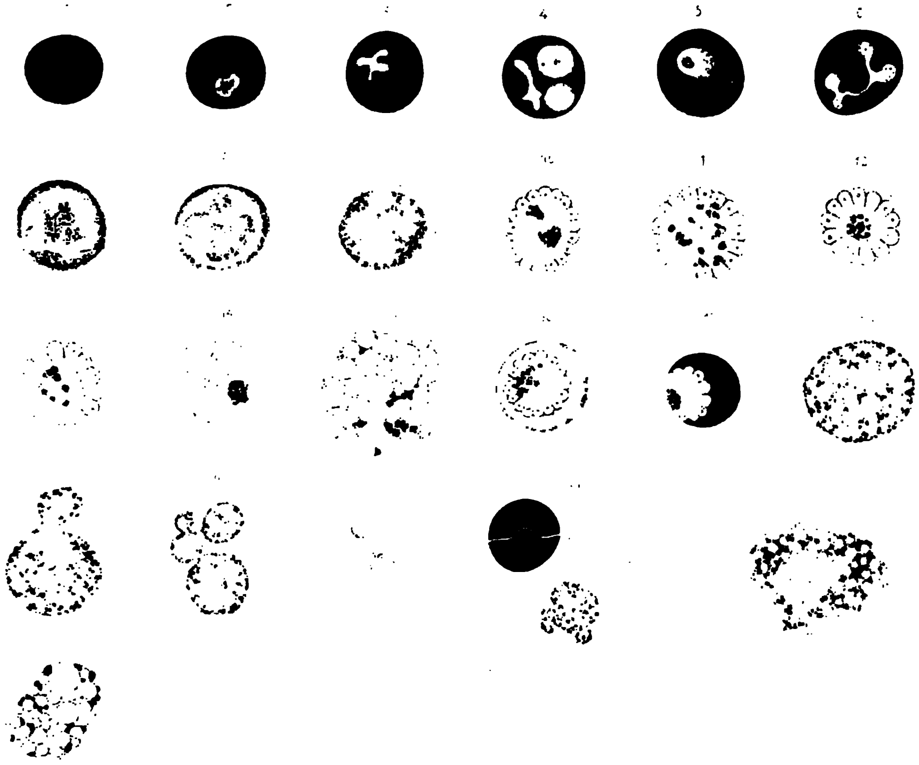
1. Normal red corpuscle.
2, 3, 4. Young hyaline forms. In 4, a corpuscle contains three distinct parasites.
5, 21. Beginning of a pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became, almost immediately, deformed and motionless, as shown in Fig. 21.
6, 7, 8. Partly developed pigmented forms.
9. Full grown body.
10–14. Segmenting bodies.
15. Form simulating a segmenting body. The significance of these forms, several of which have been observed, is not clear to the writers, who have never met with similar bodies in stained specimens so as to be able to study the structure of the individual segments.
16, 17. Precocious segmentation.
18, 19, 20. Large swollen and fragmenting extra-cellular bodies.
22. Flagellate body.
23, 24. Vacuolization.

The Parasite of Quartan Fever.

25. Normal red corpuscle.
26. Young hyaline form.
27–34. Gradual development of the intra-corpuscular bodies.
35. Full grown body. The substance of the red corpuscle is no more visible in the fresh specimen.
36–39. Segmenting bodies.
40. Large swollen extra-cellular forms.
41. Flagellate body.
42. Vacuolization.

PLATE XIII.

[illegible]



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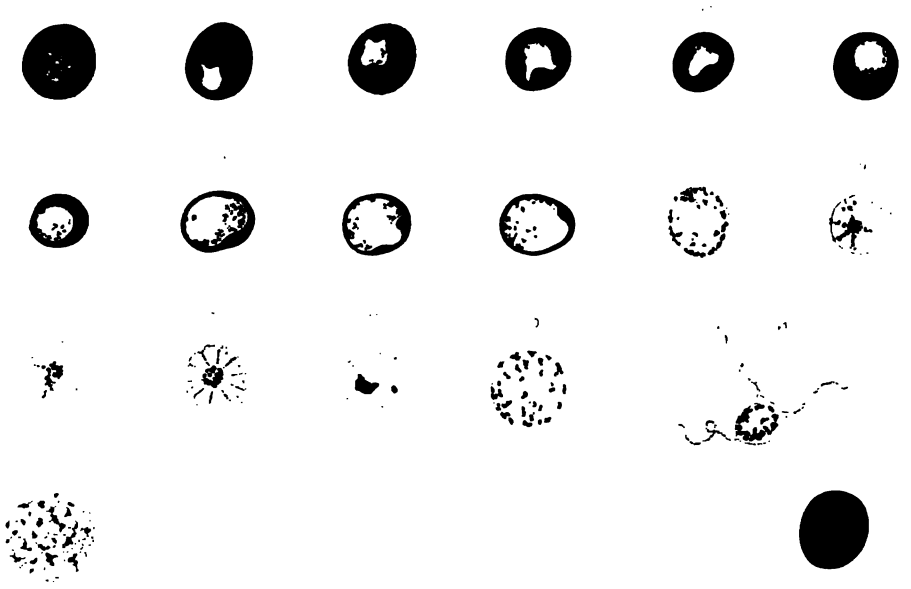


It still prevails in many regions of the South, especially on the Gulf Coast. The Northwestern States, the Pacific Coast, and the regions north of the St. Lawrence are practically free from it.

Season.—In the tropics the maximum prevalence corresponds to the rainy season, the minimum to the dry. In temperate climates there are a few cases in the spring, usually relapses—*vernal intermittent.* The greater number of cases occur in the early autumn—primary infection.

Locality.—Genera of the subfamily Anophelinæ, the only mosquitoes in which the malarial parasite of man develops, are distinctly rural insects, breeding in small, shallow pools and stagnant waters, in contradistinction to Culicinæ, which prefer human habitations and cities and deposit their ova in tanks and cisterns. Hence malarial diseases prevail more extensively outside of cities.

EXCITING CAUSE.—To Laveran, a French Army surgeon, is due the credit of having discovered in 1880, in Algiers, in the blood of patients suffering from malarial fever, the hæmatozoön which he recognized as parasitic and regarded as the cause of the disease. A great number of observers have contributed to our knowledge of the subject. Among the most important steps in the development of that knowledge are the following: that the febrile paroxysm coincides with the sporulation or segmentation of a group of parasites; that the tertian, quartan, and pernicious fevers are due to different parasites; that infection takes place by the bite of the mosquito, species of the subfamily Anophelinæ; that the infecting mosquito must itself be previously infected by the blood of an individual suffering from malaria; and that the malarial parasites of man require two different hosts for their complete development—the asexual cycle taking place in the blood of man, the intermediate host, and the sexual cycle in Anophelinæ, the definitive host. So far as our present knowledge of the life history of the malarial parasites goes they exist only in the mosquito and man.

The parasite belonging to the class Sporozoa, order Hæmosporidia, has received many different names. It was designated *Plasmodium malariæ* by Marchiafava and Celli, and this term, although unsuitable according to the rules of zoölogical nomenclature, has remained in general use.

The Parasite in Man.—*Schizogonous Cycle.*—Three species are recognized, differing morphologically and in the form of fever which they cause. They are (a) the tertian (Plasmodium vivax), (b) the quartan (Plasmodium malariæ), and (c) the estivo-autumnal (Plasmodium immaculatum).

TERTIAN PARASITE (*Plasmodium vivax*).—This species is the cause of tertian fever. Its cycle of development occupies forty-eight hours. It appears first in the red blood-corpuscles as a round or irregular unpigmented body which gradually increases in size. In the course of a few hours it has become ring-shaped and shows fine melanin granules. It contains a large nuclear body in which there is a small chromatin mass. There are now active amœboid movements which do not cease upon exposure to the temperature of the room. The affected blood-corpuscles become enlarged and lose their color. The pigment increases in amount. Toward the end of forty-eight hours the full-grown parasite occupies the greater part of the swollen corpuscle. At this time many of the parasites undergo the process known as segmentation or sporulation, in which the melanin granules are collected into a compact mass and the protoplasm divides into spores, numbering from 15 to 20, mostly collected into an irregular heap around the pigment mass, sometimes having a radial arrangement. The spores—merozoïtes—which represent the sexually undifferentiated individuals, finally separate from the central mass of pigment granules and from each other, pass into the blood-serum, and, attacking fresh blood-corpuscles, cause subsequent paroxysms of fever. Some of the full-grown tertian parasites do not undergo this process of sporulation, but, attaining a size larger than the red corpuscles, show abundant coarse pigment granules in active commotion and represent the sexually differentiated forms—gametocytes.

QUARTAN PARASITE (*Plasmodium malariæ*).—This species is the cause of quartan fever. Its cycle of development is seventy-two hours. It appears abruptly after the paroxysm in the form of a small, unpigmented body with sluggish amœboid movements on the surface of the red corpuscle. As it increases in size it penetrates within the corpuscle, where it presents an appearance very much like that of the tertian parasite, but smaller. At the end of twenty-four hours melanin granules, coarser than those of the tertian parasite and mostly situated at the periphery, begin to form. As the pigment increases and the parasite develops the amœboid movements become more sluggish and finally cease. Forty-eight hours after the attack the parasites have attained a diameter of one-half to two-thirds that of the corpuscle. In sixty hours they completely fill the corpuscles, of which only a narrow rim of a yellowish-green or brassy tint remains, which in turn presently disappears. The melanin grains assume a radial arrangement and move toward the centre, while the periphery now becomes pigmentless and shows the indication of commencing segmentation, which about the expiration of the third day is complete, each parasite separating into nine to twelve spores, a process corresponding to a fresh attack of fever. Sexually differentiated parasites—gametocytes—persist.

ESTIVO-AUTUMNAL PARASITE (*Plasmodium immaculatum; Plasmodium præcox*).—This parasite is the cause of estivo-autumnal fever and the various forms of malarial fever designated tropical, pernicious, and malignant. It is very small, not exceeding when fully developed one-third to one-half the diameter of the red blood-corpuscle. It is very active; its pigment is colored, scanty, and finely granular. The affected corpuscles are frequently shrunken, crenated, and brassy. Its cycle of development is forty-eight hours, but the processes of pigment accumulation and development are not often seen in the peripheral blood since they take place in the spleen, liver, bone-marrow, and cerebral capillaries. The spores are smaller than in the other species, arranged radially, and range in number from 7 to 12. After the illness has lasted some days—never at first—larger, crescentic, ovoid, and round bodies, highly refractive and containing central masses of coarse pigment, are seen. The crescentic and ovoid bodies do not undergo sporulation and represent gametocytes. If the disease continues these bodies increase rapidly and finally may be the only form present.

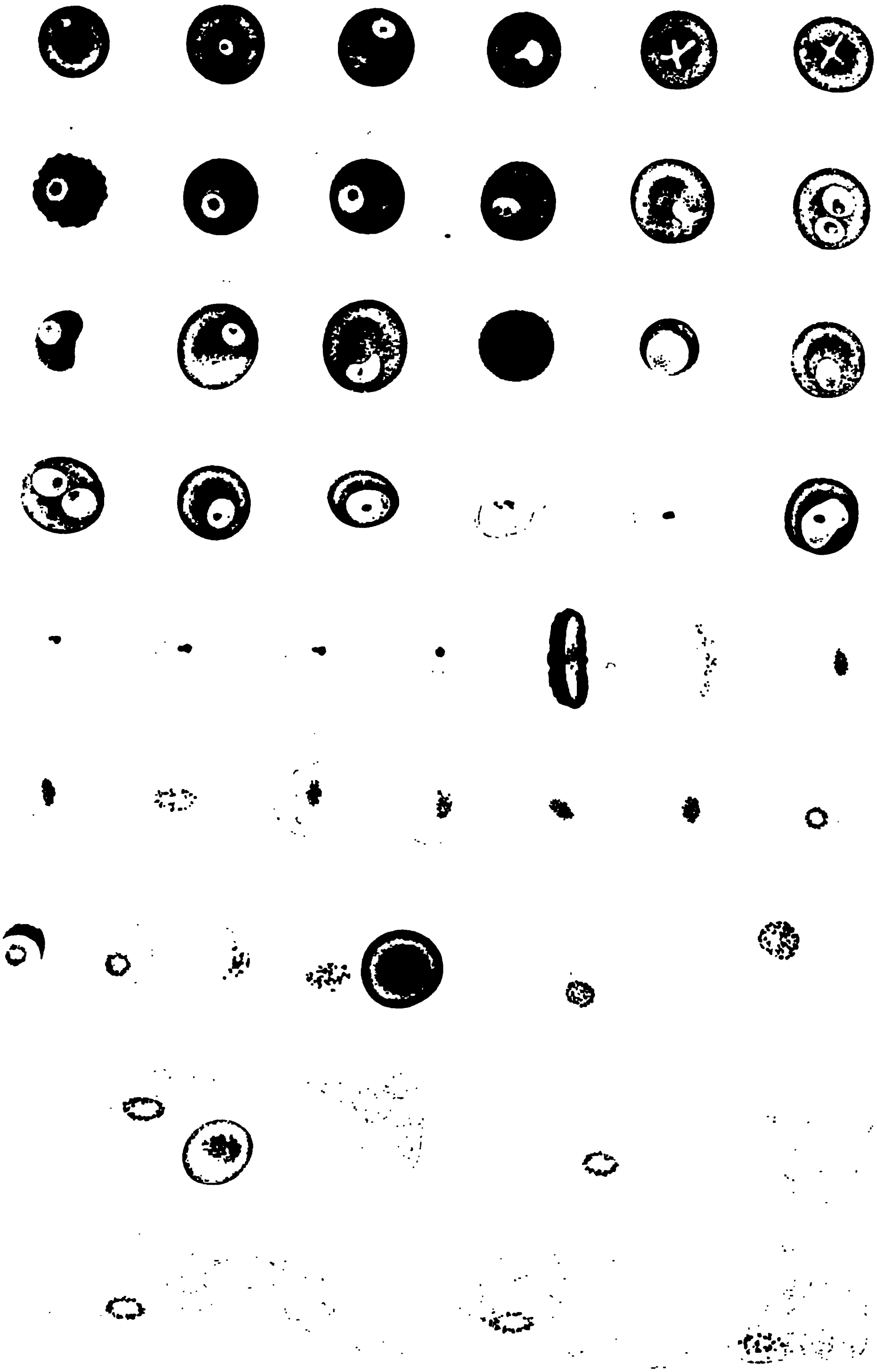
These sexually differentiated forms—*gametocytes*—are incapable of further development within the human host, but in the abstracted blood

DESCRIPTION OF PLATE XIV.

The Parasite of Aestivo-Autumnal Fever. (Drawn by Mr. Brödel for Thayer and Hewetson's paper, The Malarial Fevers of Baltimore, Johns Hopkins Hospital Reports, Vol. V. We copy the original legend.)

1, 2. Small refractive ring-like bodies.
3–6. Larger disc-like and amœboid forms.
7. Ring-like body with a few pigment granules in a brassy, shrunken corpuscle.
8, 9, 10, 12. Similar pigmented bodies.
11. Amœboid body with pigment.
13. Body with a clump of pigment in a corpuscle, showing a retraction of the hæmoglobin-containing substance about the parasite.
14–20. Larger bodies with central pigment clumps or blocks.
21–24. Segmenting bodies from the spleen. Figs. 21–23 represent one body where the entire process of segmentation was observed. The segments, eighteen in number, were accurately counted before separation as in Fig. 23. The sudden separation of the segments, occurring as though some retaining membrane were ruptured, was observed.
25–33. Crescents and ovoid bodies. Figs. 30 and 31 represent one body which was seen to extrude slowly and, later, to withdraw two rounded protrusions.
34, 35. Round bodies.
36. "Gemmation," fragmentation.
37. Vacuolization of a crescent.
38–40. Flagellation. The figures represent one organism. The blood was taken from the ear at 4.15 p.m.; at 4.17 the body was as represented in Fig. 38. At 4.27 the flagella appeared; at 4.33 two of the flagella had already broken away from the mother body.
41–49. Phagocytosis. Traced by Dr. Oppenheimer with the camera lucida.

PLATE XIV.



upon the slide or in the intestine of Anopheles the male elements—*microgametocytes*—form actively moving flagella—*microgametes*—which detach themselves and penetrate into the coarsely granular female forms—*macrogametes*—which they fecundate.

The independence of the three species of malarial parasites, distinguished by their morphologic and pathologic characters, has been fully established by the results obtained by the experimental inoculation of the blood of patients suffering from malaria into the veins of healthy persons. After a period of latency, varying with the particular species under investigation, the inoculated individuals have developed malarial fever, always of the same type as that of the patient from whom the blood was obtained.

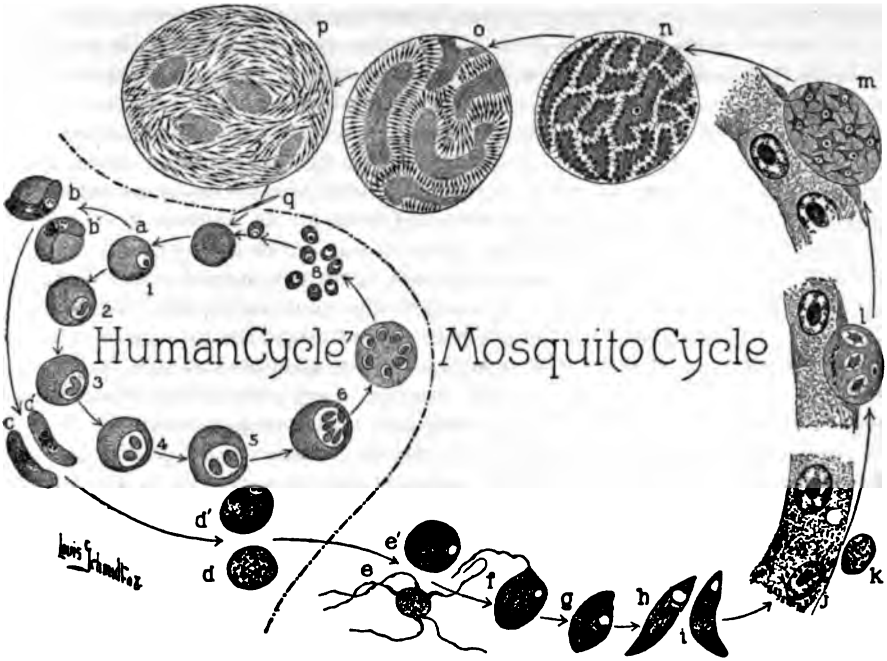


FIG. 286.—Diagram of the complete life-cycle of the estivo-autumnal malaria parasite. 1–7, stages in the development of the asexual cycle (schizogony); 8, spores or merozoïtes; *b b'*, *c c'*, *d d'*, gametocytes in blood of man, sexual cycle; *e'*, macrogamete; *e*, microgametocyte; *f*, macrogamete penetrated by microgamete; *g*, *h*, *i*, motile zygote, vermicule, or oökinete; *j–o*, stages of development in the stomach wall (sporogony); *p*, mature oöcyst showing sporozoïtes; *q*, sporoid.—Based upon the plates of Grassi.

The Parasite in the Mosquito.—The common genera of mosquito in temperate climates are *Culex* and *Anopheles*. Of each there are many species. The Culex appears to play no part in malarial infection, but wherever there is malaria the Anopheles is to be found. If the Anopheles has not been infected by sucking the blood of a human being suffering from malaria, it is of course incapable of communicating the disease by its bite, and the parasite does not develop in cold climates. These two facts serve to explain the presence of Anopheles in regions not malarious. Of the many species of Anopheles described, it is probable that in temperate climates the A. maculipennis is most active in the diffusion of malaria. The distinction between Culex and Anopheles under ordinary conditions within a house is an easy matter and depends upon the following facts:

CULEX: *The Mature Insect.*—The palpi in the female are short, projecting only a little distance beyond the base of the proboscis. The wings show no special markings. Resting upon the wall or ceiling Culex holds its posterior pair of legs turned up over its back, and its body, unless dragged down by the weight of blood, is nearly parallel to the wall or ceiling.

ANOPHELES.—The palpi in the female are of nearly the same length as the proboscis. The wings of different species show distinct mottlings, hence the names of species—*A. maculopennis*, *A. punctipennis*. The posterior pair of legs, when the insect is resting, rest upon the wall or ceiling or hang down, and the body is held at an angle of forty-five degrees with the surface upon which it rests. The sex-ripe forms of the malarial parasite, when taken into the stomach of Anopheles, rapidly mature with flagellation and fecundation. The resulting motile fusiform body bores into the wall of the mosquito's stomach and there rests, undergoing a definite cycle of development, with the formation of oöcysts at first oval, later globular. The nuclei within these cysts divide into a great number of daughter nuclei, which form sporoblasts, from which sporozoïtes develop. The mature oöcysts burst and discharge their sporozoïtes into the body of the host. Circulating in the blood these accumulate in the course of a few hours within the cells of the venenosalivary glands and are inoculated with the saliva by the bite of the insect. They are transformed in the blood of the human host into the amœboid form of the parasite and multiply by sporulation (schizogony) until they attain sufficient numbers to produce the paroxysm of fever. The early generations of parasites in the human host are asexual, sexual differentiation occurring later.

The asexual forms serve as the means of prolonging the infection in the human host; the sexual forms, sterile so long as they remain in the human host, become fertile in the mosquito and maintain the life and dissemination of the parasite.

Symptoms.—The cases may be grouped into the regularly intermitting fevers, the irregular, remittent, or continued fevers, and malarial cachexia.

(a) **The Regularly Intermitting Fevers; the Agues.—1. Tertian Fever. 2. Quartan Fever.**—The period of incubation varies from a few days to a fortnight. Latent malarial infection, not attended by symptoms, may be called into activity by removal from a malarious district, other change of climate, or by an attack of illness.

THE PAROXYSM.—The febrile paroxysm, known as the "chill" or "ague fit," may be divided into three stages: cold, hot, and sweating.

Cold Stage.—There are usually premonitory symptoms, consisting of lassitude, yawning, epigastric distress, sometimes nausea and vomiting, and headache. Shivering occurs and quickly passes into a fully developed rigor with chattering teeth, violent shaking of the whole body, and distressing sensations of cold. The face is cyanotic, the body and limbs covered with goose-flesh, cold to the hand, and showing, when tested by the surface thermometer, a subnormal temperature. At the same time the rectal temperature is high—105°-106° F. (40.5°-41° C.). During this stage nausea, vomiting, and headache may occur. The pulse is small, frequent, and tense. The duration of the stage varies from ten or fifteen minutes to an hour or more. The danger of the attack lies in the prolongation of the cold stage.

Hot Stage.—The sensations of cold are replaced by those of heat; pallor and cyanosis give way to flushing, and the appearance of collapse is followed by that of more or less intense fever, with bounding pulse, headache, and sometimes delirium. The rectal temperature does not rise, having as a rule reached its maximum about the conclusion of the cold stage. There is urgent thirst and the patient is distressed by subjective sensations of heat. The duration of this stage varies from thirty minutes to three or four hours.

Sweating Stage.—Perspiration starts in drops upon the forehead and face and soon covers the entire body profusely. In some cases the sweating is moderate. The duration of this stage is variable. At its conclusion the patient commonly falls into a sleep from which he awakes refreshed but weak.

The duration of the entire paroxysm varies from an hour or two, as is common among the inhabitants of malarious districts, to six or eight hours. The cold stage is sometimes slight and transient and occasionally not followed by a hot stage. A more common variation from type consists in the hot stage alone, followed by very slight sweating. During the paroxysm the spleen is usually tender and palpable, herpes labialis occurs, and there are the rational symptoms and physical signs of a mild bronchitis, which passes off with the sweating stage.

In the intervals between the paroxysms the patient commonly feels well and regards himself as in his usual health. The paroxysm is the result of a hæmodyscrasia, at once morphological and toxic, produced by the segmentation of the parasites.

1. Tertian Fever.—In this type, caused by the presence in the blood of the tertian parasite, the paroxysms recur every forty-eight hours or every third day. Hence the name tertian. If two groups of parasites, reaching maturity and undergoing segmentation every alternate day, are present, there are daily—quotidian—paroxysms, and the type is double tertian.

2. Quartan Fever.—The paroxysm caused by sporulation of the quartan parasites recurs about the end of seventy-two hours, or every fourth day, and is for this reason known as quartan. If two groups of parasites are present, maturing upon different days, paroxysms occur upon successive days followed by a free day—double quartan; if three groups are present the paroxysms occur daily—triple quartan, likewise quotidian.

The course of the regularly intermittent malarial fevers is greatly influenced by circumstances. Mild cases frequently recover without treatment, especially if removed from the opportunity of further infection and kept in bed. Untreated cases are, however, liable to relapse. The attacks yield promptly to proper treatment by quinine. Repeated reinfection or the persistence of the disease results in anæmia and hæmolytic jaundice, ultimately in malarial cachexia.

The Irregular, Remittent, Continued, and Pernicious Fevers.—This [illegible] tropical countries, [illegible] form occur in temperate [illegible] and in the autumn. [illegible] associated with the presence [illegible] characterized by irregularity [illegible] the fact that the parasite, [illegible] hours, is subject [illegible] groups which [illegible] The intensity is due [illegible] segments at the time of [illegible] the capillaries and peri[illegible]

[illegible] cases begin as irregular [illegible] without chills [illegible] temperature rise is gradual [illegible] anticipation of the par[illegible] rapidly converts inter[illegible] In other cases there is fever [illegible] and the clinical picture is suggestive [illegible] flushed face, a bounding [illegible] enlargement of the spleen. The temperature [illegible] C., with remissions and [illegible] Intestinal symptoms are not [illegible] a moderate bronchitis with [illegible] enlargement and tenderness [illegible] resemblance. Delirium may occur. [illegible] discoloration of the [illegible] a group of cases—*bilious remittent* [illegible] with nausea and vomiting [illegible] and misleading designation, [illegible] this group of malarial fevers.

[illegible]

[illegible] fevers is extremely variable. [illegible] the attack may run its course with [illegible] The clinical picture suggests [illegible] enteric fever—*typhus le[illegible]* [illegible] are more severe. The fever is [illegible] exacerbations. There is intense [illegible] and vomiting, with enlargement [illegible] tenderness—*bilious remittent fever.* [illegible] types are (i) the algid, (ii) the [illegible]

[illegible] begin with a prolonged chill. [illegible] sensations of cold. Gastric [illegible] distress, are prominent. Extreme [illegible] feeble, small pulse and rapid [illegible] in some instances attended with [illegible] may be present and with it great

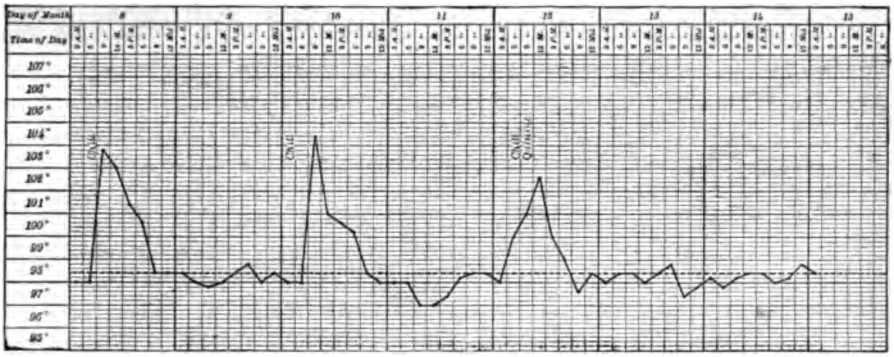


FIG. 287.—Tertian fever.—Craig. (*International Clinics.*)

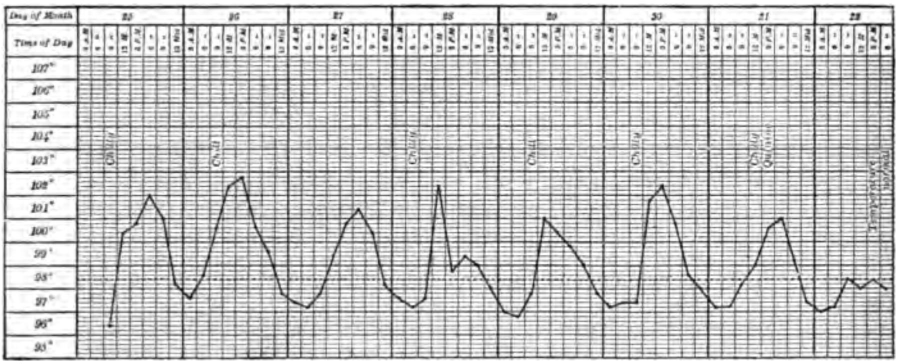


FIG. 288.—Double tertian fever.—Craig. (*International Clinics.*)

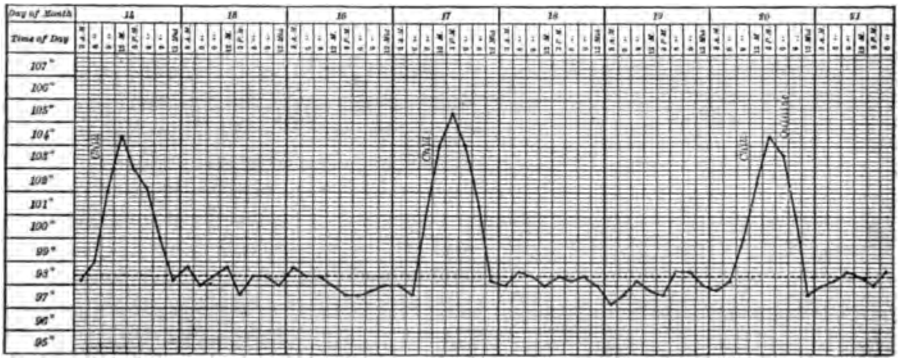


FIG. 289.—Quartan fever.—Craig. (*International Clinics.*)

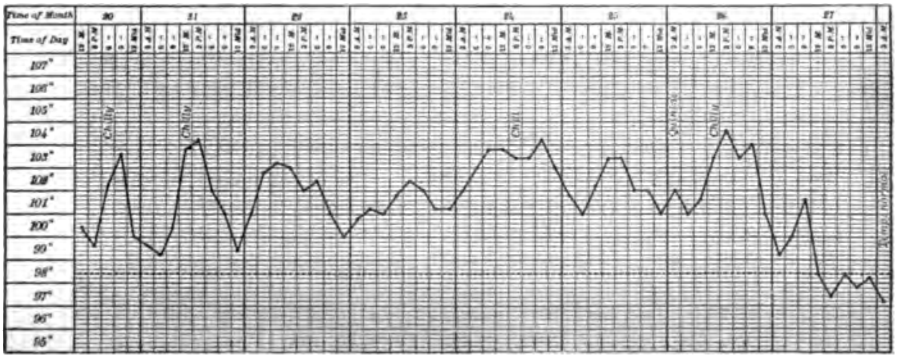


FIG. 290.—Estivo-autumnal fever.—Craig. (*International Clinics.*)

diminution of the urine. Fever is at first absent, the temperature being, as a rule, subnormal. Later irregular febrile exacerbations may occur. In default of energetic treatment death takes place in the course of a few days with the evidences of profound asthenia. Sudoral, syncopal, cardialgic, and choleraic varieties are described.

(ii) *Comatose Form.*—The attack begins abruptly with cerebral symptoms, as intense headache with acute delirium or stupor deepening to coma. In some cases the seizure may be apoplectiform. A chill may mark the onset, but this is not invariably the case. There is a hot, dry skin with high temperature. The patient may die without regaining consciousness; or he may recover consciousness in the course of twelve or twenty-four hours. The second or third attack is usually fatal.

(iii) *Hemorrhagic Form.*—This form is also designated hæmoglobinuria and is identical with the African black-water fever. It is rare in temperate climates, its chief distribution being on the Gulf Coast of the United States, Central America, Lower Italy, and Africa. The disease is malarial, but whether it is due to a special parasite or not remains to be settled. As a rule the patients have suffered from repeated attacks of malarial fever and are in poor health. Parasites have been found in the blood prior to, and in a more limited number of cases at the onset of, the attack. Later they are not found in a majority of the cases. The evidence that malarial hæmoglobinuria is caused by quinine is not conclusive. The attack begins with fever, to which, in the course of a short time, hæmoglobinuria supervenes.

(c) **Malarial Cachexia.**—Prolonged exposure in a malarious district with repeated infection by way of the parasites is frequently followed by the development of an anæmia of high grade with enlargement of the spleen. Emaciation, a muddy complexion with general cutaneous pigmentation, subcutaneous and retinal hemorrhages, breathlessness upon exertion, œdema of the ankles, are usual symptoms. There is irregular temperature varying from normal to subfebrile ranges with occasional exacerbations—102°-103° F. (39°-39.5° C.). The splenic enlargement is often massive, constituting the tumor known in the Southern States as "*ague cake.*" Hæmatemesis occasionally occurs and may be fatal.

Diagnosis.—DIRECT.—The recognition of the essential nature of the malarial fevers is not usually attended with difficulty. The two important tests are the presence of the blood parasite and the curative effect of quinine. A history of exposure, the well-defined periodicity of the regularly intermitting tertian and quartan fevers, enlargement of the spleen, herpes, and the absence of tuberculosis, syphilis, sepsis, or other causes of periodical fever are diagnostic criteria of secondary importance.

DIFFERENTIAL.—The estivo-autumnal form often closely imulates enteric fever. On the one hand the malarial parasite and control by quinine, on the other the Widal reaction and the powerlessness of any drug to arrest the course of the attack, are positive tests. The parasite may be demonstrated in fresh blood taken a few hours before the expected chill. In doubtful cases a carefully prepared cover-slip preparation of the blood may be stained for examination. The intermittent pyrexia which refuses yield to quinine in daily doses of 15 to 20 grains (grammes 1 to 1.3

e mouth or hypodermically is not malarial.

mb ague—blurred types which occur in

persistent infections or after inadequate treatment manifest themselves by indefinite symptoms and attacks of irregular fever or an afebrile temperature with an occasional chill. Here the therapeutic test is conclusive. In septic states with intermittent pyrexia, abscess, malignant endocarditis, hepatic fever and the like, a study of the blood is important, since leucocytosis, which does not often occur in uncomplicated malaria, is usually present in these conditions.

Prognosis.—The malarial fevers of the regularly intermitting forms and the estivo-autumnal fevers of temperate climates yield readily to treatment by quinine. If neglected or when reinfection occurs they recur from year to year with vernal or autumnal relapses. Malarial cachexia may thus develop. The prognosis in the tropical forms of estivo-autumnal fever is grave. Life can be saved in many of the cases only by the prompt, skilful, and judicious administration of quinine in sufficient doses, and the removal of the patient from the danger of reinfection. The prognosis in malarial cachexia is fairly good. Health may be regained by proper treatment sufficiently prolonged. The main indication is the avoidance of reinfection.

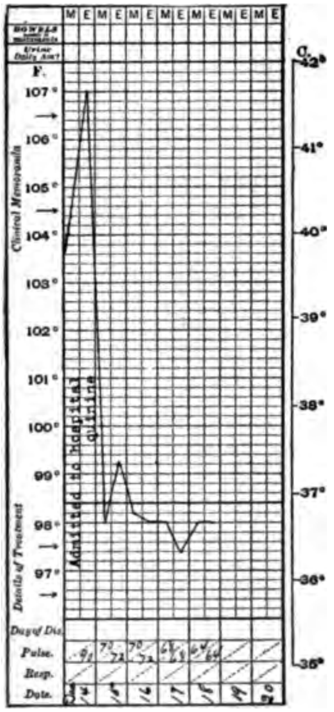


FIG. 291.—Malarial fever. Prompt effect of quinine.—German Hospital.

vi. Pellagra.

Definition.—A chronic disease of undetermined causation, characterized by a peculiar dermatitis of definite distribution, marked gastro-intestinal derangements, great physical and mental depression, profound disturbances of nutrition and terminal dementia. Its progress is at first periodic, later continuous. Its occurrence may be sporadic, endemic or epidemic. It is not directly transmissible from the sick to the well.

Etiology.—PREDISPOSING INFLUENCES.—The geographical distribution is largely restricted to northern Italy, Spain and the south of France. Cases have been observed in Egypt. Recent observations have shown it to be widely prevalent in the United States, especially in institutions for the care of the insane. Its prevalence is neither coterminous with nor restricted to maize-consuming districts. It is a disease of the poor and of rural populations. It occurs chiefly in middle life, but cases have been observed at every age. Women suffer more frequently than men.

EXCITING CAUSE.—Two hypotheses are defended: (1) That of Lombroso, who maintains that pellagra is caused by eating bad maize and is due to some poisonous principle associated with maize—an opinion until recently universally accepted; (2) That of Sambon, who holds that the presence of pellagra is closely associated with streams of running water and

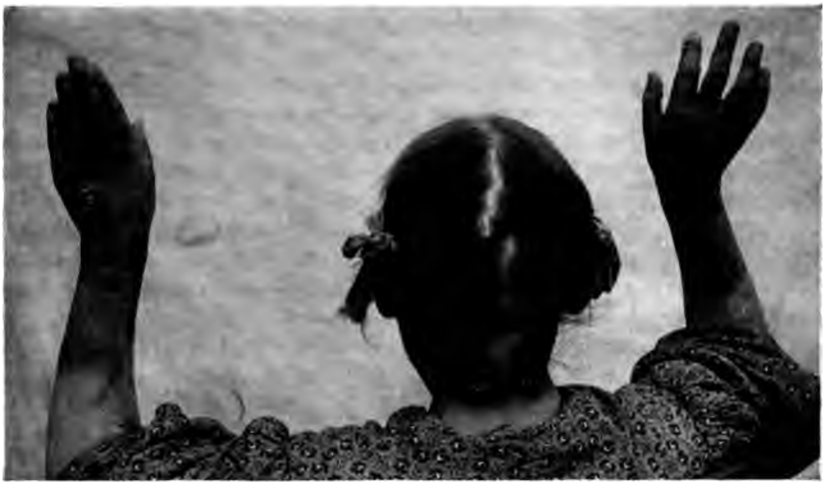


FIG. 291a.—The "pellagrous gauntlet," with cervical lesions known as the "Collar of Casal."—Zeller.

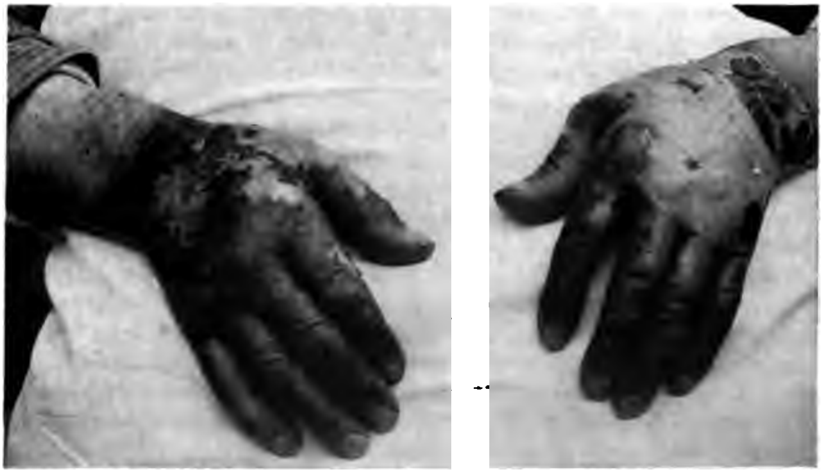


FIG. 291b.—Pellagrous eruption in stage of desquamation.—Zeller.

the presence of a suctorial fly of the genus *Limulium*, which abounds in certain districts bordering upon the streams.

Symptoms.—The early symptoms are not distinctive. They consist of weakness, sleeplessness, headache, vertigo, dyspepsia, muscular cramps and pains referred to the spine and joints. These attacks occur in the spring or summer and subside in the autumn, only to recur again in the following spring. Fever is not common except in the severer cases, in which irregular pyrexia—102°-105° F. (39.0°-40.5° C.) is frequently observed. The eruption is symmetrical and affects chiefly the parts exposed to the sun, as the hands, wrists, neck and upper part of the chest and the feet and legs. It has the characters of an intense erythema with petechiæ and in severe cases bullæ, which rupture and leave indolent ulcers. Upon its subsidence the skin is left thickened and deeply pigmented. The dermatitis recurs annually, but after the fourth or fifth year the integument involved undergoes atrophic changes. The nails and hair are not affected. After some years the symptoms become continuous. Dyspepsia and diarrhœa are pronounced. Chronic stomatitis characterized by salivation and a cardinal red or bluish black stippled tongue, often denuded of epithelium, is common. Wasting, weakness and mental depression progressively increase. Vertigo, unilateral or bilateral mydriasis, twitchings, tremors, epileptiform seizures of the cortical type and palsies occur. The reflexes are increased; ankle-clonus is often present. The gait is uncertain but not ataxic. Definite derangements of sensation do not occur. The later stages of the disease are characterized by melancholia, mania and dementia. At this period there are forms of palsy with diminished or absent reflexes, the signs of a peripheral neuritis. A majority of the cases in the United States have been discovered in asylums for the insane poor, but recent observations and a growing knowledge of the facts among practitioners render it probable that many obscure cases of ill health among the country people of the South and Southwestern States are pellagrous.

Diagnosis.—The direct diagnosis rests upon the foregoing periodical and progressive phenomena and may be made without difficulty. The resemblance to general paresis and leprosy is of the most superficial character.

Prognosis.—After the third or fourth annual periodic recurrence, especially when mental symptoms have shown themselves, the outlook is very unfavorable. Death occurs from progressive wasting and asthenia or from intercurrent disease.

VII. Epidemic Anterior Poliomyelitis. See p. 1326.

B. DISEASES DUE TO FLUKES—DISTOMIASIS.

The parasitic trematodes are widely distributed among vertebrate animals. These are important in man: (1) Fasciola hepatica—Distomum hepaticum; (2) Paragonimus westermani—Distoma pulmonis; (3) Fasciolopsis (Distoma) buski; and (4) Schistosomum hæmatobium—Bilharzia hæmatobia. The following clinical forms of distomiasis are to be considered:

1. Hepatic Distomiasis.—Several species of liver flukes have been observed in man, of which the most important is Opisthorchis sinensis, widely distributed in the East, especially in Tonquin, China, and Japan.

Imported cases have been encountered in the United States. This parasite is 10–14 mm. long by 2.4–3.9 mm. broad. The eggs are oval with a well-defined operculum at the pointed pole. They measure 0.027–0.030 by 0.015–0.0175. The parasite infests the gall-bladder and gall-ducts of domestic dogs and cats and human beings. They have also been found in the pancreas of human beings. Their number is sometimes enormous. The changes produced in the gall-ducts are local dilatations with sacculation and proliferation of the connective tissue of the wall, and in the liver interstitial hepatitis followed by atrophy.

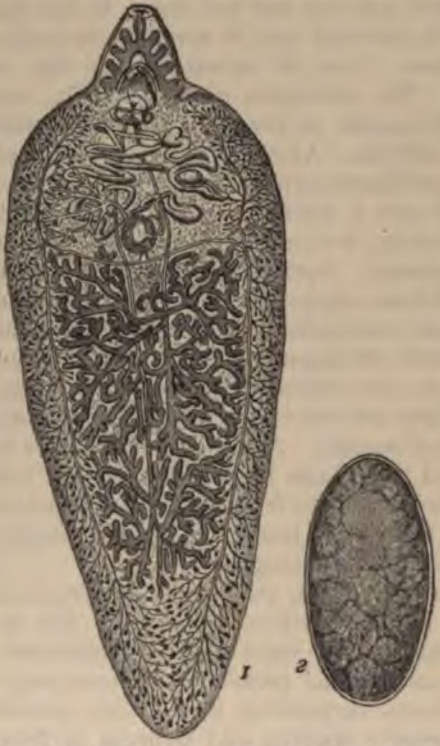


Fig. 292.—1. Fasciola hepatica.—After Claus. 2. Egg of parasite.—After Braun.

Symptoms.—Intermittent diarrhœa, sometimes bloody; enlargement of the liver, with pain, and jaundice which is intermittent; and slight fever. After two or three years œdema of the feet occurs, followed by anasarca and ascites. The ova are found in the stools. Recovery takes place, but relapses occur. The mortality is about 14 per cent.

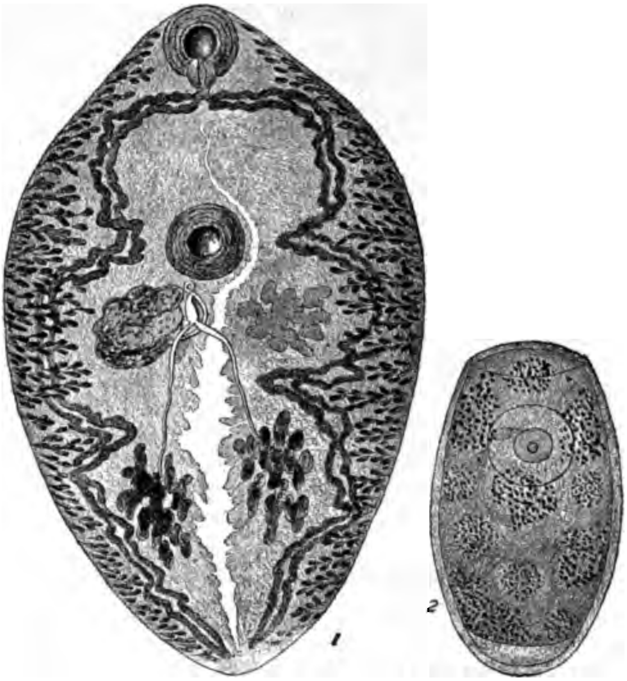
2. **Pulmonary Distomiasis.**—Paragonimus (Distoma) westermani has been observed in China, Korea, Formosa, and Japan. Imported cases have been studied in the United States. The body is reddish-brown in color and plump. It is 8–10 mm. in length, 4–6 mm. in breadth. The eggs are oval, brownish-yellow, thin-shelled, and have approximate average diameters of 0.09 mm. in length by 0.06 mm. in breadth. They are found in large numbers in the sputum.

Symptoms.—There is cough and blood spitting but the symptoms are usually slight. The patients are able to follow their occupations. Copious hæmoptysis sometimes occurs. Males are principally affected; women and children rarely. The mode of infection has not been discovered.

3. **Intestinal Distomiasis.**—Fasciolopsis (Distoma) buski has only been observed in the intestine of man. The cases have occurred in Eastern and Southern Asia. Seven cases only have been reported (Braun).

4. **Hæmic Distomiasis—Bilharziasis.**—A parasitic disease endemic in Egypt, Abyssinia, The Sudan, and in many other districts of Africa. There appears to be a centre of infection in Arabia. Elsewhere beyond the borders of Africa it is encountered in imported cases. The parasite—Schistosomum hæmatobium—was discovered by Bilharz in 1852. Unlike the other flukes the sexes are separate and the male carries the female in

æcophorous canal. The male is of a whitish color and 12–14 mm. in , varying from 1 mm. to 0.4–0.5 mm. in diameter. The dorsal surface posterior part of the body is covered with spinous papillæ. The is filiform, pointed at the ends, about 20 mm. in length, and 0.25 mm. meter. The eggs are oval, of a transparent yellow color, thin-shelled, rovided with a terminal spine. They vary greatly in size. They in water. The development of the embryo has not been worked out. er infection takes place by the mouth, the urethra, or through the bathing is unknown. The young specimens are found in the portal the sexes separate. Hence the males bearing the females penetrate



293.—1. Paragonimus westermani.—After Leukart. 2. Egg of parasite.—After Katsurada.

venous plexus of the pelvis, from which they reach the wall of the r and rectum, the ova being deposited in the tissues but wandering ans of the spine and being voided with the fæces and urine. They sily found in the latter, especially in the flakes of mucus present. remain in the tissues, causing inflammatory irritation, fibroid thick- and papillomatous growths. Others collecting within the bladder and undergo calcification, thus forming the nuclei of the vesical so common in bilharziasis. The ova may be transported to distant by the blood stream, and have been found in all the organs, though ll numbers.

ymptoms.—The infection is sometimes latent, the parasites giving no symptoms. This is especially the case while they remain in the vein. Early symptoms are catarrh of the bladder, with pain in the bladder and rectum and in the lumbar region. The urine is at first normal in appearance, but after a time there is tenesmus with bloody mucus and pus at intervals or daily. As the disease progresses the vesical inflammation becomes more intense, the urine contains blood and pus in increasing quantities, and calculi are found which produce their characteristic symptoms. The ureters, the pelvis of the kidneys, the kidneys, the rectum, and occasionally the vagina become involved. The nutrition is greatly impaired and death may result from general marasmus. In a considerable proportion of the cases it is due to uræmia, sepsis, or an intercurrent pneumonia. Perineal and urethral abscess formation is common.

Diagnosis.—The urinary symptoms are suggestive and the direct diagnosis may be made by finding the characteristic ova in the bloody urine or in the blood and mucus discharged from the rectum.

Prognosis.—In mild infections under circumstances in which reinfection can be avoided, the symptoms sometimes disappear. As a rule, the prognosis is highly unfavorable both as to mitigation of suffering and as to recovery.

Katsurada in 1904 described a fluke, closely resembling Schistosomum hæmatobium, to which he attributed an endemic disease, characterized by enlargement of the liver and spleen, cachexia, and ascites, and to which he gave the name S. japonicum. Three months later the same parasite was found by Dr. John Catto and named by Blanchard Schistosoma cattoi. The ova are smaller than those of S. hæmatobium, brownish in color, and not provided with the characteristic terminal spine. This parasite infests the blood-vessels of the intestinal canal and the organs related to it, and the ova are found in the fæces.

In Porto Rico there exists a rectal form of bilharziasis in which the ova are lateral-spined. Sambon has considered this a new species and has called it S. mansoni.

C. DISEASES DUE TO CESTODES.

Tapeworms; Hydatid Disease.

i. Intestinal Cestodes—Tapeworms.

Cestodes are flat worms without mouth or intestine, consisting of a scolex and proglottides, which develop by generation in alternate hosts and by gemmation with elongated tape-like colonies. They combine, except in a limited number of species, the male and the female sexual organs in the same segment. The scolex or head serves as the means of attachment for the entire worm to the wall of the intestine and is for that purpose provided with suctorial organs and clinging organs or hooklets. These organs of attachment are differently arranged in different species. The narrow thread-like part immediately posterior to the scolex is known as the neck. The proglottides or segments are joined to the scolex longitudinally in such a manner that the youngest proglottis is nearest the neck and the oldest most distant from it.

The number of segments varies in different species from a few to several hundred. They are quadrangular and, as a rule, the younger ones have their long diameter transverse to the long axis of the worm, those in the middle are squarish, and the most distant have their long diameter in the long axis of the worm. The lateral borders usually converge toward the anterior extremity in such a manner that the anterior border of the segment is shorter than the posterior border of the next younger segment to which it is attached. About the middle of one margin is the projection of the genital pore alternating irregularly. The uterus has a median trunk with lateral branches, which may be seen when the segment is lightly pressed between glass plates.

The segments, single or in tape-like sections of several, become detached from the posterior end and after lingering in the intestine for a time are evacuated with the fæces, or work their way out of the anus and are sometimes found in the clothing of the host. In violent vomiting single or several united segments may be ejected and segments or an entire worm may find the way through abnormal communications into contiguous organs, as the bladder or the peritoneal cavity. The length of tapeworms depends upon the size and number of the segments. The largest species may attain a length of 8 to 10 metres.

The number of genera is about eighty. Certain species in the adult, sexually ripe stage infest the small intestine of man—the definitive host; the corresponding larval forms live normally in the intramuscular connective tissue and viscera of certain animals which constitute the intermediate host. Exceptionally man, by swallowing the embryos—oncospheres—becomes the host of the larval forms—Cysticercus cellulosæ; Echinococcus. The most common tapeworms of man are:

(a) **Tænia Solium** (*Armed Tapeworm; Pork Tapeworm*).—This cestode was so called because it was supposed to exist as a solitary parasite in the intestine. It is now known that two or more tapeworms may be present at the same time.

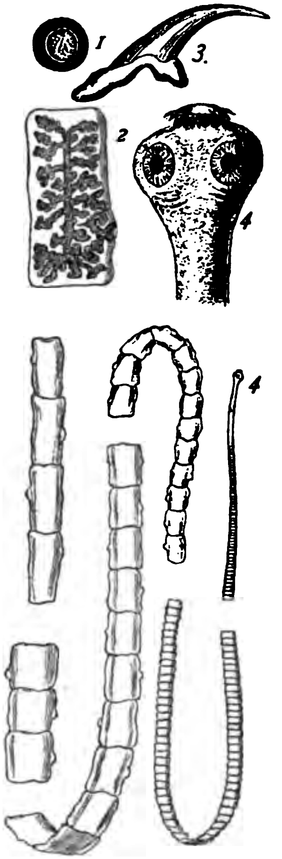


FIG. 294.—Tænia solium. 1, ovum; 2, segment, showing uterus; 3, hook; 4, head.

Average length 3 metres; head globular, 0.8–1.0 mm. in diameter and armed with a double row of hooks; suckers hemispherical; neck slender and 5–10 mm. in length; proglottides 800–1000 in number when mature and ready for detachment, 10–12 mm. in length by 5–6 mm. in breadth; genital pores alternate; uterus consists of a median trunk with 7–10 lateral branches on each side, some of which again branch; eggs oval with very delicate shell; embryonal shell thick, globular, of a pale yellowish color with radial stripes, 0.031–0.036 mm. in diameter; the embryo armed with six hooklets. This parasite when fully developed is found exclusively in the small intestine of man. The embryos are voided with the fæces but undergo no further development unless taken into the stomach of a suitable animal, especially the hog or man himself. The embryo shells are then digested, the armed embryos are set free, and, finding their way to various parts of the body, develop into the larvæ or cysticerci.

The geographical distribution of T. solium corresponds in general with that of the domestic hog and the customary use of raw or insufficiently cooked pork. It is relatively common in North Germany, rare in the United States, and for obvious reasons in Mussulman countries and among the Jews.

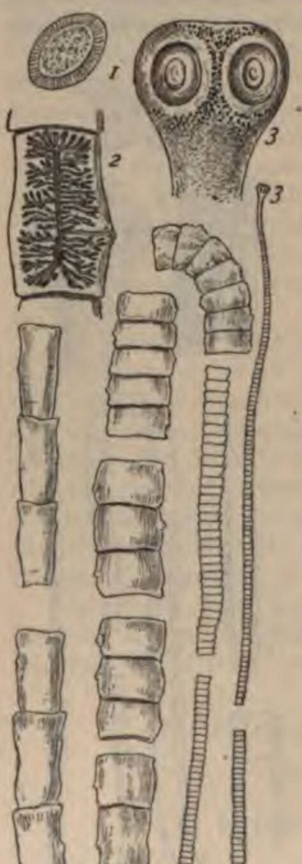


FIG. 295.—Tænia saginata. 1, ovum; 2, fully developed segment showing uterus; 3, head.

(b) **Tænia Saginata** (*T. mediocanellata; Unarmed Tapeworm; Beef Tapeworm*).—Length variable, up to 10 metres, even 36 metres; head cubical, 1.5–2 mm. in diameter and without hooklets; suckers spherical and pigmented; neck long and about half the diameter of the scolex; proglottides average in number 1000; when mature, pumpkin-seed shaped, 16–20 mm. in length by 4–7 mm. in breadth; genital pores irregularly alternate; uterus median with twenty to thirty-five lateral branches on each side, also ramifying. Eggs globular, shell provided with one or two filaments. Embryonal shell oval, thick, transparent, and striated, measuring 0.03–0.04 mm. in length by 0.02–0.03 mm. in breadth. T. saginata in its adult stage is found only in the intestinal canal of man. The ripe segments and ova are voided in the fæces and swallowed by cattle, in the muscles and organs of which they develop into Cysticercus bovis. T. saginata is the most common tapeworm of man and is widely distributed. It is the ordinary tapeworm of North America, is very common in Europe and Africa, and has been known in the East for centuries. The Jews, who are forbidden to eat pork, especially suffer from the beef tapeworm. The eating of uncooked beef is liable to be followed by this form of parasite.

Much less common are:

(c) **Tænia Cucumerina** (*T. elliptica; Dipylidium canium*).—A small tapeworm found in great numbers in the intestines of the dog and cat. The larvæ develop in the lice and fleas of those animals. This parasite is sometimes observed in little children.

(d) **Tænia Nana** (*Hymenolepis nana*).—A small parasite having the genital pores all upon one side. This parasite is common in Italy. Stiles states that H. nana is the most common tapeworm in children in the United States. It is supposed to have cysticercus stages in the intestinal villi, no secondary host being necessary. There are marked nervous symptoms and signs of profound infection.

(e) **Tænia Flavopunctata** (*Hymenolepis diminuta*).—This small cestode is extremely rare. The proglottides show posteriorly a yellow area corresponding to the male sexual organs; hence the name.

(f) **Tænia Lata** (*Dibothriocephalus latus; Bothriocephalus latus*).—The designation of this cestode indicates the lateral pitting of the head and its relatively large size. Length up to 9 or more metres; head almond-shaped, 2–3 mm. in length and flattened, a deep suctorial groove with sharp edges being situated at each side; no hooklets; neck very thin; proglottides numbering 3000–4500, greater in breadth than in length; eggs large with brownish shells, deposited in the intestines, voided with the fæces, and hatched in water. The intermediary hosts are the pike and other fish. This parasite is widely encountered in the Baltic provinces and Switzerland and is the common tapeworm in Japan.

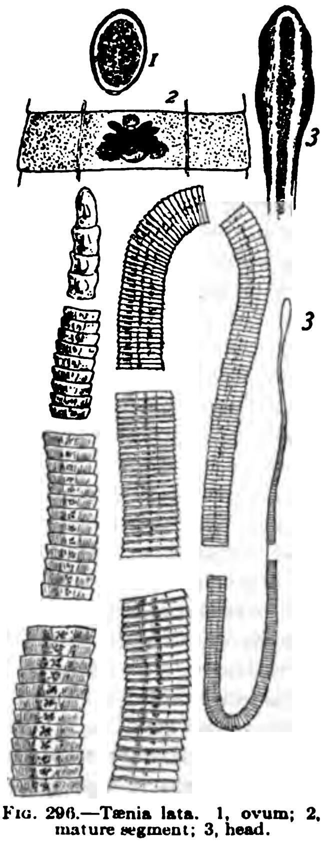


FIG. 296.—Tænia lata. 1, ovum; 2, mature segment; 3, head.

Etiology.—The eating of the raw or insufficiently cooked flesh of animals and fish, and uncleanly habits, constitute predisposing influences of great moment. Tapeworm, owing to systematic food inspection, is rapidly becoming less prevalent in well organized communities. The parasites may be encountered at any period of life. They are common in children and have been met with in infants at the breast.

Symptoms.—Tapeworms may give rise to no inconvenience. They are rarely dangerous. Their presence may, however, occasion symptoms, partly gastro-intestinal, partly nervous. Among the former are a ravenous appetite, abdominal uneasiness and distress, nausea, and diarrhœa; among the latter, nervous depression and hypochondria. Convulsions, chorea, vertiginous attacks, often attributed to the parasite, are rarely directly caused by it. The cessation of any group of symptoms upon its removal is important. Autosuggestion is to be considered. On the other hand troublesome symptoms are sometimes undoubtedly due to intestinal irritation or to toxic substances, evolved by the worm, acting upon the nervous system and the blood—hæmolysis. The Bothriocephalus may be the cause of a severe anæmia having the characters of pernicious anæmia, which sometimes proves fatal, but which in some instances has terminated in prompt recovery after the removal of the worm.

Diagnosis.—The presence of the segments in the stools or in the garments of the patient is positive. The ova and oncospheres may be found in great numbers upon microscopic examination of the stools. Tapeworm treatment should never be inaugurated until the direct diagnosis has been made by the discovery of the segments or ova in the stools. Various substances found in the fæces, such as shreds of mucus, bits of aponeurosis or tendon, or seeds are brought to the physician by the patient who suspects that he has tapeworm. The differential diagnosis between the ova may be uncertain, but the difference between T. solium and T. saginata is at once apparent upon examining the ripe segments between glass slides. The rare species must be submitted to an expert.

The **prognosis** is favorable. There are several efficient tæniacides.

ii. Visceral Cestodes.

(a) **Cysticercus Cellulosæ.**—Infection of human beings by cysticerci takes place by the introduction of the ripe ova (oncospheres) of T. solium into the stomach. This occurs by drinking contaminated water, eating salads or other raw vegetables washed with such water, or in uncleanly persons by their accidental introduction from the fingers. Autoinfection doubtless frequently takes place in this manner and sometimes from the retropulsion of the mature segments into the stomach in the act of vomiting. The development of Cysticercus cellulosæ takes from two to three or four months. Their length of life in animals is unknown. After a time they die and become calcified or undergo caseation. They have been found in almost every organ in the human body. They appear in subcutaneous tissues and in the muscles as ovoid whitish bodies, on the surface of which a spot may be found which is the invaginated head. They are most common in the brain, in which they sometimes attain considerable size. They infest next in the order of frequency the eye, muscles, heart, the subcutaneous tissue, the lungs, and liver. They were demonstrated by Von Graefe in the vitreous humor and many cases have since been recorded. The number of cysticerci in a single individual varies from a few to several thousand.

Symptoms.—In the hog the cysticerci are often present in enormous numbers without impairing the nutrition or giving rise to noticeable symptoms. In America they are extremely rare in man. When present in small numbers in the subcutaneous tissues or the muscles they cause little or no trouble. When present in large numbers or in regions where their growth is unrestrained by pressure they may cause very marked disturbances. Their general distribution causes muscular pain, stiffness, tingling, and numbness; in the silent region of the brain they may cause no symptoms, but elsewhere they have the same effect as other forms of tumor.

Diagnosis.—DIRECT.—In the eye a positive diagnosis can be made by ophthalmoscopic examination. Subcutaneous nodules may be excised and examined. The sublingual tissues should be examined in a suspected case. The cysticercus of the ox has been found in man only in a few instances.

(b) **Echinococcus Disease.**—The echinococci are the larvæ of T. echinococcus, a minute cestode measuring 2.5–6 mm. in length and 0.06 mm. in breadth, having a scolex armed with a double row of twenty-eight to fifty hooklets on the rostellum and composed of three or four segments, of which the posterior only is mature. The mature segment contains about 5000 ova. This parasite lives in the small intestine of the domestic dog.

The larval or cysticercus stage is passed in various organs of numerous species of mammals, especially the sheep, ox, and hog. Man occasionally acquires echinococcus by ingesting the oncospheres in caressing or otherwise coming into too close contact with infected dogs, or using the same dishes.

STRUCTURE AND DEVELOPMENT. — Echinococcus or bladder-worm consists of a cyst or vesicle filled with a watery fluid, which may attain in man the size of a child's head but in cattle does not often exceed the dimensions of an orange or apple. The thin wall of the cyst consists of two distinct layers, an external, laminated, cuticular membrane or capsule and an internal, germinal, or parenchymatous layer, the endocyst. After a time the cyst acquires an outer or accidental fibrous investment. The development in cattle is often arrested at this point and the cysts are then known as acephalocysts or sterile echinococcus cysts. In other cases in domesticated animals brood capsules are formed within the space, upon the outer surface of which the granular or parenchymatous layer is found. From this surface arise little buds or projections which develop into scolices supplied with four sucking discs and a circle of hooklets. When transferred to the intestine of the dog each scolex may constitute the head of a mature tapeworm. This form is termed Echinococcus veterinorum or fertile echinococcus cyst. In man the mother cyst forms daughter cysts which resemble it in structure and organization and originate from detached portions of the parenchymatous layer. These daughter cysts may develop outwardly and lie between the outer wall of the mother cyst and the adventitious fibrous capsule, or inwardly and, becoming detached from the wall of the mother cyst, float free within the latter. Their number is variable. The daughter cyst may remain sterile or in time may produce brood capsules or granddaughter cysts. Finally the mother cyst may undergo destruction and the daughter cysts, surrounded by thick capsules of connective tissue, may form an irregular tumor mass—multilocular echinococcus. The fluid is of a faint yellowish color, neutral or faintly acid in reaction, non-albuminous, and of a specific gravity of 1.005–1.015.

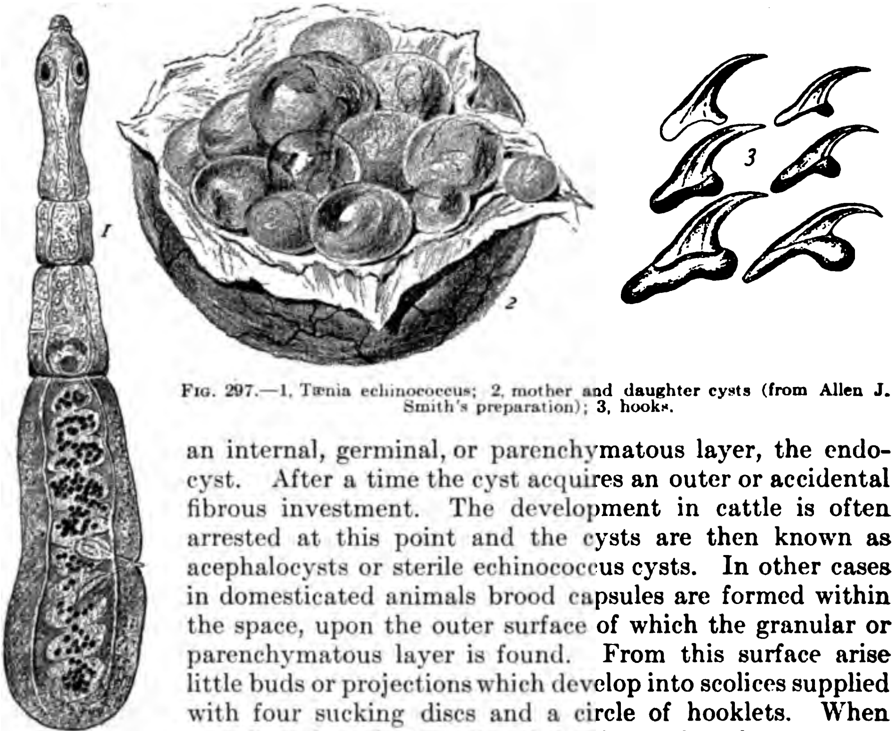


FIG. 297.—1, Tænia echinococcus; 2, mother and daughter cysts (from Allen J. Smith's preparation); 3, hooks.

It contains sugar, inosite, leucin, tyrosin, and succinate of lime and soda. Scolices and hooklets may be found in the fluid of the cysts.

The changes which the cysts undergo are as follows: (a) Death, gradual resorption of the fluid contents, and the conversion of the cyst into a granular, partially calcified mass. Such masses are not uncommon in the liver. (b) Rupture, which may take place into a serous sac, a hollow viscus or a bronchus, the intestine or the bladder, into the bile passages or inferior vena cava, or externally. These accidents are all unfavorable, though recovery may follow the external rupture of the cyst. (c) Suppuration, which may occur with or without rupture and is most frequent in hydatid cysts of the liver.

The geographical distribution of echinococcus disease is wide. In Iceland and Australia it is most common. In European countries it is not rare. It is extremely infrequent in the British Isles and North America.

Symptoms.—The condition is encountered at all ages, but is infrequent in children and old persons. The period of greatest liability comprises the third and fourth decades. Women are more frequently affected than men. The organs most commonly involved are the liver, other abdominal and pelvic organs, brain, and circulatory system. In a majority of the cases one organ only is affected. The primary infection may, however, implicate several organs; later, infection may take place, or in consequence of accidental or surgical traumatism daughter cysts, brood capsules, or scolices may find their way into a serous sac, especially the peritoneum, and colonize, forming new tumors. Hydatids of the liver, when small and deep-seated, cause neither symptoms nor physical signs. When large and superficial they have the attributes of solid or cystic tumors in general. Upon the anterior surface they appear as circumscribed round or oval tumors of firm consistence or obscurely fluctuating; a cyst of the left lobe may displace the heart upward and give rise to extensive dulness in the left hypochondrium; a cyst of the right lobe yields dulness extending upward into the chest. *Hydatid Fremitus.*—When the cyst is superficially situated, it yields in some instances, upon direct finger percussion with the right hand and palpation with the fingers of the left, a peculiar prolonged vibratile tremor. Very large cysts are attended with distressing sensations of weight and dragging, sometimes of actual pain. When suppuration occurs septic symptoms arise. Rupture into the bile passages causes a suppurative cholangitis with deep jaundice; into the vena cava sudden death from the action of the daughter cysts as plugs in arresting the circulation at the tricuspid orifice or in the pulmonary artery. A toxic substance in the fluid contents, probably a leucomaine, causes, when introduced into the peritoneal cavity, a general peritonitis. To this substance has been attributed the urticaria which frequently accompanies the rupture of hydatid cysts or operation upon them.

Diagnosis of Hydatids of the Liver.—DIRECT.—Moderate-sized cysts produce no symptoms by which they can be recognized. A large circular or oval tumor or, in the case of multiple cysts, a similar large, irregular mass, unaccompanied by pain, firm and elastic, or fluctuating, and especially when there is the hydatid tremor connected with the liver and not attended by derangement of the health, justifies a provisional diagnosis of hydatid cyst. If, upon exploratory puncture, a fluid having the above characters and containing hooklets is withdrawn the diagnosis is positive. If there is a history of acutely developing pulmonary symptoms—rupture into the lung—and hooklets or cysts in the matter coughed up, the diagnosis is certain. The presence of hooklets in a doubtful fluid is diagnostic.

DIFFERENTIAL.—*Abscess.*—When suppuration occurs the condition is actually hepatic abscess. The history of a tumor in the hepatic region, unaccompanied by failure of health, is suggestive of hydatid cyst; of dysentery or traumatism in the absence of previous enlargement, in favor of primary abscess. *Syphilis.*—The tumor or tumors are firm and non-fluctuating. The anamnesis is important. *Cancer.*—As a rule the course of the disease is very different from that of carcinoma hepatis; but there are cases in which the multiple tumor formation simulates cancer very closely. Large, single tumors and fluctuation, especially the hydatid fremitus, are in favor of hydatids. *Dilatation of the Gall-bladder.*—Empyema of the gall-bladder, in the absence of adhesions, constitutes a pear-shaped tumor which is often movable in a lateral direction more freely at its lower than its upper extremity. *Hydronephrosis.*—The discrimination is sometimes beset with difficulties. In this condition the tumor may repeatedly disappear with great diuresis. If a hydatid cyst ruptures into the bladder, hooklets may be found in the urine. *Pleural Effusion upon the Right Side.*—The diagnostic difficulties here also are great. Exploratory puncture is necessary. The character of the fluid is distinctive.

Diagnosis of Hydatids of the Lungs and Pleura.—*Lungs.*—The direct diagnosis cannot be made in the case of small cysts which produce only trifling symptoms. Larger cysts compress the pulmonary tissue and lead to inflammation and necrosis with ulceration into bronchi and the discharge of membrane, daughter cysts, and hooklets. Hemorrhage is common. *Pleuræ.*—Hemorrhage into the pleura with empyema and pleuropulmonary fistula occurs. The condition simulates ordinary empyema but the anatomical findings in the sputa—membranes, cysts, or hooklets—are diagnostic. The larvæ may first develop in the pleura and reach a large volume, simulating effusion. The upper line of dulness is irregular. Inflammation may ensue with perforation of the chest wall. The condition is a serious one, liable to be followed by sepsis.

Diagnosis of Echinococcus of the Kidneys.—The kidney may be dilated and simulate hydronephrosis. The diagnosis can be made only by an exploratory puncture and examination of the fluid.

Diagnosis of Echinococcus of the Brain.—The symptoms are not characteristic, being those of tumor—persistent headache with vertigo and vomiting, convulsions of Jacksonian type, and optic neuritis with atrophy. The differential diagnosis cannot be made. Echinococcus cysts in the liver or elsewhere would justify a probable diagnosis. Cystic disease of the choroid plexus is to be considered.

A form of multilocular echinococcus, encountered in men and oxen in Russia, Bavaria, Switzerland, and the Austrian Alps, deserves special consideration. A few imported cases have occurred in North America. The tumor is confined to the liver and consists of dense strands of connective tissue in which are embedded numerous cysts so that the cut section has a honeycomb appearance. The cysts are filled with a transparent or opaque gelatinous fluid and present the appearance of alveolar colloid cancer. There is a tendency to disintegrating ulceration. The spleen is usually enlarged. Jaundice is common. There is a tendency to hemorrhage and the prognosis is unfavorable.

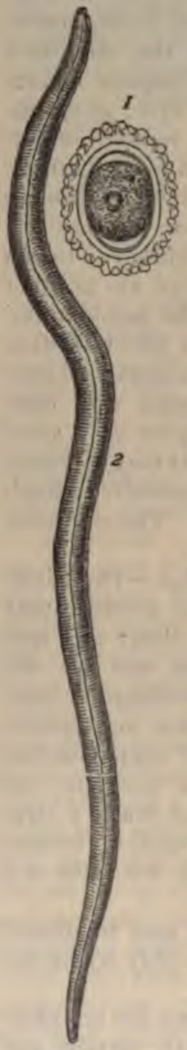


FIG. 298.—Ascaris lumbricoides. 1, ovum (after Braun); 2, female worm.

D. DISEASES DUE TO NEMATODES.

Nematodes are elongated round worms of a filiform or fusiform shape, provided with a mouth and intestinal apparatus. The sexes are in most species separate and the male can be distinguished from the female by its smaller and more slender form and spiral or incurvated posterior extremity. Fertilization takes place within the uterus, and the ova, according to the species, are deposited before or during segmentation or with the embryo fully developed. A few species are viviparous. The mode of infection of the host differs according to the species of worm.

i. Ascariasis.

Ascaris Lumbricoides.—The body is spindle-shaped and of a reddish or grayish-yellow color, with four longitudinal bands and transverse markings. The male measures 12–25 cm. in length and about 3 mm. in transverse diameter at its thickest part; the female from 20–40 cm. by 5 mm. The ova are elliptical with a thick brownish-red covering. They measure 0.05–0.07 mm. in length by 0.04–0.05 mm. in breadth. They are deposited before segmentation and are sometimes present in the stools in great numbers. Generation takes place without intermediate host. This nematode worm is the most common parasite of man and is distributed over the entire world. It is most frequent in young children, but occurs at all periods of life. It is extremely common in the negro races. As a rule only a few worms are present, but cases have been reported in which hundreds of them have been harbored by one individual.

The upper portion of the small intestine is the normal habitat of the round worm. They migrate, however, into the stomach and are frequently evacuated by vomiting. This is especially common in febrile diseases. They sometimes pass through the œsophagus into the pharynx and creep out through mouth or nostrils. They have been known to penetrate the Eustachian tube and appear at the external auditory meatus. They sometimes occupy the biliary and pancreatic ducts, or inflammatory adhesions between the intestine and adjacent parts—*worm abscess*. Passing from the pharynx into the larynx they have caused fatal asphyxia. In other cases they have escaped into the trachea and, penetrating into the ronchi, have been the occasion of gangrene of the lung. They sometimes nd their way into the bladder and are passed with the urine. These anderings are the cause of most serious and often unaccountable symptoms, ut in neurotic persons even the presence ' a small number of worms within the testine may give rise to nervous pheomena—chiefly hysterical—which cease oon their expulsion. In other cases regular fever with gastro-intestinal sympms occurs. These symptoms have been garded as reflex, but are probably due a toxin. The presence of Ascaris mbricoides in the intestine may be monstrated by finding the ova upon icroscopical examination of the fæces.

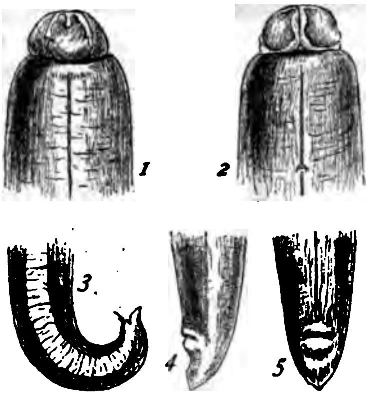


FIG. 299.—1, dorsal aspect of head of Ascaris lumbricoides; 2, ventral aspect of head; 3, tail of male; 4, lateral aspect of tail of female; 5, ventral surface of tail of female—After Claus.

Oxyuris Vermicularis (*Thread Worm; at Worm*).—One of the most common d widely distributed of human parates. The color is whitish and the males may be seen in lively movement the recently voided fæces of infected rsons. The male measures 3–5 mm. in length and the female is 10 mm. length and 0.6 in breadth. The ova are deposited with the embryo fully veloped and are very rarely found in the fæces. This parasite lives the rectum and colon. It is usually present in enormous numbers. Its anderings are chiefly nocturnal and give rise to troublesome itching. The calities affected are the sulcus between the nates, the perineum, and the lva. As the result of scratching, the larvæ may be carried to the nose d mouth and the patient reinfects himself, or he may infect others by e hand directly or indirectly. The primary infection takes place by eans of water or fruits or vegetables eaten raw, or perhaps through e intervention of flies. Direct development takes place without an termediate host.

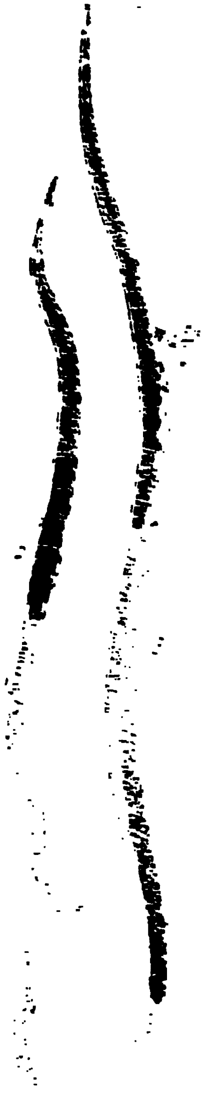
Oxyuris in rare instances penetrates the wall of the gut and causes perictal abscess. Most common in children, it may be encountered at any age.

The symptoms in addition to local irritation and itching are restlessss, disturbed sleep, loss of appetite, and anæmia.

ii. Trichiniasis.

The disease is caused by the embryos of Trichinella spiralis, which pass om the intestines and are distributed widely throughout the body, but d the conditions necessary to their further development only in the ores of the transversely striated muscles, in which they develop into capsulated larvæ.

Trichinella Spiralis.—The male measures 1.4–1.6 mm. in length d 0.04 mm. in breadth; the female 3–4 mm. in length and 0.06 mm. in ameter. This parasite in the adult stage inhabits the small intestine of an and various mammals, especially the hog. The larvæ are 0.8–1 mm.



[illegible] the striated muscles, in which they [illegible] capsules which mostly have [illegible] axis parallel with the long axis of the [illegible] fibres.

It has been experimentally demonstrated that the ingested larvæ shortly after their introduction into the stomach are freed from their capsules by the action of the gastric juice and pass into the upper part of the small intestine, where they quickly attain their adult form. Copulation takes place in the course of [illegible] days, after which the males die and the females, which are viviparous, penetrate the intestinal mucosa and reach the lymph spaces, in which they deposit their young. Carried by the lymph stream and ultimately by the blood current the embryos invade the striated muscles, in which encapsulation takes place. On the ninth or tenth day after ingestion of the affected meat the first embryos have reached their destination. Two or three embryos may occupy the same capsule. The infested muscular fibres undergo degeneration and lose their striation. The intramuscular connective tissue undergoes an inflammatory hyperplasia and forms the cystic capsule. In the course of several months the capsular walls, at first transparent, undergo calcification, which, beginning at the poles, gradually progresses until in the course of [illegible] the enclosed larva also become calcified. In [illegible] calcification is usually long delayed, so that the [illegible] ordinary examination.

The [illegible] are not evenly distributed throughout the [illegible] system. Favorite locations are the [illegible], the muscles of the diaphragm, [illegible] and tongue. In their encysted state [illegible] preserve their capacity for development [illegible] years. The beginning of calcification [illegible] this period. Rats appear to be the [illegible] of T. spiralis. They infect themselves [illegible] flesh of their own kind and the hog [illegible] and infect other animals, as pigs, [illegible] which they are sometimes caught [illegible] by feeding upon the offal of [illegible]. Man is infected by eating the raw [illegible] flesh of infected hogs. The geographical [illegible] of T. spiralis is much more [illegible] occurrence of trichiniasis in man. [illegible] raw or only partially cooked [illegible] cause of trichiniasis. Where [illegible] of trichiniasis do not occur even [illegible] hogs. North Germany cially suffers and affords many examples of grave epidemics. In South Germany, France, England, and the United States the disease is infrequent. Post-mortem investigations indicate that mild sporadic cases are more common in this country than was formerly supposed and that they are frequently overlooked.

The anatomical lesions consist of minute local wounds of the intestine caused by the boring female trichinellæ and important chiefly in proportion to their number; of the lesions in the muscles, the primitive bundles undergoing granular degeneration with local myositis; and of important changes in the blood, which shows a marked leucocytosis—25,000–30,000. The eosinophiles are enormously increased, comprising 20 per cent. or more of all the leucocytes. Fatty degeneration of the liver and enlargement of the superficial lymph-glands have been described. New broods of embryos are produced from time to time and adult trichinæ are found post mortem in the intestine in cases fatal at the end of four or five weeks.

Symptoms.—Sporadic cases occur, but the disease is more commonly endemic and local epidemics are common. The last can almost always be directly traced to the pork supply and not rarely have followed a "Fest" or entertainment in which uncooked ham, sausages, or similar food have been largely partaken of. Trichinous flesh may be eaten without causing trichiniasis. This occurs when the cysts have been thoroughly acted upon by heat in cooking, when a limited number of embryos have been ingested, and when active purgation has promptly occurred.

(a) STAGE OF GASTRO-INTESTINAL IRRITATION.—A few days after eating trichinous meat loss of appetite, abdominal pain, vomiting, and diarrhœa occur. These symptoms are of varying intensity, sometimes being absent altogether, sometimes almost choleraic. These symptoms are often attended with great general debility. (b) GENERAL INFECTION.—The invasion of the muscles gives rise in man to a more or less intense myositis, manifested by pain upon movement and pressure, swelling and tension of the muscles, and œdema of the overlying skin. The muscles of mastication and deglutition are especially involved and the predilection of the embryos for the muscles of the diaphragm and the intercostal muscles is the occasion of serious, sometimes fatal dyspnœa. The onset of these symptoms, which follows infection in about ten days or two weeks, is accompanied by fever of remittent or intermittent type—102°–104° F. (39°–40° C.)—and local œdemas, especially under the eyes. Excessive sweating, itching, and urticaria occur. Anæmia, rapid wasting, and loss of strength are common. In the severer cases delirium, tremor, and dry tongue occur. Albuminuria is common and polyuria may occur.

Diagnosis.—DIRECT.—When a number of persons fall ill at the same time shortly after a festival or who are customers of the same pork butcher, suspicion should be aroused. The finding of the parasites in the pork, in the stools of the patients, in shreds of muscle removed for the purpose under local anæsthesia; muscular tenderness upon movement or pressure; œdema under the eyes; the blood count, showing high leucocytosis with marked eosinophilia, constitute positive diagnostic criteria.

Trichinellæ in the stools, when examined with a low power, appear as short, silvery, glistening threads, which are sometimes still in movement.

Differential.—*Enteric Fever.*—Any resemblance that may occur is superficial. On the one hand we have a definite symptom-group characterized by the gradual rise of temperature, relatively slow pulse as compared with the rise of temperature, palpable spleen, and rose spots, together with a positive agglutination test; on the other the equally characteristic symptom-complex described in the foregoing paragraph. *Rheumatic Fever.*—Pain on movement and tenderness are suggestive. But the joints and not the muscles are involved and the œdema is periarticular. *Cholera.*—The urgency of the intestinal symptoms and the great number of persons simultaneously affected in some of the epidemics has aroused the suspicion of cholera. Rice-water discharges, collapse, and the rapid course of the latter disease are diagnostic points of importance.

Prognosis.—The duration and severity of the attack depends upon the number of the invading embryos. The symptoms are aggravated by the access of fresh groups. In mild cases the symptoms are slight and disappear in the course of two or three weeks. In the more severe cases the active symptoms continue for several weeks and convalescence is tardy. The death-rate varies from 1 or 2 to 20 or 30 per cent. in different outbreaks. Death most commonly occurs in the fourth, fifth, or sixth week.

iii. Uncinariasis.

Ankylostomiasis; Hook-worm Disease.—The parasite of this disease belongs to the Strongylidæ. There are two species parasitic in man, distinguished by specific anatomical differences, especially in the mouth, and by differences in size—the (a) *old-world Ankylostoma duodenale* and (b) *Necator americanus.* The general characters are similar. The body is cylindrical, attenuated anteriorly, and of a reddish color. The males measure 8–10 mm. in length and 0.4–0.5 mm. in breadth; the females 10–18 mm. in length. The eggs are elliptical, thin-shelled, and measure 0.05–0.06 mm. by 0.03–0.04 mm. and are laid in a state of segmentation. In the European species the mouth is supplied with four sharp, hook-like ventral teeth, projecting backward, and with two teeth projecting forward on the dorsal surface, while in the base of the oral cavity there is one tooth directed forward. The cauda bursa of the male has one small dorsal and two large lateral alar processes. The development is direct without an intermediate host.

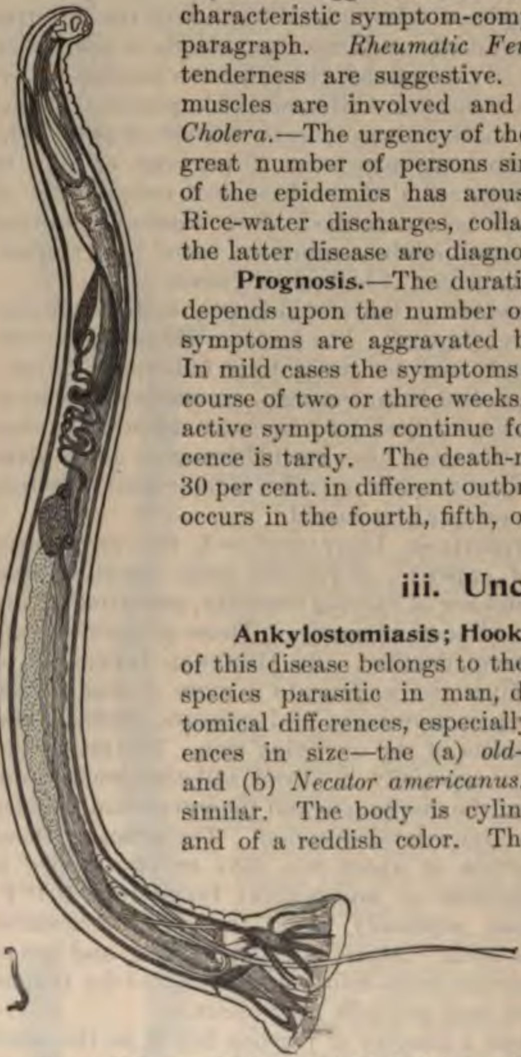
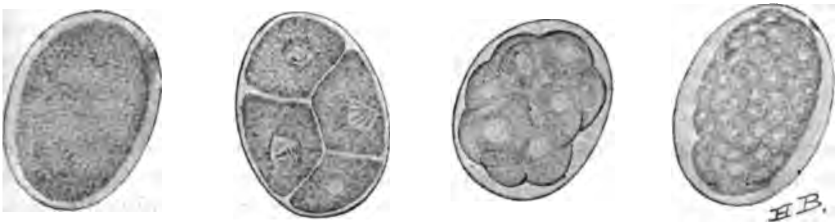


Fig. 301.—Ankylostoma duodenale, natural size below, and much magnified male.—After Schulthess.

The adult worm infests the duodenum, less frequently the jejunum, nd sucks blood with its head buried in the mucosa, changing its position :om time to time so that minute hemorrhages continue. The number of rorms varies from a few to a thousand or more. The duration of life in he bowel is unknown. The disease is essentially chronic. It may be kept p by the prolonged life of the parasite, or by reinfection.

This parasite is the cause of "Egyptian chlorosis," the tunnel disease f St. Gotthard, miners' and brickmakers' disease, and tropical anæmia. t is widely distributed in warm countries, but occurs in all parts of the old orld. Since the Spanish-American War uncinariasis has attracted much ttention and Necator americanus (Stiles) has been found to be the cause f the so-called southern anæmia. It is endemic in Virginia, North and outh Carolina, Georgia, Florida, Alabama, and Texas. It is extremely revalent in Porto Rico; less so in Cuba and Brazil.

The larvæ live in water and moist soil. There are two hypotheses s to the mode of their introduction: first, that they are ingested by the iouth in drinking water, upon uncooked vegetables, from the soiled hands f men who work and children who play in moist earth, or by clay eaters; nd second, that they penetrate the skin by way of the hair-follicles, and re transported by the venous blood to the right side of the heart and the ıngs, whence they pass by way of the bronchi and trachea to the pharynx, nd are then swallowed. This extraordinary observation of Loos has been onfirmed by others, and Smith of Atlanta produced uncinariasis in man by ıe application of mud containing the larvæ to the arm. The long vexed uestion of the relation of "ground itch" to uncinariasis is thus settled.



ıg. 302.—Eggs of Uncinaria duodenalis. *a*, unsegmented; *b*, with four segments and showing nuclear spindles; *c* and *d*, later stages of segmentation. × 400.—Emerson.

Symptoms.—The clinical phenomena are due to the constant, proınged drain of blood from the intestinal mucosa by the parasites themılves and from the wounds which they have made, bacterial infection at ıe site of the lesions, chronic local inflammation and thickening of the owel, and the deleterious action of toxins produced.

The ova are frequently found in the stools, especially in children, in ıe absence of symptoms. From this fact it has been inferred that a rge number of the parasites are necessary to cause the disease. The næmia is the most striking condition. Some associated pigmentation ves the skin a peculiar dirty appearance. The facies has been regarded s characteristic, its peculiarities consisting in a pallid, waxy color with int pigmentation, and a lustreless, blank expression of the eyes. When ıe disease is marked in children, nutrition and growth are seriously interfered with. Enlargement of the liver and spleen, with œdema, occurs in advanced cases, and the symptoms of anæmia—breathlesnesss and palpitation upon exertion, pallor, puffiness, and headache—are common. The blood shows corpuscular and hæmoglobin reduction, infrequent leucocytosis, and a moderate eosinophilia. In old cases with marked anæmia, which has lasted a long time, the eosinophile count is low.

Diagnosis.—DIRECT.—The presence in fresh fæces of ova showing segmentation, or in older fæces of ova containing the curled embryos within or penetrating the thin shell, is characteristic. The blotting-paper test may be employed. A little of the fæces placed on white blotting paper after an hour will show a reddish color like blood.* Eosinophilia is of diagnostic value.

DIFFERENTIAL.—*Pernicious Anæmia.*—Many of the cases suggest this condition. The presence of the ova in the stools, the locality from which the patient comes, his occupation, the facies, the blood picture, and the therapeutic test with thymol are all to be considered.

Prognosis.—The outlook under thymol treatment, except in advanced cases with marked anæmia, is fairly favorable.

iv. Filariasis.

Filariæ are long, slender nematodes, which live parasitically in the serous cavities and subcutaneous tissues of the mammals which they infest. The males are usually much smaller than the females, having the tail sharply bent or spiral and being supplied with wing-like appendages.

Nineteen genera are described, of which the following are classed under the general term Filaria sanguinis hominis:

1. **Filaria bancrofti.**—The male is colorless and measures 40 mm. in length by 0.1 mm. in breadth. The female is of a brownish color and 70–80 mm. in length, 0.2–0.3 mm. in diameter, and possessed of two uteri which occupy the greater part of the body. The embryos are contained in an elongated, scarcely perceptible membrane in which they move freely. Their length is 0.13–0.3 mm., their breadth 0.007–0.011 mm. By way of the lymph stream they reach the blood and are distributed to all parts of the body. Their appearance in the peripheral circulation is peculiar, showing a remarkable periodicity. During the day there are very few if any to be found; but towards sunset they appear and steadily increase in number until midnight, when they gradually decrease and disappear by the middle of the morning. If the patients sleep during the day and remain awake at night the order of the appearance of embryos is reversed, namely, they are present during the day and not to be found at night. The further development of filariæ is associated with the mosquito, which constitutes the intermediate host.

This parasite is distributed in nearly all tropical countries, and filariasis is common in India, China, Japan, the Australasian Islands, Egypt, The Sudan, Zanzibar, Madagascar, and the Southern United States. Sporadic cases are occasionally encountered in the Middle States and elsewhere in temperate climates in the course of routine examinations of the blood. Some of these cases are imported, but there are instances in which the source of the infection could not be traced.

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* Ashford and Igaravidez, U.S. Senate Document No. 808, 1911, mention this test only to condemn it.

Symptoms.—In animals and during a long period of latency in man, filariæ may exist in the blood without causing any inconvenience. Their presence becomes known only upon examination of the blood. After a time anæmia, splenic enlargement, and irregular fever of moderate intensity may occur. When the lymph-vessels become blocked by the adult worm or the ova, characteristic conditions arise, namely, hæmatochyluria, lymph scrotum, and elephantiasis.

Hæmatochyluria.—The urine is opaque, milky white, or blood-tinged. On standing a reddish coagulum sometimes forms. Microscopically there are minute globules and molecular fat and erythrocytes in varying numbers. The quantity of urine is normal or it may be increased. The condition is intermittent, and after passing chylous urine for a short period the patient commonly passes for weeks or even months urine that is normal. The general health is often fair. In other cases there is more or less uneasiness in the lumbar region, anæmia, and vesical irritation, with difficulty in passing the blood-clots which form in the bladder.

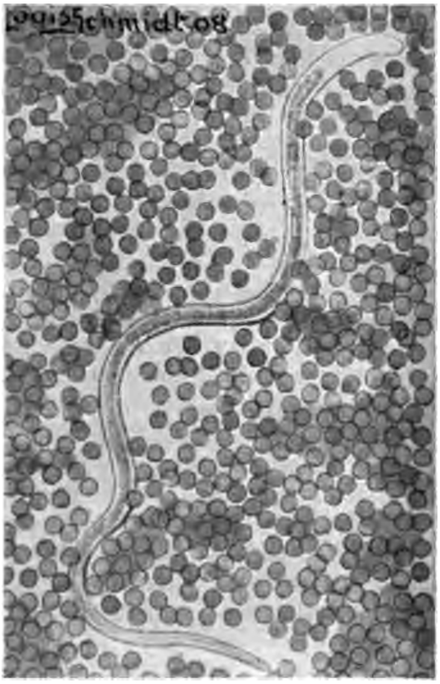


Fig. 303.—Microfilaria nocturna.

Lymph Scrotum.—Blocking of the lymph channels is followed by a dilatation of the lymph plexuses that is sometimes enormous. When the scrotum is involved there is great thickening of the tissues and the distended lymph-vessels are plainly visible. Upon puncture a clear or sometimes a turbid fluid exudes.

A form of elephantiasis follows permanent occlusion of the lymph channels of the lower extremities. The lymphatic glands, especially in the groin, are much enlarged.

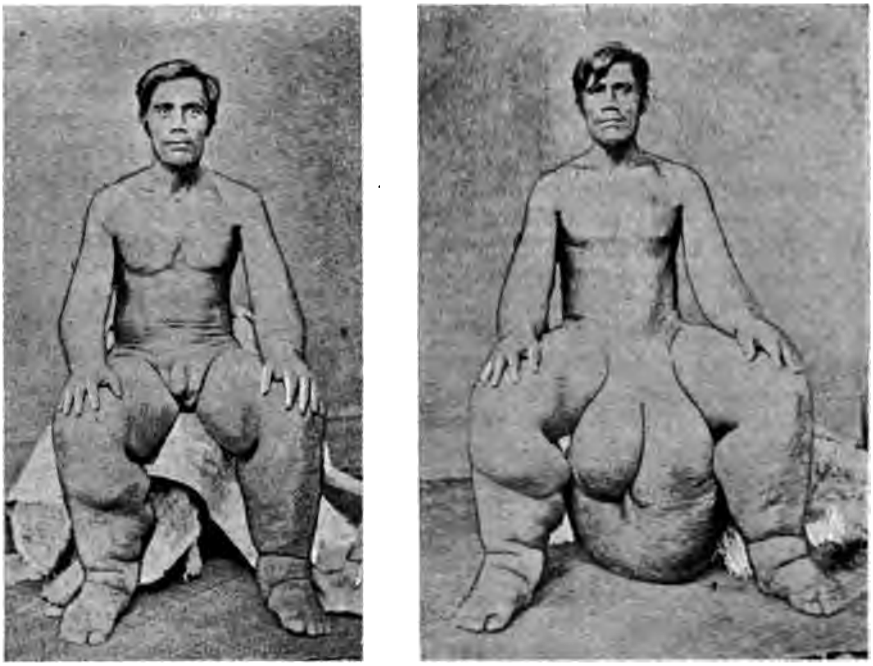
Diagnosis.—The living embryos in the recent blood are readily recognized. Their presence is made known by the commotion which they cause among the red corpuscles, and the worm itself, usually in active movement, may be made out with a low power. Chylous urine may occur under other circumstances not well understood. The non-parasitic form is very rare. Withdrawal of fat from the diet is usually followed by disappearance of the chylous appearance, but a glass or two of milk will render the urine again opaque.

Most cases of elephantiasis in temperate climates are non-parasitic.

Prognosis.—A large proportion of the cases remain latent for an indefinite period. Many of the cases of lymph scrotum and elephantiasis are progressive, and the patient succumbs to exhaustion or intercurrent disease. The removal of an adult worm from the enlarged inguinal lymph-glands has been followed by the disappearance of the embryos from the blood. If two or more were present this result would not occur.

2. **Filaria diurna.** — This parasite closely resembles F. bancrofti. It appears in the blood during the day only, or at night when the patient remains awake. Manson found the larvæ in the blood of several negroes from Congo. The mangrove fly is supposed to be the intermediate host. F. loa is the adult form.

3. **Filaria perstans.**—The larva was discovered by Manson in 1891. Manson found in the blood of Carib Indians sent from British Guiana two forms of larval filariæ differing in type, one closely resembling those of F. perstans, the other slightly larger and likewise without a sheath. Daniels in 1898 found the adult worm, both male and female. The female is 70–80 mm. in length by 0.12 mm. in breadth; the male 45 mm. by 0.06 mm.; the embryos 0.2 mm. in length by 0.004 in breadth and possess no sheath. The adult worms inhabit the connective tissue of the mesentery at its spinal attachment. This parasite infests the tribes who dwell in dense swamps and deep forests. It abounds on the West Coast of Africa, in British Guiana, and in Porto Rico. It may be observed in the blood both during the night and day. It is thought to be the cause of a pustular disease of the skin common among the negroes of the West Coast of Africa. The intermediary host has not been settled.



a *b*

FIG. 304.—*a*. Patient aged twenty-three years, affected with elephantiasis arabum. *b*. Same patient, aged forty-seven years. (*International Clinics*.)

v. Dracontiasis.

Guinea-worm Disease.

Filaria (Dracunculus) medinensis (*Guinea-worm; Medina worm*).—The female measures 50–80 cm. in length and 0.5–2 mm. in diameter and is cylindrical in form with a blunt anterior extremity and a pointed posterior end terminating in a hook.

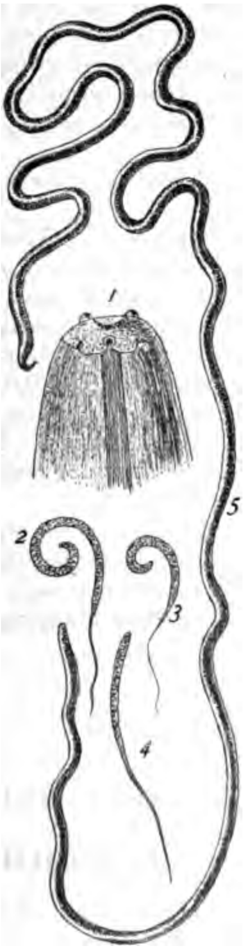


Fig. 305.—Filaria medinensis. 1, head; 2, 3, 4, larvæ; 5, adult worm.—After Claus.

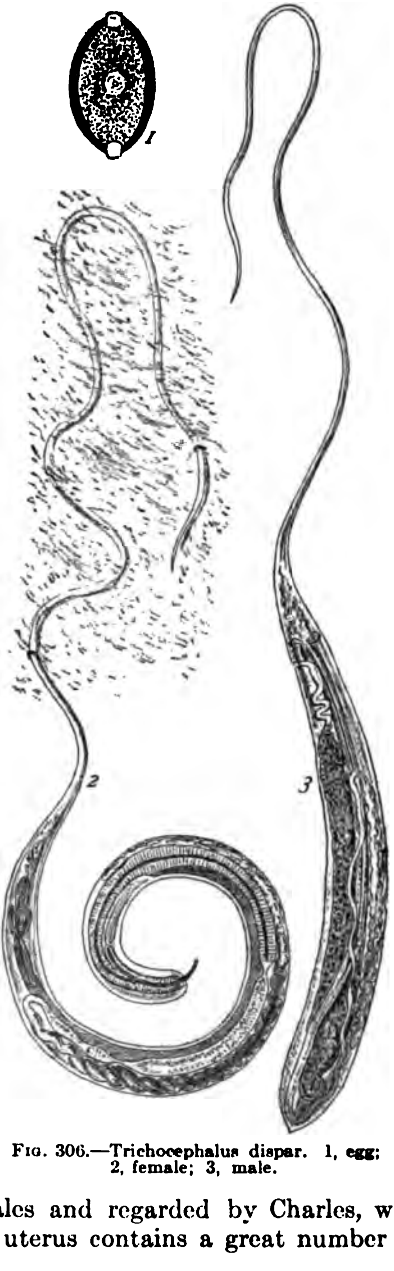


Fig. 306.—Trichocephalus dispar. 1, egg; 2, female; 3, male.

Only the female has been known. Quite recently, however, small worms about 4 cm. in length were found in two instances attached to the females and regarded by Charles, who made the observation, as males. The uterus contains a great number of living embryos, which may reach the open by the rupture of the body of the adult female. They develop in the body of Cyclops. The male and female are probably ingested by the mouth, the former dying and the latter, after impregnation, finding its way to the subcutaneous tissues, in which it slowly develops, remaining quiescent during a period of eight to ten months. It feels like a coil of string under the skin. As the embryos develop the adult worm slowly makes its way downward to the leg or foot, where it forms a small vesicle or abscess, which bursts, leaving an ulcer in the base of which its head appears. The uterus ruptures and the embryos are discharged in a whitish fluid. The worm may now leave the host spontaneously. Guinea-worms are usually solitary, but several have been observed in the same individual. It attacks all races without distinction. It has been known from the earliest historical periods. It is especially prevalent in Africa and the West Indies, but imported cases are occasionally observed in the United States.

When the worm first becomes palpable there is sometimes fever together with an eosinophilia.

Trichocephalus dispar (*T. trichiurus; Whip-worm*).—This parasite may be recognized by the difference between its filiform anterior extremity and its much thicker posterior portion. Its length is 40–50 mm., the male being slightly shorter than the female. The ova are lemon-shaped, and have a thick brownish shell at the ends of which are light yellow plugs or buttons. They measure 0.05 mm. in their long diameter and 0.02 mm. in their transverse diameter. This is a common and widely spread parasite of man infesting the cæcum and other parts of the intestine. It usually gives rise to no symptoms, but exceptionally its presence in great numbers is associated with anæmia and diarrhœa.

A number of less important nematode worms have been observed in man, but the infrequency of their occurrence, and the facts that they do not cause definite internal diseases and that their life history is mostly unknown renders any extended consideration of them at this time inappropriate.

III.

THE DIAGNOSIS OF THE CHRONIC INTOXICATIONS.

I. ALCOHOLIC INTOXICATION; ALCOHOLISM.

The discussion here will be confined to a brief statement of the effects of alcohol on the nervous system. As is well known this poison acts most injuriously upon other tissues of the body also, but these effects are best discussed under other and appropriate headings.

The ravages of alcohol are greater in modern life than among the ancients, for the reason that strong alcoholic drinks are now distilled and marketed at a low price. The first modern writer to attempt to treat this subject adequately was Magnus Huss, who made his observations on the Swedish brandy drinkers.

Pathology.—Changes are found in the coats of the blood-vessels, in the brain membranes, and in the neuroglia, as well as in the kidneys, liver, stomach, and heart. Bevan Lewis and Berkley described with minute care the changes in the central nervous system. According to Lewis these changes are particularly marked in the blood-vessels and neuroglia, and Berkley found microscopic changes in the neurons of the brain. Inflammation of the peripheral nerves, constituting the well-known multiple neuritis, is often seen in chronic alcoholics.

Symptoms.—Alcoholic intoxication is either acute or chronic.

ACUTE INTOXICATION requires only a passing notice. In the first stage there is exhilaration, with slight confusion of ideas; then follows a stage in which memory is impaired, self-consciousness is obscured, and the victim may be violent and even maniacal. In the terminal stage a condition of sleep, or even of stupor and coma, supervenes; the pupils are slightly dilated, the face congested or even cyanosed, the breathing normal in frequency, the pulse regular, and the consciousness usually not so lost but that the patient can be roused slightly at least. After some hours he wakes with a sense of depression, headache, nausea, and a dry mouth and throat. Profound intoxication may simulate cerebral hemorrhage, uræmia, diabetic coma, and opium poisoning.

Delirium tremens is caused by protracted acute poisoning. It is the result of a disturbed nutrition of the brain-cells, and continues even after the alcohol is withheld; in fact, it sometimes does not occur until after the patient is deprived of his drink—the *delirium potu suspenso.* This latter form is seen especially in hospital practice—as after a fractured leg, a surgical operation, or even in acute disease, such as pneumonia. The patient may seem to do well for a day or two, when delirium suddenly develops.

Delirium. tremens is a psychosis in which the mental faculties are in entire confusion, with terrifying hallucinations, such as the sight of snakes, bats, and other repulsive objects, and the disturbing sounds of voices; there is restlessness, tremor, incoherence, muttering, insomnia, aversion to food, rapid pulse, and great physical prostration. The so-called typhoid state may develop, with irregular fever and a dry brown tongue, and the case may end fatally. The prognosis is in the main favorable, except in old, broken-down topers.

Subacute forms of delirium occur, in which the patient is able to be about and to continue his drinking. He is in a dream-like, confused state, irritable, irresponsible, insomnious, unable to attend to his work, eating little, and subject to violent outbursts in which he may even commit murder. Alcoholic *melancholia* is also seen in some hard drinkers, and occasionally leads to suicide. It occurs especially after protracted sprees. The affection known as *mania a potu*, often confused with delirium tremens, is, as its name implies, a state of furious maniacal excitement, in which the inebriate is especially dangerous.

The CHRONIC INTOXICATION from alcohol shows itself in the gradual deterioration, mental, moral, and physical, of the individual. The character and reliability of the man suffer; he is unfit for business or society; subject to moral lapses of various kinds; untruthful, indifferent, cruel, and sometimes dangerous. His memory and his mental faculties generally are impaired. He has a bad color, injected capillaries, a tremor of his hands and tongue, and is a poor eater and sleeper. There is always a chance also that he has a bad liver and bad kidneys. Optic neuritis and atrophy may occur in chronic cases.

Neurasthenia and *hysteria* are among the conditions seen in chronic alcoholics. These complications are seen especially in persons who drink not to such great excess, as steadily—a form of slow chronic poisoning. *Chronic alcoholic insanity* occasionally occurs in confirmed inebriates. The patient has a form of paranoiac deterioration. In addition to the mental failure already noted, he begins to have hallucinations of sight and hearing, somewhat as in delirium tremens. He sees disgusting objects and hears insulting voices, and these are readily excited by every fresh debauch. He then begins to form delusions, often quite well systematized. They are of the persecutory type: he has enemies, who are in league against him, or who would poison him. Sometimes he is markedly hypochondriacal, and has an animal or some unknown disease within him; but his commonest delusion is the delusion of marital infidelity. This is so common as to be held typical by almost universal testimony; and whatever its cause, whether due to failing sexual power or to the natural aversion of the wife, it is in a sense pathognomonic. In time these persecutory delusions may give way to delusions of an expansive type, just as in paranoia, but by that time the chronic inebriate is usually passing into well-marked dementia, and his case is hopeless. Some of these patients under the influence of their delusions commit crime. The terminal stage is one of permanent dementia. In France nearly 14 per cent. of the insane in the asylums are alcoholics.[1] In America the percentage may be not so high. *Epilepsy* is sometimes a result of alcoholism. It is occasionally seen after prolonged sprees in susceptible individuals, but often it sets in later in life in chronic topers. In the latter case it is usually a sign of beginning degradation of brain tissue. *Alcoholic general paresis*, or pseudoparesis, is a condition which closely simulates the genuine disease. There is the expansive psychosis, with tremor and speech defects, but these patients may recover rapidly when they are deprived of their drink. *Dipsomania* is the overpowering impulse to drink which is seen in a class of excitable and neurotic persons, and which, according to Spitzka, is allied to periodical mania. *Korsakoff's psychosis* is a mental disorder, seen especially in alcoholic multiple neuritis, in which there is mental confusion with loss of identity of time, place, and person, and a tendency to fabulation.

Diagnosis.—The diagnosis of the various forms of alcoholic intoxication is, as a rule, easy. The history in most cases is clear and condition unmistakable.

Mistakes, however, occur in the diagnosis of acute drunkenness, especially in hospital practice when the history is unknown. The odor of the breath is entirely unreliable, for the drunken man, or even the man who has only been drinking moderately, may have opium poisoning, or uræmia, or cerebral hemorrhage, or a fractured skull. In opium poisoning there are contracted pupils and slow breathing, and the coma is more profound, as a rule, than in drunkenness. In cerebral hemorrhage there is usually a hemiplegia, which is shown by the diminished resistance to passive motion on the paralyzed side, and there may be unequal pupils and deviation of the head and eyes. Injury to the head, or fractured skull, is usually detected on careful examination. Uræmia is indicated by the state of the urine and the subnormal temperature, but the latter is seen also in alcoholic intoxication, and the former is not always conclusive. Convulsions are rare in drunkenness, but not unheard of.

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[1] Semaine Méd., July 10, 1907.

Delirium tremens is not likely to be mistaken, but when it breaks out suddenly in a surgical case or in pneumonia it may be confusing. The tremor and peculiar wandering delirium, with frightful hallucinations, are significant. Patients with delirium tremens should always be carefully examined for head injuries, fractured ribs, and other bodily damage; also for pneumonia.

The other forms of alcoholic insanity are usually recognized with ease from the history and the character of the symptoms as given above.

II. OPIUM POISONING; MORPHINISM.

The use of opium and its alkaloid, morphia, has increased greatly in America, until it is now one of the recognized evils of the time. The drug is taken in its crude form, or as laudanum, or occasionally as paregoric, but the commonest practice is to use morphia by the hypodermic syringe. Accidental poisoning is rare, but suicide by laudanum or morphia is more common.

Pathology.—Opium or morphia, when taken habitually, is a slow and insidious poison to the nerve centres, but it does not cause such marked organic changes in the tissues as does alcohol. In old *habitués* there is often a condition of malnutrition or cachexia, shown by a sallow complexion, loss of weight, and gastro-intestinal disorders; but some narcomaniacs show remarkably little physical effect from the drug.

Symptoms.—The effects are acute and chronic. The symptoms of *acute poisoning,* after an initial stage of excitement or dreaminess, are somnolence, passing into stupor and coma, congestion and even cyanosis of the face, full and slow pulse, slow respiration, and strongly contracted pupils. As death approaches the pupils may dilate widely, and they may even be unequal; the respirations are not only slow but also imperfect; the surface pallid or cyanosed, and covered with a clammy sweat; and the pulse rapid. Trismus and convulsions are rare symptoms. This stage of opium poison closely resembles apoplexy and uræmia, and a positive diagnosis cannot always be made unless the history is clear.

The *chronic effects* of opium poisoning are seen in the habitual users of the drug, and they are most marked in the nervous system, especially the brain.

Attempts have been made to distinguish the various forms of the opium habit, and their pathological effects. In India opium is eaten; in China it is smoked; and in America it is drunk as laudanum or used as morphia under the skin. To draw distinctions between these varieties of the same vice is, however, hardly possible. Opium, in whatever way it is taken into the system, acts in the end very similarly, especially on the brain and nerves. When taken by the mouth it acts, indeed, more directly and injuriously upon the digestive system; nevertheless old *habitués* acquire a wonderful tolerance. The opium habit becomes a disease, and this disease presents some well-marked symptoms. An abnormal mental state results from the habitual use of the drug. There are moral perversions, enfeebled will, loss of power of attention, and, in advanced cases, a delirious delusional psychosis. In these chronic cases the victim is often insane and irresponsible, although he may be able to present a fairly good front and to conceal his disorder. There is loss of memory, loss of volition and power of attention, loss of initiative and energy, diminished muscular power, often with trembling, blunting of the higher moral and ethical sense, insomnia, nutritive disturbances, and, finally, illusions, hallucinations, and delusions. The tendency of the morphinomaniac to lie and to romance is proverbial. The patient lives so much of his time in an unreal world that he loses his ability to distinguish the true from the false. He becomes both delusional and mendacious. In advanced stages the patient may be violently insane, even maniacal, and may commit acts of violence. Obscure medicolegal problems arise occasionally, for the narcomaniac is sometimes also a kleptomaniac, a pyromaniac, etc.[1]

The physical symptoms vary greatly; in fact, they can hardly be said to be characteristic. Among them are anæmia, rapid exhaustion and lack of endurance, tremor, itching of the skin, anorexia, constipation, wasting, blunting of the special senses, and contracted pupils. The sexual power is weakened and finally destroyed, and in women amenorrhœa is common. Children born of mothers addicted to morphia have a low vitality and often die early.

Diagnosis.—The only rule is to detect the habit. Obscure mental changes, such as those mentioned above, may exist for a long while and not excite suspicion; and he must be a shrewd diagnostician who can detect the morphia taker by his mental symptoms alone. The history, therefore, is all important. Spells of irritability and unrest occur when the victim is deprived of his drug, and are followed by a mysterious calm and serenity when he has taken his secret dose. But such signs require to be interpreted with great caution. Instances are known of judges presiding on the bench, and clergymen preaching in their pulpits, when fully under the influence of their accustomed doses. Diarrhœa, faintness, collapse, are among the graver "abstinence symptoms."

The morphia fiend may also take cocaine, and is occasionally addicted to alcohol, with bromide or chloral as a help. These all-round drug-takers sometimes break down in a state not unlike general paresis—a sort of *pseudoparesis*—from which recovery is possible if they are pulled up in time.

A state of neurasthenia is seen in some morphia takers, especially in women who take small doses and are successful in concealing the habit. Hence the necessity of careful inquiry. The morphia taker often has sores on the skin from the use of the needle.

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[1] Wharton and Stillé's Med. Jurisprudence, 5th ed. Chapt. on "Narcomania" by Lloyd, vol. i, p. 874.

III. COCAINE POISONING; COCAINISM.

Erlenmeyer was the first to describe *cocomania* as a distinct disease. The habit has prevailed only in recent years, for cocaine, the active principle of the coca plant, was only discovered by Gardeke in 1855.

Symptoms.—The *physical* symptoms are loss of weight, muscular weakness, tremor, anæsthesia, and disturbance of the heart and circulation.

The *mental* symptoms are marked. No other narcotic makes such a pleasing impression on the brain. It is a sense of well-being, of strength, and of entire happiness. But the duration is brief. Later, as the habit is formed, a delusional insanity occurs, with hallucinations, which may appear suddenly and develop rapidly. The delusions are of the persecutory type, such as fear of enemies, suspicion of marital infidelity, and dread of burglars. The hallucinations are both visual and auditory, and are disturbing or alarming, such as the sight of insects, the sound of insulting words, etc. One of the most characteristic is the so-called "cocaine-bug:" the patient imagines he feels a hard object beneath the skin of his fingers or hand. It is called Magnan's sign, and is probably due to anæsthesia of the sensory nerve-endings.

Cocaine is a virulent poison to the nervous system—much worse and much more rapid in its effects than morphia. Many of these patients, in fact, are also addicted to morphia. Their career is short; they go to pieces quickly and suddenly; and for some time before the final collapse they have their spells of depression, in which there is acute distress. They are also, sometimes, excitable and violent. A peculiarity of some cases is an extreme slowness of the mental processes, and prolixity in speech and letter writing. The habit has much increased lately, especially among the lower classes, even the negroes in some places becoming addicted.

Diagnosis.—This presents no difficulty, as a rule, for the secret will out. It is important to bear in mind that a large number, probably the majority, of cocomaniacs also use morphia. They are worse physical wrecks, as a rule, than the pure morphia takers. Kerr says that the bodily wasting appears quickly; there is great loss of the sense of time; and in fatal cases stupor and coma, with convulsions, occur. Mosso directs attention to what he calls tetanus of the respiratory muscles. In some cases there is great muscular unsteadiness. Insomnia is common, as in all drug takers when deprived of their allowance. While individually these symptoms are common to many conditions, it is the grouping of them that characterizes the chronic poisoning by cocaine. Delusions and hallucinations are rather more common than in chronic opium poisoning. Some of these patients are covered with wounds and scars from the hypodermic needle.

IV. LEAD POISONING; PLUMBISM.

Lead is widely used in the arts; hence lead poisoning in its various forms is not uncommon. The persons most exposed to the poison are the workmen in white lead factories, painters, and smelters. Plumbism occurs also in type-founders, file-makers, shot-makers, gilders, bronzers, lace-makers, glass-cutters, plumbers, and in other artisans.

Chronic lead poisoning is sometimes unrecognized, for the patient may have been exposed in unsuspected ways, as by hair-dye, food stuffs, etc., drinking water, and may not even know himself the nature of his ailment. The chromate of lead has been used by bakers to give color to their products, as reported by D. D. Stewart, of Philadelphia; wines, beer, and cider have been contaminated by lead pipes and lead vessels; and sewing thread is sometimes weighted with lead, so that poisoning has occurred in a seamstress from the constant biting off of the ends.

Pathology.—Lead affects many of the tissues. In the nervous system it causes a peripheral neuritis, and also poisons the brain-cells, but the pathological changes in the brain, according to Berkley, are not as yet clearly demonstrated. Optic neuritis is seen occasionally. The motor cells in the anterior horns of the spinal cord are sometimes affected. Contracted kidneys and arteriosclerosis are not uncommon in chronic lead poisoning. Gastro-intestinal irritation and inflammation are the results of the ingestion of lead. T. Oliver found changes in the liver, such as atrophy of the hepatic cells and increase of connective tissue. Lead is found in the muscles and in other tissues, and may even be detected in the urine and fæces for long periods after its introduction into the system has stopped.

Symptoms.—These are acute and chronic. ACUTE LEAD POISONING is marked by pain in the stomach and bowel, vomiting, constipation or diarrhœa, the stools being black from the sulphuret; cramps, neuralgic pains, paralysis, and anæsthesia in the limbs; and finally syncope and collapse. Convulsions and coma occur in some cases. CHRONIC LEAD POISONING is known by the following symptoms: a blue line on the gums, anæmia, colic, pain, vomiting, paralysis, anæsthesia, ataxia, tremor, cramps, convulsions, encephalopathy, neurasthenic and hysterical symptoms, optic neuritis or atrophy, contracted kidneys, and the so-called lead gout.

The blue line at the gingival border is very commonly seen, but it has no necessary relationship to the severity of the other symptoms. As a diagnostic sign, however, it is of great value. Anæmia is said to be common among lead miners, and it can occur in any one who is much exposed.

Colic and epigastric and precordial pains are very characteristic. The familiar term "colica pictonum" dates from the seventeenth century when the affection prevailed as an epidemic in Poitou from the contamination of wine with lead. The attacks are often acute, are even seen in persons only recently exposed to lead, and may or may not be associated with wrist-drop. The colic centres about the navel and is often severe and usually without diarrhœa. It may be accompanied with vomiting. This pain and distress about the precordia may simulate angina pectoris or pericarditis. Myalgic and arthralgic pains are also observed, especially about the knees and in the lumbar muscles, and they may simulate gout.

Paralysis is often seen. The commonest form is the familiar *wrist-drop*, caused by involvement especially, but not exclusively, of the interosseous branch of the musculospiral nerve. The supinator longus muscle and the extensor of the metacarpal bone of the thumb, for some unknown reason, always escape, and as a rule there is no anæsthesia. The paralysis is of the peripheral or flaccid type, with muscular atrophy and reactions of degeneration. The hand falls at the wrist and is almost powerless, for not only are the extensors paralyzed, but the flexors act at such disadvantage that they can only be partially brought into use, as can readily be shown by asking the patient to grasp the observer's hand, when the grip will be found weak; but if the hand is passively extended the grip is much improved. This wrist-drop is always bilateral. In some cases the paralysis is not confined to the extensors of the hand: the muscles of the upper arms are occasionally involved, and the deltoids seem especially prone to suffer. Again, the paralysis may be even more wide-spread, involving the legs, and presenting the form of a more or less generalized peripheral neuritis, with abolished deep reflexes.

A true *progressive muscular atrophy* and a pseudotabes, due to lead, are occasionally seen.

Tremor and cramps are not common in lead poisoning; the former resembles other forms of metallic tremor—it is at first fine, gradually increases in amplitude, and is worse on voluntary movement and during emotion. Anæsthesia likewise is rare in uncomplicated cases. This is true especially of the cases of wrist-drop. Slight hypæsthesia or retardation may occasionally be seen. In the rare pseudotabes various modes of anæsthesia are noted, especially deep anæsthesia, such as alteration in the muscular sense, pressure sense, and sense of position. But there is a form of hysterical anæsthesia which is not so rare, as has been pointed out by the French. It may be of the segmental type and is sometimes influenced by suggestion.

Under the head of *encephalopathy* are included a group of symptoms sometimes seen in lead poisoning. These comprise headache, confusion, delirium, convulsions, and coma. The attack may come on suddenly, and is seen even in persons not long exposed, but it is probably more common in the victims of chronic poisoning. There is sometimes an initial headache, with restlessness and insomnia, or the attack begins abruptly with a fit. The delirium persists for some hours or even days, and is accompanied with hallucinations of sight and hearing. The convulsions may recur, the delirium persisting between them. An isolated attack, without delirium before or after it, may also occur. If the convulsions recur frequently, the patient may pass into an epileptic status, with fever, coma, stertorous breathing, rapid pulse, and failing vitality, and die. If these attacks occur often there is risk of permanent damage to the brain, as is shown by recurring epilepsy, maniacal seizures, melancholia, and dementia. It must not be forgotten that a type of so-called hystero-epilepsy, totally different from the preceding, sometimes occurs in lead poisoning; it is purely hysterical, and is to be known by the hysterical stigmata. Changes in the optic nerves occur in lead poisoning. Atrophy is observed, and it may follow a neuritis. The association of kidney disease with chronic plumbism must not be overlooked, and hence the possibility of albuminuric retinitis.

Neurasthenic and hysterical symptoms may complicate lead poisoning. Among the symptoms are anæsthesia, including hemianæsthesia and segmental anæsthesia, tremor, hysterical paralysis, and hysterical convulsions. The association of gout with plumbism is insisted on by many English physicians. They point to the contracted kidneys, arteriosclerosis, arthralgia, and in some cases arthritic changes and deposits, as evidences of this "lead-gout." Bright's disease is a not uncommon complication of chronic lead poisoning.

Diagnosis.—When the history is clear the diagnosis is easy; but when the history of exposure to lead is wanting, the case may be most obscure. The blue line on the gums is of the greatest value, but it is not always present. The precordial pain in lead poisoning may simulate angina pectoris, but the history and the blue line are usually determinative. The wrist-drop due to lead is always bilateral, although it may be worse on one side; and the supinator longus and the extensor of the metacarpal bone of the thumb escape; thus the case differs from one of trauma of the musculospiral nerve, which is usually unilateral, and involves more muscles. Other forms of paralysis are rare in lead poisoning, but the history and the blue line are usually clear. The pseudotabes due to lead is distinguished from locomotor ataxia by the presence of muscular atrophy and the reactions of degeneration, and the absence of fulgurant pains, Argyl-Robertson pupils, and other true tabetic symptoms. Lead encephalopathy closely simulates uræmia, also idiopathic epilepsy. If the history is obscure the difficulty may be great. The blue line would be important, and there may be an absence of urinary symptoms. If the history is clear there need be no great difficulty in the diagnosis of epilepsy, of uræmia, or of encephalopathy.

The stigmata of hysteria can usually be recognized with a little care. Hemianæsthesia and segmental anæsthesia are not caused by lead alone. Traces of lead in the urine are important.

V. ARSENICAL POISONING.

Chronic poisoning by arsenic is much less common now than some years ago, partly because of more widely disseminated knowledge concerning the dangers of the use of this metal and its salts in the arts and partly because of the enactment of laws limiting the amount of arsenic in wall-papers and dress goods. This form of intoxication is at present occasionally encountered in persons engaged in certain occupations, as the manufacture of wall-papers and other papers, playing cards, book covers, and artificial flowers. Those who handle, and more particularly those who wear, articles of apparel, such as stockings, gloves, and certain dress fabrics, dyed with arsenical pigments in excess, or furs cured by arsenic, are exposed to a theoretical danger which is, however, trifling as compared with that of a generation ago. Not only the greens, as popularly supposed, but dyes of various colors often contain arsenic in dangerous amounts. The danger of acute or chronic poisoning as the result of the introduction of arsenic into articles of food as a preservative is no longer to be considered.

It is a remarkable fact that a large proportion of cases of chronic arsenical poisoning at the present time are caused by the use of this metal for therapeutic purposes. This untoward occurrence may be due to excessive doses, an improper prolongation of the treatment by proper doses, or to an unusual degree of susceptibility on the part of certain individuals to some of the toxic effects of arsenic. Patients and, in particular, those subject to diseases of the skin frequently continue the use of prescriptions containing arsenic without regularly reporting to the physician. It is important that medical men should protect themselves and their patients by emphasizing the risks attendant upon such a course. The growing use of less toxic arsenical preparations, as atoxyl and sodium cacodylate, will diminish this danger.

Arsenic eating, as practiced in Styria and elsewhere for the purpose of stimulating the powers of endurance and the sexual capacity or improving the complexion, has in many cases resulted in chronic poisoning. The presence of arsenic in beer, derived from the sulphuric acid used in the manufacture of the glucose employed in brewing, recently attracted much attention in England as a source of danger. The use of Paris green and other arsenical preparations as insecticides is attended with little risk in itself. The chief danger lies in having such substances about under conditions in which they may give rise to accidental or intentional poisoning. Miners and smelters of ores containing arsenic are much exposed to the danger of chronic poisoning. Acute arsenical poisoning is rarely accidental, but by no means uncommon in suicidal or homicidal cases.

Pathology.—In the acute form gastro-enteritis, nephritis, and fatty changes in the muscles and viscera, especially the liver, constitute the chief lesions; in the chronic form anæmia, pigmentation of the skin, and lesions of the nervous system, particularly of the peripheral nerves, are common.

Symptoms.—The chief clinical manifestations of chronic arsenical poisoning relate to the skin and the peripheral nervous system. The general health may not be at first greatly impaired. As a rule, however, anæmia, emaciation, loss of strength, cardiac asthenia, and vasomotor derangements are present in varying degree.

THE SKIN.—Hyperidrosis, glossiness, local ulceration, herpes, and erythromelalgia are occasionally observed. Pigmentation is very common and often marked. It varies from a faint brownish-yellow to a deep brown, and is sometimes distributed over the greater part of the surface; sometimes collected in circumscribed areas and frequently diffuse, with patches of deeper coloring upon exposed parts, in the folds about the joints, and in regions normally pigmented, as the nipples and pudenda, especially in those of dark complexion. Small dense collections of pigment may form and present the appearance of pigmented moles. Patches of pigmentation are occasionally observed upon the mucous membranes. As a rule, to which there are exceptions, the skin, after the exposure to arsenic ceases, gradually resumes its normal appearance. Keratosis is less common. It is usually confined to the palms and soles and occurs in circumscribed patches. Epitheliomatous degeneration may occur. Less common are erythema, which is sometimes symmetrical, polymorphous lesions, furuncles, acne, depraved nutrition of the nails, and alopecia. Puffiness of the eyelids is a common and early symptom.

THE NERVOUS SYSTEM.—The principal symptoms are those of a peripheral neuritis, which involves the lower extremities much more commonly than the upper, and when both are affected the lower to a greater degree. Motor derangements, varying from a slight palsy to actual paralysis, may appear in a few hours, or not for several days after a single toxic dose, when the acute gastro-intestinal and depressive symptoms have wholly subsided. In some instances palsy has appeared several weeks after a single poisonous dose, recovery having in the intervening period been apparently complete. In chronic poisoning the paralysis develops gradually after a variable period, the length of which is determined by individual peculiarities and the degree of intoxication. DERANGEMENTS OF SENSATION consist of paræsthesias of various kinds, and pain. The latter is frequently severe and associated with tenderness along the nerve-trunks. Anæsthesia, hyperæsthesia, polyæsthesia, and other derangements of sensation occur. The nervous symptoms, both motor and sensory, do not often extend above the knees or elbows, and the thighs and arms, together with the trunk, remain uninvolved. The sphincters escape. In severe cases the muscles of the legs and feet and arms and hands undergo atrophy; the knee-jerks are lost. Reactions of degeneration are present and contractures occur. There are cases in which the resemblance to tabes is very striking. Mental symptoms occur in severe and prolonged cases. They comprise loss of memory, mental weakness, hallucinations of sight and hearing, and may gradually assume the form of a terminal dementia.

Diagnosis.—DIRECT.—This rests upon the association of the foregoing symptoms, especially those relating to the skin and nervous system, and a history or knowledge of exposure to, or the ingestion of, arsenic. The disappearance of the symptoms upon the removal of arsenic would render the diagnosis positive. When any of the above symptoms arises in a patient who is under treatment by arsenic for chronic skin disease, anæmia, chorea, habit-spasm, or Hodgkin's disease, or in any person who is employed in arts or manufactures in which arsenic is freely used, or who lives under conditions involving exposure to arsenic, who habitually eats it for any purpose, or who has a history of acute poisoning by arsenic, a provisional diagnosis of chronic arsenical poisoning is justified but should be at once tested by the complete withdrawal of the patient from exposure to the action of arsenic in any manner whatever.

DIFFERENTIAL.—Peripheral neuritis due to other causes, as lead, alcohol, and the infections. Lead palsy may be recognized by the absence of pain, the more common and severe implication of the upper extremities, bilateral wrist-drop without involvement of the supinator longus or the flexors, the blue line on the gums, colic and constipation, and a known history of exposure to lead. Alcoholic neuritis may be recognized by pain, a history of alcoholic excesses, other signs of alcoholism, as the facies, the condition of the mind, or visceral lesions, as for example fatty liver or cirrhosis. Post-infectious neuritis usually shows a clear history of some foregoing acute or chronic infectious disease, as enteric fever, diphtheria, or pulmonary tuberculosis. Tabes dorsalis may be distinguished from the rare cases of arsenical poisoning which somewhat resemble it, by the gradual onset, the commonly prolonged and progressive course, the ocular phenomena, the various "crises," a history of syphilis in nearly every case, the lightning pains, and the absence of the cutaneous lesions produced by arsenic.

In any doubtful case it may be necessary to examine the urine for the presence of arsenic, though it must be borne in mind that arsenic is not always present in the urine when it is known to have been ingested, and that when present it disappears within two or three weeks after its administration has been discontinued. For this purpose it is best for the practitioner to secure the services of a competent chemist.

VI. POISONING BY MERCURY.

As in the case of arsenic, chronic poisoning by mercury is far less common than formerly, because the employment of this metal in the arts is now much restricted and far greater care is exercised in its use in medicine.

Etiology.—As an occupation disease chronic mercurial poisoning occasionally occurs in those employed in making certain dyes, the use of amalgams, the preparation of fulminate, the manufacture of fireworks, among taxidermists, and in those engaged in the making of felts for hats and other purposes (Edsall). Especially hazardous are occupations such as the mining and smelting of mercurial ores. The manufacture of scientific instruments depending upon the physical properties of mercury, as thermometers, barometers, and manometers, involves prolonged exposure to the risk of a slow intoxication. The number of persons engaged in these various occupations is comparatively limited, but for this very reason a knowledge of the effects caused by mercury, while of relatively slight importance to the general practitioner, is of especial interest to the hygienist because they are preventable, and to the diagnostician because they are rare. It is of diagnostic importance that not only those actually engaged in occupations concerned with mercury are liable to this metallic intoxication, but also others who breathe an atmosphere containing volatilized or dust-borne mercury, or drink water in the neighborhood of mines.

Pathology.—Chronic gastro-enteritis, anæmia, emaciation, and fatty degeneration of the viscera are encountered. Neuritis is very rare. Its occurrence has been questioned.

MODE OF ACCESS.—Chiefly by inhalation, since mercury volatilizes at ordinary temperatures; to a subsidiary extent through the skin. In medicinal mercurialization most commonly by ingestion; less frequently by inunction. Fortunately ptyalism, mercurial stomatitis (which see), is a danger signal and usually leads to the immediate discontinuance of the drug.

Symptoms.—Ptyalism does not, however, always occur in chronic mercurial poisoning. In a small proportion of the cases it is an early and persistent condition. Early symptoms are headache, disturbed and unrefreshing sleep, and mental and physical depression, especially in the early hours of the day. Anæmia is common. The association of tremor and a curious emotional condition suggestive of hysteria dominate the symptom-complex. Tremor is usually at first absent when the patient is at rest, but shows itself at once upon voluntary movement and is greatly aggravated by emotional excitement. It chiefly involves the hands and lips, but may, in severe cases, affect all the extremities to such an extent as to interfere seriously with the ordinary movements of everyday life. In the early cases the tremor is fine, but later it increases in amplitude and becomes coarse and rapid. When it affects the muscles of articulation, speech is much deranged and this, with the tremor of the lips and tongue, may suggest disseminated sclerosis. Attacks of vertigo occasionally occur. In grave cases muscular weakness, choreiform movements, and various palsies, together with anæsthesia and other disturbances of sensation, are encountered. These motor and sensory phenomena are, as a rule, irregularly distributed, incomplete, and transitory. Epileptiform convulsions, paroxysmal tonic spasm involving chiefly the flexors of the forearm, and clonic spasms also paroxysmal and affecting all the muscles of the body but without loss of consciousness, have been described. The emotional derangements assume a form highly suggestive of hysteria. But the conclusion that groups of previously healthy individuals collected in one locality from various sources and developing emotional symptoms along with the other phenomena and the tremor, after prolonged exposure to mercurial poisoning, should be hysterical is unwarranted. This group of symptoms includes sensations of weakness, powerlessness, and fright, which, in rare instances, gradually increase to actual obsessions of fear and doubt which render the life of the patient one of abject wretchedness and have in some instances amounted to a form of dementia.

Diagnosis.—DIRECT.—The fact of habitual exposure to mercury, either in occupation, therapeutically, or under other circumstances mentioned under the subtitle *etiology*, together with the character of the tremor and the emotional disturbances, render a positive diagnosis a comparatively easy matter. If necessary a chemical examination of the urine may be made.

DIFFERENTIAL.—The following conditions are to be considered: *Hysteria.*—The presence of the stigmata of this condition (see article on Hysteria) and of the visual phenomena, the intermittent and paroxysmal character of the symptoms in many cases of hysteria, and the absence of the peculiar tremor of mercurial intoxication, are significant. *Disseminated Sclerosis.*—The finer tremor, absence of emotional or hysterical phenomena, the staccato speech, nystagmus, occasional focal phenomena, and the persistent and progressive character of the symptoms, are important in the differential diagnosis. *Paralysis Agitans.*—The passive tremor, unemotional mental condition, peculiar mask-like facies, persistent muscular rigidity and festination of this condition are in strong contrast with the symptom-complex of mercurial intoxication. *Paresis.*—The tremor is suggestive, but the differentiation may be made without difficulty. The emotional states are wholly different. In paresis delusions of grandeur and perversions of the moral sense occur. There are apoplectiform seizures, epileptiform convulsions, remarkable remissions of all symptoms, occasional focal phenomena, and progressive mental and physical deterioration. *Alcoholism.*—The two conditions are frequently associated. The anamnesis, the slight degree of influence of emotional states upon the tremor, and the facies, dilated venules, circulatory and visceral conditions attendant upon chronic alcoholism, are important. *Plumbism.*—The history of exposure to lead, constipation, colic, the gingival line, and the wrist-drop are characteristic.

Prognosis.—In the early cases the outlook is favorable. Even after prolonged and well-characterized symptoms recovery may take place.

In such cases, however, tremor may persist. Persistent palsy, headache, progressive anæmia, and the cachectic state are unfavorable as regards prognosis. Change of occupation and complete freedom from exposure are essential to recovery. If such requirements cannot be met the prognosis is uncertain or unfavorable.

VII. PHOSPHORUS POISONING.

Etiology.—Acute poisoning by phosphorus is rare and usually results from the swallowing of match-heads with suicidal intent. Chronic poisoning is likewise rare and occurs as an industrial disease in localities in which matches are manufactured.

Pathology.—ACUTE PHOSPHORUS POISONING.—The coagulability of the blood is reduced; there are diffuse hemorrhages into the skin and viscera and from mucous surfaces; jaundice; fatty degeneration of the muscles and parenchymatous organs, especially of the liver, which also rapidly undergoes enlargement and changes to a bright saffron color. Leucin, tyrosin, cystin, sarcolactic acid, peptones, and sugar are found in the urine and blood. Acid intoxication occurs, as a result of which the urinary ammonia is greatly increased and the urea decreased. In its derangement of metabolic processes phosphorus acts like a ferment.

CHRONIC PHOSPHORUS POISONING.—In man the chief pathological change consists of necrosis of the inferior maxilla with suppuration and the formation of sequestra. Exceptionally the upper jaw bones are affected. This process is accompanied by destructive ulceration of the soft parts with more or less abundant pus formation.

Symptoms.—Acute poisoning presents a close clinical resemblance to acute yellow atrophy. Early symptoms are vomiting and diarrhœa, which presently subside, to return in the course of forty-eight or seventy hours, and are then accompanied by intense jaundice, epigastric distress, and diffuse pains in the muscles. At this time petechiæ, submucous hemorrhages, and blood in the vomitus and stools appear. When, as is not rarely the case, match-heads have been taken to produce abortion, this accident frequently occurs. There is profound asthenia, with maniacal excitement which rapidly passes into stupor and coma and is followed by death. The vomited matters may be phosphorescent.

The necrosis of the jaw, which constitutes the chief morbid condition in the chronic form, commonly begins about a single tooth, with caries and abscess formation. The process involves the tooth and surrounding alveolar process and rapidly extends to the neighboring teeth and contiguous structures. The pus is often abundant and very foul. It burrows in various directions and may form sinuses which discharge in the neck. One or several sequestra may form. Rapid anæmia and general sepsis may occur, and in neglected cases amyloid disease, tuberculous infection, and basal meningitis have been observed.

Diagnosis.—Both in acute and chronic phosphorus poisoning, the diagnosis depends largely upon the anamnesis. In the former suicidal intention, or rarely the eating of match-heads by young children; in the latter the exposure to phosphorus, which is volatile at ordinary temperatures, as an occupation risk, are of great diagnostic significance. The main points of discrimination from acute yellow atrophy of the liver consist in the period of relief which occurs in the interval between the early gastro-intestinal symptoms in phosphorus poisoning and the graver phenomena, the enlargement of the liver, the occasional absence of leucin and tyrosin or their relatively smaller amount, and the less intense nervous symptoms. There are, however, cases in which, in the absence of a clear history, the differential diagnosis is attended with difficulty. As to chronic poisoning, there are no conditions characterized by similar persistent and extending necrotic processes in the jaw. Actinomycosis may be recognized by the presence of the ray fungus in the pus.

Prognosis.—The mortality in acute poisoning is about 50 per cent. The fatal issue occurs in less than a week. In the chronic form recovery frequently follows early operation. In neglected or very severe cases, even extensive resection may fail to arrest the advance of the necrotic process.

VIII. ACUTE AND CHRONIC POISONING BY ILLUMINATING GAS.

Etiology.—A number of gases enter in varying proportion into the composition of illuminating gas, but carbon monoxide constitutes the chief toxic agent. Water gas is especially active as a poison, because of the relatively large amount of carbon monoxide which it contains.

Acute poisoning commonly occurs as the result of the escape of gas by way of an unlighted burner into sleeping-rooms. This has resulted accidentally from a gust of wind, from leaving the gas turned on, from ignorance, or with suicidal intent. This method of suicide has become very common and when, as is usually the case, the access of air is carefully guarded against and rubber hose, is used it is a very certain one. Acute poisoning may occur, however, in those employed in gas works, and I have seen a case in which it resulted from the escape of gas from a street main. Chronic poisoning is probably rare and is likely to arise among those employed in gas works or those dwelling in houses into which slow but continuous leakage occurs from pipes or fixtures. Toxic symptoms may arise upon breathing an atmosphere containing 0.02 per cent. of carbon monoxide; 0.05 is highly dangerous, and above this the air speedily becomes irrespirable.

Pathology.—The toxic action is chiefly upon the red blood-corpuscles, the oxyhæmoglobin being converted into carbon monoxide hæmoglobin, and the function of the affected corpuscles as carriers of oxygen and carbon dioxide destroyed. The blood is cherry-red in color. There are areas of bluish-red discoloration upon the neck, chest, and elsewhere, visceral hyperæmia, and local hemorrhages. In the chronic cases fatty degeneration of the heart, anæmia, and enlargement of the spleen have been noted.

Symptoms.—The clinical phenomena depend upon the proportion of gas in the atmosphere and the duration of exposure. Progressively they are as follows: malaise, sensations of throbbing, especially in the head, headache, vertigo, muscular weakness, nausea, vomiting, drowsiness, loss of consciousness, and relaxation of the sphincters. Muscular twitchings and general convulsions occur. In the comatose state there are rapid respiration, a rapid and full pulse, and cyanosis. The blood is cherry-red in color and there is leucocytosis, high in proportion to the gravity of the case. When recovery takes place, sequels relating to the respiratory tract, bronchitis, and bronchopneumonia are common. Lobar pneumonia is rare. Cardiac derangements of a functional kind also occur. To these, in many cases, are added gastric irritability and epigastric pain and tenderness, symptoms of a subacute gastritis. Icterus has been observed and glycosuria is common. Local œdema and various inflammatory and necrotic cutaneous lesions occur. Nervous symptoms are common and important. They comprise various neuralgias and forms of neuritis, tremors and choreiform movements, and neurasthenic manifestations, among which fatigue symptoms are very marked. Amaurosis, nystagmus, and ocular palsies may occur. Persistent headache, dulness of hearing, and tinnitus have been noted. Psychical derangements vary from confusional states with hallucinations to dementia. The foregoing sequels may develop directly after exposure, or they may come on after several days or even some weeks of apparent recovery and undergo gradual intensification. In the suicidal cases the antecedent condition of the patient is to be considered in estimating the actual relation between the carbon monoxide poisoning and the mental state at a more or less remote period after recovery from such poisoning. The symptoms of chronic poisoning are those of the milder forms of acute poisoning continued through an indefinite period, namely, headache, vertigo, nausea, occasional vomiting, muscular weakness, fatigue symptoms, and inability to perform the ordinary physical and mental duties of life. A slow pulse, anæmia, and absence of the deep reflexes have been observed.

Diagnosis.—DIRECT.—This rests upon the history of the case, which is usually very clear, and the demonstration of carbon monoxide in the blood.

The most satisfactory tests are: (a) Hoppe-Seyler's sodium hydrate test: A solution of specific gravity of 1.30 is added to the blood; if carbon monoxide is present the clot formed is of bright red color, while with normal blood the color of the mass is greenish-brown. (b) Katagáma's ammonium sulphide and acetic acid test: To 10 c.c. of blood diluted with water are added, first, 0.2 c.c. of ammonium sulphide solution, and then 0.2 c.c. of 36 per cent. acetic acid. Blood containing carbon monoxide gives a bright red precipitate; normal blood a green precipitate. (c) The Kunkel-Welzer test consists in the addition to the undiluted blood of an equal volume of 20 per cent. potassium ferrocyanide and a small quantity of 36 per cent. acetic acid. Carbon monoxide blood yields a bright red reaction, while the color of normal blood changes to a deep brown. The spectroscopic test may be employed.

In a comatose patient of obscure or uncertain antecedents a positive result may be of great importance, (a) in the immediate diagnosis, (b) in determining the character of later morbid conditions, and (c) from a medicolegal point of view.

DIFFERENTIAL.—*Alcoholic Coma.*—The anamnesis is most important. The fact is, however, to be borne in mind that a drunken man may blow out the gas, turn the burner off and on again, or conclude to end his life by gas poisoning. The history of a spree and the odor of alcohol have, therefore, no positive value in the differentiation of these two conditions. Of greater moment are the appearance of the blood, the above-named tests, cyanosis, and the cutaneous hyperæmia. *Uræmic Coma.*—Urinary suppression, the presence of albumin, and casts, dropsy, and cardiovascular lesions are of importance, particularly when associated with negative results upon testing for carbon monoxide in the blood.

The diagnosis of chronic poisoning by illuminating gas is not readily made. When all the occupants of a dwelling habitually awake with nausea, headache, and vertigo and these symptoms steadily increase, and there is associated muscular and mental weakness and depression, it would become necessary to carefully test the gas-pipes and fixtures. Under such circumstances the gas present in the atmosphere might produce toxic symptoms, though insufficient to give positive reactions either in the air or in the blood of patients. If certain members of the household, upon removal to a different locality, were gradually to recover their health, while those remaining continued ill, chronic gas poisoning would become a sound provisional diagnosis and the point of departure for further systematic investigation.

IV.

THE DIAGNOSIS OF FOOD POISONING.

Certain articles that are always injurious are, sometimes through ignorance or by accident, used as foods. Examples of these are poisonous mushrooms, which contain muscarine, and some species of fish. The poison present in such substances is said to be *endogenous*. Entirely different are the poisons which are occasionally present in foods otherwise normal. These consist of, (a) poisonous metals, (b) animal parasites, (c) fungi, and (d) bacteria. These poisons are *exogenous*.

(a) POISONOUS METALS.—This subject is considered under the appropriate headings (see pp. 881, 884, and 887, etc.). The contamination of water in the neighborhood of mines—lead, mercury; of beer by arsenic (outbreak in Manchester in 1900); of wine (lead shot used in washing bottles); the presence of chrome yellow as a coloring matter in cakes; and the possibility that canned vegetables and other foods may become poisonous by slow chemical changes of the lead in the solder or the tin, are to be considered. Under all these conditions the poisoning is chronic, the symptoms are slowly progressive, and a number of persons are affected, very often within the boundaries of a limited district. (b) ANIMAL PARASITES.—Certain meats are occasionally rendered poisonous by the presence of trichina or cysticercus (q.v.). (c) FUNGI.—The grains used for food may be infected with the ergot fungus, spurred rye—Claviceps purpurea—and become the cause of outbreaks of epidemic disease—*ergotismus*. (d) BACTERIA.—Vegetable micro-organisms constitute the chief factor in poisonous foods. Meats obtained from diseased animals may give rise to actual infections

Foods contaminated with specific infectious organisms, for example, B. typhosus, may cause circumscribed outbreaks of enteric fever. Finally foods infected with saprophytic bacteria, which evolve poisonous products in the substance of the food itself, may become highly poisonous—*ptomaine poisoning*—a term which in popular parlance is used interchangeably with "food poisoning."

I. **Fish Poisoning—Icthyismus; Ichthyotoxismus.**—The toxic substances may be endogenous, as in species of Tetrodon and Diodon, found in Japan and the East Indies. The nature of the poison is unknown. It is located in the testicles and ovaries. In its effects it resembles curare. The symptoms are vertigo, vomiting, dyspnœa, cyanosis, muscular relaxation, and dilatation of the pupils. Death results very rapidly. This is the *fugu* poisoning of Japan. In certain species of sturgeon, pike, and barb a poisonous substance is developed during the spawning season. The intoxication is grave and often fatal, the symptoms being those of an acute gastro-enteritis. Exogenous poisons are more common. If diseased fish is eaten raw, the specific infection may be communicated, or wholesome and edible fish may, if not properly cared for, speedily develop toxic substances associated with putrefactive changes. These substances, in some instances, resist boiling, so that the infected flesh is also injurious after cooking. The intensely poisonous ptomaines are present during the early days of putrefaction, even before changes recognizable by the taste or smell have occurred, while those of a later period are less toxic. The symptoms do not usually appear until after a period of eight to twenty-four hours. There are two groups of cases. In one the clinical phenomena relate chiefly to the nervous system. These are collapse symptoms, with subnormal temperature, abdominal pain, dry mouth, inability to swallow, dull pain in the belly, dyspnœa, and nervous symptoms, as vertigo, dilatation of the pupils, and diplopia. In another group the clinical manifestations are those of an acute gastro-enteritis, uncontrollable vomiting, griping pains, diarrhœa, and profound cardiac asthenia. Beri-beri and leprosy have been ascribed to the habitual eating of certain kinds of fish.

SHELL-FISH.—Poisoning by mussels is not uncommon in Europe and Great Britain. Brieger, in 1885, isolated a ptomaine—*mytilotoxin*—which proved to be highly poisonous. It resists the temperature at which the mussels are cooked, and in this respect mussel poisoning is analogous to poisoning by mushrooms. The poison is not regarded as endogenous but as the result of changes caused by bacteria present in polluted waters. The symptoms are variable, sometimes not occurring until after the lapse of several hours and being choleraic in character; in other cases not showing themselves for a few days and indicating an action of the poison upon the nervous system. They consist of a general urticarious eruption, associated with asthma-like attacks of dyspnœa. Recovery takes place in the course of several days. In other cases the symptoms come on rapidly and resemble those produced by curara. Death has occurred with great rapidity.

OYSTERS which have begun to decompose, and those obtained from beds in waters defiled by sewage, frequently cause poisoning, with gastro-intestinal symptoms. Intense and fatal poisoning from this cause is very infrequent. Much more important is the occasional conveyance of specific infections, especially that of enteric fever, and the causation of epidemics by the eating of oysters and other shell-fish from sewage-polluted water.

LOBSTERS AND CRABS, when not fresh, frequently cause symptoms of poisoning similar to those produced by other fish and shell-fish under like circumstances. All kinds of canned fish may, under certain conditions, as decomposition, or infection previous to canning, or injury to the cans, develop poisonous qualities.

II. **Meat Poisoning—Sausage Poisoning.**—As in the case of fish poisoning, the great majority of the cases are due to bacterial infection, either specific, as in the case of animals infected at the time of slaughter, or accidental, from contact with various articles that are unclean, or from improper care in other respects. In the former case the organisms are those comprised in the *paratyphoid* and *paracolon* groups; in the latter they are saprophytes, as Proteus vulgaris and B. botulinus, or members of the *colon* group. Clinically the cases may be referred to two categories: first, those in which the symptoms chiefly relate to the nervous system, and second, those in which they are gastro-intestinal. Botulismus—allantiasis—sausage poisoning, a specific intoxication caused by B. botulinus, comes on twenty-four or thirty-six hours after eating the food. The symptoms comprise, on the one hand, gastro-intestinal derangements, as epigastric distress, nausea, vomiting, sometimes diarrhœa, sometimes constipation, dryness of the mouth and throat, choking attacks, and stomatitis, with tough, adherent secretion; on the other, nervous disorders, as dimness of vision, mydriasis, diplopia, strangling sensations, aphonia and profound muscular weakness. The pulse and temperature remain normal. Recovery is slow. In fatal cases maniacal delirium, passing into coma, constitutes a terminal event.

III. **Poisoning by Milk and Milk Products.**—Milk is especially exposed to bacterial infection, and constitutes a favorable culture medium. For these reasons as a raw food it is an abundant cause of intoxications and infections. Boiled milk, properly protected until used, is safe. Pathogenic bacteria may reach milk directly from a diseased animal, as in tuberculosis, or by way of water polluted with excreted matter, as in enteric fever, or from cases of diphtheria or scarlet fever in various ways. The gastro-intestinal diseases of infants in hot weather are due to the bacterial infection of milk. Acute intoxications, in contradistinction to the specific infections, are common, and not only milk itself, but also articles made from it, such as ice-cream, custards, and cream puffs, may give rise to serious poisoning. Vaughan isolated from cheese a poisonous ptomaine, which has been found in milk, but among the milk poisons it is not "the one most frequently present, nor is it the most active one." Of the many different bacteria for which milk forms a culture medium, each has its special toxin. The bacteria which have been especially studied belong to the B. enteritidis group. These organisms do not cause any apparent change in the milk, which presents an alkaline or amphoteric reaction, and is not curdled.

Cheese frequently develops highly poisonous qualities. The accidental introduction of various toxin-producing bacteria and their development are very common. The toxins are probably different, but have not yet been fully studied. Tyrotoxicon was the first to be isolated. The symptoms of cheese poisoning are those of acute gastro-intestinal irritation.

Diagnosis.—Food poisoning may be recognized by the history of the case in respect to the eating of certain articles, and the character of the symptoms, which are usually urgent and nervous, or gastro-intestinal, or both. It commonly occurs in circumscribed epidemics, and all the victims are seized at about the same time, which varies according to the nature of the intoxication from a brief period to two or three days. In a suspected case the careful investigation of the antecedent facts, and the character of the symptoms, are sufficient for a provisional diagnosis, which may be confirmed by bacteriological studies, including agglutination tests.

IV. **Grain and Vegetable Poisoning.**—ERGOTISMUS.—The cause is a parasitic fungus—Claviceps purpurea—which grows in the flowers of several grains, especially rye, and is known as ergot. It contains a number of poisons, among which the more important are *sphacelinic acid*, the cause of the trophic or gangrenous form, and *cornutin*, the cause of the nervous or convulsive form of the disease. Ergotismus does not occur in America but is frequently epidemic in certain parts of Europe. It is due to the prolonged ingestion of ergot and is a chronic intoxication, though the onset may be marked by acute symptoms. In the gangrenous form, distant parts of the body, as the toes, fingers, ears, and the tip of the nose, suffer, and the necrosis is preceded by tingling, anæsthesia, spasms, and signs of local congestion. In the nervous form, the chief symptoms are weakness, headache, cramps in the muscles, and contractures. There may be moderate fever with mania, and, in the severer cases, melancholia and dementia occur. There are tabetic symptoms, and at the autopsy sclerosis of the posterior columns has been observed.

LATHYRISMUS; LUPINOSIS; VETCH POISONING.—Chick-pea poisoning occurs in extended outbreaks in Austria, Italy, Northern Africa, and India as the result of the admixture of the powdered seeds of Lathyrus sativus with flour from wheat and other cereals in the making of bread. The symptoms are pain in the lumbar region, girdle sensations, spastic paralysis of the lower extremities, which may increase to complete paraplegia, tremor, and fever.

PELLAGRA; MAIDISMUS.—An affection attributed to the continued eating of food prepared from fermented or diseased Indian corn. See page 854.

MUSHROOM POISONING; MYCETISMUS.—The symptoms are due to putrefactive and other changes, as autolysis, or the growth of parasitic fungi in the edible varieties; or the accidental ingestion of inedible varieties containing muscarine, phallin, helvellic acid or other toxic substances. They are (1) gastro-intestinal and may come on in the mild cases promptly after the eating of the poisonous fungi, or in the intense or choleraic cases not for several hours; (2) cerebral or nervous and characterized by contraction of the pupils, alternating mania and depression with muscular cramps, salivation, slow pulse and collapse; and (3) hæmatogenous, as subcutaneous œdema and hæmoglobinuria. The last have been produced experimentally by phallin, which has a marked hæmolytic action and causes hæmorrhages and fatty degeneration of the viscera. On the whole our knowledge of the subject is unsatisfactory. The common edible mushroom is *Agaricus campestris.*

Potatoes.—Local outbreaks of acute poisoning traced to eating potatoes that have sprouted have recently been recorded. The toxic principle is *solanin*—present in considerable amounts as the result of the growth of the Bacterium solaniferum colorabile and B. solaniferum non-colorabile. The symptoms are those of an acute gastro-intestinal catarrh, with headache, jaundice, and great prostration.

Examination of Food in Cases of Suspected Food Poisoning.—As much of the food as can be obtained should be preserved for examination. The quantity is usually small. The investigation should be conducted without unnecessary delay. Meanwhile, perishable articles should be kept on ice without the addition of chemical preservatives. The bacteriological examination should precede the chemical unless there are clear indications of poisoning by definite substances, as arsenic, lead, etc.

The methods comprise animal experimentation by feeding, the injection of sterile water in which the material has been macerated, further injections of such macerations after filtration through a Berkefeld or Pasteur filter, and the determination, when necessary, of the presence of a heat-resisting toxin by injecting the macerations after boiling. Culture and agglutination methods are necessary.

V.

THE DIAGNOSIS OF AUTOINTOXICATIONS.

The term autointoxication is used to designate the intoxications of endogenous metabolic origin. It has been used vaguely for a long time to suggest hypothetical conditions rather than demonstrable facts. Quite recently, under the application of scientific methods, the subject has been to some extent cleared up.

I. **Gastro-intestinal Autointoxication.**—There appears to be no proof that intoxication takes place from the resorption of digestive juices, or of the products of normal digestion, or of the abnormal products of digestion, except in the case of the acetone bodies. Nor has it been demonstrated that, in the normal action of bacteria upon the contents of the alimentary canal, toxic substances are produced. Even in the case of intestinal putrefaction, which, to some extent, is a normal process, an increased amount does not necessarily mean an intoxication. Intestinal putrefaction is largely dependent upon the diet; an excess of protein affords an abundant medium for bacterial growth, yet there is no constant ratio between the protein intake and the output of aromatic substances. An increase of these bodies, especially indican in the urine, actually indicates increased bacterial activity, whereas it is constantly assumed to be the sign of an intoxication. Intestinal putrefaction is to be distinguished from tissue putrefaction, which is the cause of an excess of aromatic bodies in the urine. The aromatic bodies are not in themselves toxic. It has been assumed that other substances of a poisonous character are produced by putrefaction, and that these, like the amount of putrefaction, may be approximately estimated by the aromatic substances. In point of fact the aromatic substances in the urine afford no indication of the presence or amount of any hypothetical poison and bear no constant relation to the symptoms in any particular case.

Tetany.—The extremely rare and fatal tetany in adults, occurring in the dilatation of stomach, has been attributed to poisons produced by the decomposition of food.

Gastro-intestinal Attacks Associated with Cutaneous Symptoms.—The seizures are acute and recur periodically. They consist of epigastric pain, vomiting, diarrhœa, and various skin eruptions, most commonly urticaria and erythema. Desquamation may occur.

Acute Paroxysmal Gastro-enteritis.—The attack occurs suddenly in the absence of errors in diet, particularly in the absence of food poisoning. The symptoms are vomiting, often uncontrollable; severe abdominal pain; diarrhœa, frequently profuse; and tympanites; together with marked nervous phenomena, as vertigo, spasms, shock, and in grave cases general convulsions and coma.

Intestinal Obstruction.—This condition, either partial or complete, is very commonly attended by symptoms of autointoxication, namely, headache, fever, sleeplessness, and albuminuria, with increase in the aromatic substances in the urine; all of which subside when the obstruction is relieved.

Constipation.—Many symptoms are attributed to this condition, but there are few that are constant and none that is characteristic. A furred tongue, poor appetite, headache, lassitude, and mental depression may occur in habitual constipation, but these symptoms are common in those who have a regular daily action of the bowels. There are those who are miserable if the daily morning movement is missed, while others are uncomfortable if by drugs or injections their bowels are moved more frequently than once in the course of some days. *Copræmia*, a hypothetical intestinal autointoxication from constipation, lacks the support of accurate clinical observation and objective chemical investigation. The work of Horace Fletcher has shown that constipation amounting to the evacuation of small masses of dry fæces at intervals of several days is not incompatible with excellent health. The secondary mechanical effect of the accumulation of fecal matter in the intestines is considered under its appropriate heading.

Gastric Neurasthenic and Other Conditions Vaguely Described as Nervous Dyspepsia.—This group of nervous affections is sometimes attributed, upon wholly insufficient evidence, to autointoxication, and the same statement may be made in regard to a number of nervous diseases, as migraine, neuritis, and epilepsy, and some of the psychoses, as melancholia and forms of dementia.

The Anæmias.—The theory of Sir Andrew Clark in regard to fecal poisoning as the cause of chlorosis rests upon an insufficient basis of fact and is no longer accepted. That pernicious anæmia is probably due to an autointoxication of intestinal origin finds support in the following facts, namely: that a persistent hæmolysis is the essential pathological process in the disease; that the hæmolytic process is active in the portal system; and that there are, in many of the cases, atrophic changes in the gastro-intestinal mucosa. The nature of the toxic agent has not been demonstrated.

II. **The Retention Intoxications.**—Biliary intoxication is due to the biliary salts and the pigments. The toxic influence is exerted upon the cells of the parenchymatous organs, the muscles, and the blood. Many persons suffer from marked jaundice for considerable periods of time without manifesting evidences of intoxication. Hepatic coma cannot, in the strict sense, be ascribed to cholæmia, since it occurs in cirrhosis of the liver, in which jaundice is a subordinate symptom or absent altogether. It is probably due to derangement of the hepatic functions in metabolism.

III. **Autointoxication from Extensive Abolition of the Function of the Skin.**—Extensive superficial burns are followed by rapidly oncoming collapse, associated with acute degenerative changes in the cells of the parenchymatous organs and muscles, and hæmolysis—evidences of the action of toxic agents, the nature of which is unknown.

IV. **Acidosis.**—Under this term are grouped the derangements of metabolism which result from an excess of acids in catabolism—an acid intoxication. The principal sources are: (a) the acids of carbohydrate fermentation in the alimentary canal; (b) the sulphuric and phosphoric acids derived from the catabolism of common protein and nuclein respectively; (c) lactic acid; (d) the members of the acetone group, diacetic and β-oxybutyric acids derived from the fats (A. E. Taylor). This form of autointoxication is encountered in diabetes, starvation, phosphorus poisoning, toxæmia of pregnancy, cyclic vomiting of children, in severe febrile infections, after prolonged chloroform anæsthesia, in the cachexia of carcinoma, and many other diseases. The condition may be caused by the withdrawal of fixed alkalies or the toxic action of salts of the acid compounds. James Ewing has shown "that the chemistry and pathological anatomy of these diseases lends support to the view that there are two distinct classes of acidosis following two experimental prototypes.

"*Type I.*—Hydrochloric Acid Poisoning.—Clinical forms: diabetes; starvation. Chemistry: acetone compounds. Pathological anatomy: no lesions.

"*Type II.*—Extirpation of the Liver or Eck Fistula.—Clinical forms: phosphorus poisoning; toxæmia of pregnancy; cyclic vomiting; chloroform poisoning. Chemistry: lactic acid prominent; ammonia in excess of any acetone compounds present. Pathological anatomy: extensive fatty degeneration" (Ewing).

Notwithstanding the fact that considerable quantities of acids may be present in the blood in combination, an acid reaction of the blood-serum does not occur. It could only be a terminal phenomenon.

V. **Gout** in the present state of knowledge may be regarded as an autointoxication dependent upon derangements of the purin metabolism: VI. **glycosuria** and **diabetes**, as autointoxications arising in consequence of faults in the carbohydrate metabolism.

VI.

THE DIAGNOSIS OF HEAT-STROKE AND ELECTRIC STROKE.

HEAT-STROKE.

Heat-stroke is commonly seen in laboring men, and is also not unusual in armies. In the United States Army, from 1868 to 1893, there were not less than 1250 cases, with 47 deaths.

Pathology.—Congestion of the brain and membranes, as well as of the lungs, is common. According to Gihon[1] loss of coagulability of the blood is the one great lesion in *coup de soleil.* Rigor mortis and putrefactive changes occur early. The post-mortem appearances are mostly negative, but there is rigid contraction of the left ventricle of the heart, while the right side and the great vessels contain partly coagulated dark blood. Meningitis is one of the sequels of sun-stroke. The vitochemical changes in the blood, muscles, and nerve-centres are not fully understood.

Symptoms.—Two forms are recognized: simple heat exhaustion, and heat-stroke proper.

In *heat exhaustion* the patient usually collapses, and may even fall in a partial or complete syncope. The surface of the skin is cool, the pulse rapid and feeble, and the temperature may even be subnormal—as low as 95° or 96°. In the worst cases there is sometimes mental confusion, and delirium has been occasionally reported. The prognosis in these cases is usually good, if the patient's general health is sound.

In *heat-stroke proper* the chief symptoms are as follows: headache, oppression in the epigastrium, sometimes nausea and vomiting, a sense of weakness, vertigo, dimness of vision, and unconsciousness, with fever and rapid pulse. Coplin, among the sugar refiners of Philadelphia, also describes a "cramp" in the epigastrium, and sometimes in the back and the calves of the legs, as among the premonitory symptoms. Of the various symptoms the only one that can be called pathognomonic is the exceedingly high temperature. Richards, in the Rhode Island Hospital, observed temperatures ranging as high as 110°, and Packard, in 31 cases in the Pennsylvania Hospital, saw the temperatures range up to 110°, 111°, and even 112°. These are extreme cases, usually with contracted pupils and profound unconsciousness, and many of these patients die. Death sometimes occurs so quickly that a special or *apoplectic* type is recognized, and if dyspnœa is prominent, the type is called *asphyxial.* Most authors are in accord about the contracted pupils, but an exceptional case of dilated pupils has been noted, and as death approaches the pupils may dilate. Convulsions are not common. Great oppression of breathing is sometimes experienced, with a sense of constriction of the chest. Pirrie, in his cases in Central India, observed priapism and seminal emission just before the seizure. Alcoholism is an active promoter of heat-stroke. Most cases occur in persons who have been over-exerting themselves.

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[1] "Heat-stroke," in XX. Cent. Pract., vol. iii., p. 253. This article by Gihon is a useful review of the whole subject, both historical and clinical.

Diagnosis.—Heat-stroke must be distinguished from cerebral hemorrhage, uræmia, alcoholic intoxication, and opium poisoning; but from all these conditions it differs in its history and its high temperature. The history alone is so clear and suggestive in most cases that a mistake is hardly possible. In uræmia a subnormal temperature is common, and the condition of the urine is characteristic; if the temperature rises, as it does in some cases, especially towards the end, it does not mount as high as in sun-stroke. The contracted pupils in heat-stroke may suggest opium poisoning, but in the latter there is slow respiration, and in the former high temperature. In mere alcoholic intoxication we do not see pyrexia, much less hyperpyrexia, nor contracted and immobile pupils. In fact, in all comatose conditions, as in those just named, and in diabetic coma, we do not see high fever, nor is there the history of exposure to heat. Injury to the head can usually be excluded by the history and by careful physical examination. In cerebral hemorrhage there is usually hemiplegia, which can be recognized as a rule by the difference in resistance on the two sides. The temperature often rises as death approaches. The attempt to distinguish sun-stroke from heat-stroke is not called for.

ELECTRIC STROKE.

Under this heading are included both lightning stroke and shocks from dynamos. The vast extension of the use of electricity in recent years has made these accidents not uncommon, and the use of the current as an agent for executing criminals in the State of New York has furnished rare opportunities for the systematic study of the subject.

Pathology.—Some of the lesions are purely surgical, such as the burns which are caused by immediate contact with a "live" wire. It is impossible to state in scientific terms what is the exact pathology of electric shock, especially in cases of sudden death. The results of examinations are often negative. Van Gieson, in autopsies on the bodies of criminals, found fluid blood, but no recognizable changes in the tissues or organs.

Symptoms.—Macdonald and Ward have recorded the effects as noted in the execution of four criminals at Sing Sing, N. Y.[1] With a current of 1785 volts, passed through wet sponge electrodes from the forehead to the calf of one leg, the heart continued to beat after the first contact of 27 seconds, and a noisy respiration was re-established after an interval of more than one minute. After a second contact of 26¼ seconds respiration and the heart action had ceased permanently. It seems that the action of the heart is not permanently arrested as quickly as is respiration. With a stronger voltage, as in a lightning stroke, it is possible that the respiration and the heart are arrested instantaneously.

E. A. Spitzka, whose observations are based upon thirty-one electrocutions, finds that "the death is undoubtedly painless and instantaneous. The vital mechanisms of life, *circulation* and *respiration*, cease with the first contact. Consciousness is blotted out instantly and the prolonged application of the current as it is usually practised by Mr. E. F. Davis, the State electrician of New York, ensures the permanent derangement of the vital functions so that there could be no recovery of these. Occasionally, the drying of the sponges through undue generation of heat causes desquamation or superficial blistering of the skin at the site of the electrodes, but not often. Post-mortem discoloration, or lividity, often appears during the first contact. The pupils of the eyes dilate instantly and remain dilated in death."

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[1] Medico-Legal Journal, vol. ix. Also XX. Cent. Pract., vol. iii. pp. 403–411.

Diagnosis.—This must depend largely, if not entirely, upon the history. Burns on the surface of the body, as already said, are common from contact with a "live" wire, but in the case of lightning stroke, while not unseen, they do not appear to be so extensive or so common.

The after-effects in non-fatal cases usually consist in states of neurasthenia and traumatic hysteria. Organic palsies, or permanent lesions of any kind, seem to be rare sequels.

VII.

THE DIAGNOSIS OF PREGNANCY.

Under ordinary circumstances the question as to the existence of pregnancy, as it presents itself to the general practitioner or to the specialist in midwifery or in gynæcology, presents no difficulty. As a rule, the patient has already made the diagnosis for herself. As it occurs, however, to the medical diagnostician, it frequently assumes a high degree of importance and involves responsibility of the gravest kind. This is especially the case in illegitimacy in young girls, who very often stoutly deny exposure to the possibility of such a condition until the approach of actual labor. On the other hand, married women long childless sometimes positively assert that their hopes are about to be realized, and enumerate in detail and with precision the signs of the condition even to the motion of the child, under circumstances in which the occurrence of such an event is impossible—*pseudocyesis*. There are obvious reasons why the opinion of the medical man is often first sought.

Early Gestation.—**Symptoms.**—Amenorrhœa in a healthy woman previously regular is a symptom of primary importance, and usually the first to arouse a suspicion on the part of the woman as to her condition. Cessation of the menses, under these circumstances, is physiological and unattended by the signs of the grave chronic diseases, as nephritis, tuberculosis, and the cachexias, in which it is pathological. Abrupt cessation at a later period of life may also be physiological and mark the occurrence of the grand climacteric. The occasional occurrence of this physiological event at an unusually early period of life is to be borne in mind. On the other hand, the recurrence of a menstrual flow for two or three months, or in extremely rare instances throughout pregnancy, must be considered in a doubtful case. Nausea is next in importance. It begins, as a rule, from two to four weeks after amenorrhœa and may or may not be associated with vomiting. It is troublesome and distressing at the hour of rising, but the gravida may be annoyed by waves of nausea from time to time during the day. Cravings for unusual articles of food, and hysterical manifestations, may accompany the nausea. Constipation is common. Further subjective manifestations are irritability of the bladder, increased flow of saliva, and sensations of fulness and tingling in the breasts.

Signs.—Certain signs usually show themselves during the first two months, and have a diagnostic value. Those relating to the breasts comprise general enlargement, a nodular fulness in the glandular area, slight prominence of the nipple, pigmentation of the areola, and enlargement of the sebaceous glands surrounding it. Pressure of the contents of the ducts outward toward the nipple reveals the presence of a yellowish watery fluid—*colostrum*. In multigravida a persistent secretion may follow lactation and simulate colostrum. Signs apparent upon examination of the abdomen are pigmentation of the linea alba, flattening of the hypogastrium, and retraction of the umbilicus. In fair women pigmentation may not occur, and in fat women flattening and retraction are not marked.

Pelvic signs are more distinctive. Of these the more important are a violaceous coloring of the cervical portion of the uterus, thinning and compressibility of the junction of the cervix with the body,—*Hegar's sign*,—lateral expansion of the fundus in anteflexion, and pulsation of the uterine arteries. The body of the uterus presents a peculiar softness. The presence in the os of a plug of tenacious mucus is, in connection with the foregoing changes, very suggestive.

ADVANCED PREGNANCY.—A tendency to constipation persists and waves of nausea occur. Striation of the breasts, abdominal walls, and upper parts of the thighs becomes conspicuous. There are elevation of the fundus uteri and protrusion of the navel. Recurrent rhythmical contractions of the uterus after the completion of the third month, unattended with pain—*Braxton Hicks's sign*—are highly suggestive of pregnancy. Finally, the rise and fall of the fœtus in ballottement, the movements of its limbs, and the sounds of its heart are positive and conclusive signs.

Diagnosis.—The DIRECT DIAGNOSIS rests upon the presence of the above symptoms and signs and becomes probable in proportion to the number of them in association at the time of the examination. It assumes greater certainty as pregnancy advances, and demonstrable signs, which offer more positive indications, become associated with the symptoms.

DIFFERENTIAL DIAGNOSIS.—*The Distinction between Normal Pregnancy and Conditions which Simulate it.*—As to special symptoms, the amenorrhœa of pregnancy may usually be distinguished from pathological amenorrhœa by the anamnesis. The absence of a history of disease, such as malaria, nephritis, an acute or chronic infection, is important. But the fact that women subject to such diseases may become pregnant is not to be overlooked. Amenorrhœa due to local pathological conditions—hæmatometra, pyometra, and hydrometra—is attended by uterine colic, and the enlargement of the organ does not present the softening of the pregnant uterus. On the contrary, it offers a tense, fluctuating tumor. The amenorrhœa of acquired atresia may be readily recognized by the history and local conditions. The amenorrhœa of the menopause is rarely abrupt, mostly partial and progressive, and frequently accompanied by hysterical manifestations. Accumulations of omental fat or abdominal-wall fat are common. Moreover, the uterus is not enlarged or softened, and the other local signs of pregnancy recognizable upon vaginal examination are wholly absent.

Intra-abdominal tumors arising from the level of the pelvis are to be distinguished from the gravid uterus by their consistency, as in the case of fibromyomata, where the enlargement is tense, nodular, and of slow growth. The impairment of the patient's health, and the attacks of pelveoperitonitis occurring with more or less regularity, together with the metrorrhagia, are conclusively opposed to the diagnosis of pregnancy. Tumors of the adnexa, and pelvic exudates, reveal their character both by the local symptoms and by the situation in the pelvis of the swellings to which they give rise.

Subjective manifestations of pregnancy which simulate functional disturbances arising in disease, such as nausea, and albuminuria, are to be weighed relative to their association with other symptoms and with the signs of pregnancy. The presence or absence of casts, the amount of urine excreted, and the history of the case render it possible to differentiate the albuminuria of pregnancy from that occurring in nephritis.

Abnormal Pregnancy and Simulative Conditions.—Abnormal pregnancy comprises, first, the development of the ovum in abnormal situations; second, pregnancy following the fertilization of the ovum *in loco* but pursuing an abnormal course.

As to the points of difference in the diagnosis of tubal gestation and cornual pregnancy from pelvic tumors simulating either of these conditions, the irregular uterine bleeding, the pain, the collapse occurring with rupture or tubal abortion, and the symptoms of internal bleeding, together with the localization of a mass outside the uterus—all in the presence of symptoms of early pregnancy—are suggestive.

Certain symptoms, which by their occurrence characterize the course of pregnancy as abnormal, may give rise to confusion. The continuance of menstruation, for instance, may obscure the diagnosis. The syncope which occurs in pregnancy may likewise require the observation of associated symptoms to explain its presence. The œdema of pregnancy shows itself in the presence of signs which distinguish it from the œdema of cardiac disease and the anasarca of nephritis; it is usually progressive without symptoms of nephritis, is not accompanied by pallor or waxiness of the skin, and is evidently connected with the pressure of the uterus.

Finally, hydramnios may suggest the presence of a tumor within the abdomen originating from some condition other than pregnancy. Hydramnios, however, may be diagnosticated by the presence of fluctuation, the rapid increase in the size of the tumor, the location of the latter within the abdomen, and the associated symptoms and signs of pregnancy.

SERODIAGNOSIS OF PREGNANCY—IMPROVED METHOD OF ABDERHALDEN.—This laboratory procedure depends upon the principle that the blood-serum of pregnant individuals contains a specific ferment which digests placental cells.[1]

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[1] Consult Müller's "Serodiagnostic Methods," Translated by Ross C. Whitman, p. 68, *et seq.*, J. B. Lippincott Company.

VIII.

THE DIAGNOSIS OF CONSTITUTIONAL DISEASES.

I. GOUT.

Podagra.

Definition.—A disease of disordered metabolism, due to the presence in the blood of uric acid in abnormal amounts, and characterized clinically by attacks of acute arthritis involving one or several joints and recurring at irregular intervals, the deposition of sodium biurate in and around the joints, and irregular constitutional symptoms.

Etiology.—It is necessary to consider the gouty constitution and the attack. An individual may present marked evidences of the gouty diathesis and never experience an attack; or there may, fortunately, be long intervals between the attacks, during which the health is excellent. As the disease advances the attacks become more frequent and the general health is progressively impaired.

Predisposing Influences. — *Heredity.* — The gouty constitution is inherited more frequently than it is acquired. Transmission appears to be more common in the male line, but in this connection the different mode of life of the sexes is to be considered. In hereditary cases the attack may occur in childhood. It is not common before thirty, but in a majority of the cases the first attack occurs in early middle life. Alcoholic excesses, and especially the habitual free use of alcoholic beverages—*regular drinking*—is an important causative factor. Malt liquors tend to cause gout more than wines, and these more than distilled spirits. *Excesses in food* are more important still. Eating too much, without active exercise, is a predisposing cause of the highest importance. Gouty persons are often hard workers, with excellent appetites and good gastric digestion. Moreover, rich viands and fine wines are associated in the pleasures of the table. But gout is not restricted to the rich; exhausting toil, poor food, and excesses in beer may cause it—"*poor man's gout.*" *Chronic lead poisoning* plays an important part in the etiology of this disease. *Sedentary Habits.*—Lack of fresh air and sunshine, with prolonged mental effort, predisposes to the attack. Sydenham's "Whenever I return to my studies my gout returns to me" is well known.

Exciting Causes.—When the attack is due, it may be brought on by apparently trifling causes—an unusually hearty meal, a glass of champagne, sudden chilling, worry, or sudden depressing emotion, or mental shock. It may also be precipitated by a slight injury or accident.

Pathology.—The nature of gout, beyond that it is due to faulty metabolism with overproduction and deficient elimination of uric acid, is unknown. The hypotheses regarding the actual part played by uric acid, which is now regarded as one of the purin bodies derived from the nuclein resulting from nuclear disintegration, in causing gout are very numerous. None of them, however, arises to the dignity of a theory.

Symptoms.—Gout may be acute, chronic, and irregular.

1. ACUTE GOUT.—The attack is commonly preceded by prodromes, which consist of fleeting pains in the small joints of the hands or feet, restlessness, irritability, and dyspepsia with acid eructations. In some cases there is an erythematous angina, bronchitis, or asthmatic symptoms. The elimination of uric acid has been found to be diminished before and during the early part of the attack. In many instances there are no premonitory symptoms. The attack begins, as a rule, in the early morning hours. There is agonizing pain in the metatarsophalangeal joint of the great toe, more frequently the right, or the tarsometatarsal joints, especially at the outer border of the foot. There is intra- and periarticular effusion; the skin is hot, red, tense, and glistening. Tenderness is extreme and the patient cannot endure the slightest pressure upon the affected part. There is moderate fever, the temperature rising to 102°-103° F. (39°-39.5° C.) and falling to normal toward the end of the attack by lysis. The intensity of the symptoms somewhat abates in the morning, but the foot remains swollen, red, and painful, and during the night the patient's sufferings are again intensified. Other joints, and particularly the great toe of the opposite foot, may become affected. The nocturnal exacerbations gradually subside and, in the course of a few days or a week, the acute symptoms disappear with itching and desquamation, but the affected joint remains tender and swollen, often requiring the use of a loose shoe and crutches for another week or more. Suppuration in the affected joints does not occur. Transient albuminuria or glycosuria may occur. There is a moderate leucocytosis during the acute symptoms. Physical and mental depression persist for a period, but after a time the patient regains his former health. Recurrences follow at intervals varying from a few months to a year or more. In the later attacks not only the joints of the feet, but also the knees, wrists, and fingers may be involved.

Retrocedent or Suppressed Gout; Visceral Gout.—These terms are used to designate groups of symptoms, usually grave, indicative of disease of internal organs, which sometimes arise coincidently with a sudden subsidence of the local manifestations of the attack. The principal groups are, (a) gastro-intestinal—pain, vomiting, purging, and collapse, so severe in some instances as to prove fatal; (b) cardiac—dyspnœa, precordial pain and distress, arrhythmia, and tachycardia; and (c) delirium, stupor, coma, or apoplectiform attacks. These symptoms are in many of the cases uræmic.

CHRONIC GOUT.—The attacks become more frequent and prolonged, and many joints are affected. In debilitated persons and those suffering from chronic lead poisoning, the attacks of arthritis may not occur—*atypical gout.* Deposits of sodium biurate—*tophi*—occur in the cartilages and ligamentous structures of the joints, which, in the course of time, become enlarged and deformed. The feet are first and most markedly affected, then the hands to a less extent, and in some cases tophaceous deposits form about the knees and elbows or in the line of the tendons. The cartilage of the ear frequently contains tophi which appear as yellowish-white nodules at the edge of the helix. Less frequently similar deposits take place in the cartilages of the nose, eyelids, and larynx. The skin covering the tophi frequently undergoes slow necrotic change, with ulceration exposing the felted chalk-stones. As the disease advances the patients become sallow and dyspeptic; the signs of arteriosclerosis develop; there are cardiac hypertrophy, increased arterial tension, increased urine with low specific gravity, slight albuminuria which may be intermittent, and hyaline casts. Muscular cramps, especially affecting the calves of the legs and starting in the predormitium, are common. Attacks of arthritis, implicating one or several joints, characterized by pain, redness, and swelling, occur with or without fever. Croupous pneumonia and apoplexy are common terminal events, but death often results from uræmia or from an acute inflammation of one of the great serous sacs.

IRREGULAR GOUT.—In addition to the attacks of arthritis and the chalk-stones, there are diverse morbid conditions to which gouty subjects and members of gouty families are alike especially liable, and which are commonly regarded as irregular manifestations of gout. Among the more important of these are cutaneous eruptions, and especially eczema; periodical gastro-intestinal catarrh—so-called bilious attacks; pulmonary affections, especially bronchitis and emphysema; cardiovascular lesions, myocardial degenerations, arteriosclerosis, and aneurism and nervous affections, among which migraine, headache, sciatica, and other neuralgias are the more common. Burning sensations and itching of the feet at night are very common and annoying symptoms. Among affections of the eye, scleritis, iritis, cataract, glaucoma, and hemorrhagic retinitis have been attributed to gout. It is the custom to regard gout as the cause of the recession of the gums so common in advancing life.

The urinary conditions are more especially gouty because they are based upon anatomical lesions of the kidneys. Chronic interstitial nephritis, without characteristic changes, is not uncommon in gouty subjects. More common are conditions believed to be distinctive, namely, a deposit of urates in the intertubular tissue, mostly in the papillæ; less frequently the deposits occupy both the tissue and the tubules, and there are also minute foci of necrosis in the cortex and medulla, in which are deposited crystals of sodium urate. The clinical manifestations of gouty deposits are the same as those of this form of chronic nephritis occurring in non-gouty persons: increased output of urine, albuminuria (usually slight), low specific gravity, hyaline casts, increased arterial tension, accentuated aortic second sound, and uræmic symptoms. Prior to the development of renal changes the urine is often very acid and high-colored and may, upon standing, deposit crystals of uric acid. In chronic gout the uric acid is diminished, as a rule, with occasional excess. Intermittent glycosuria, traces of albumin, and tube casts may occur. Oxaluria is common. Renal calculi are not infrequent. The association of vesical calculi and gout is often observed. I have several times seen purulent urethritis follow an attack of gout.

Diagnosis.—THE DIRECT DIAGNOSIS OF ACUTE GOUT is usually a simple matter. Recurrent attacks of arthritis, beginning in or limited to the great toe, with a hereditary history of gout and a personal history of over-indulgence in food and drink, are of positive diagnostic value. When other joints are implicated and there is fever, and a satisfactory family and personal history cannot be obtained, there may be a question as to the differential diagnosis between acute gout and rheumatic fever. The following facts are in favor of the diagnosis of gout: the occupation and habits of the patient; the involvement of a limited number of larger joints; the persistence of the arthritis in the affected joint in contrast to the migratory character of rheumatic arthritis; the appearance of the inflamed joints, which are commonly tense, deeply red or violaceous, and shiny; the suddenness of onset; and the condition of the urine, which shows a low uric acid output in the beginning of the attack, with marked increase toward its close.

THE DIAGNOSIS OF CHRONIC GOUT.—The history, the presence of tophi, and the deformities are characteristic. A tophus in the neighborhood of a joint is easily recognized. Tophi upon the ears appear earlier and are positively diagnostic. Other things at the ear margin may be mistaken for them: the helical apex, called from the English sculptor and poet Woolner's tip; small sebaceous tumors; and fibroid nodules. The last are very rare. In the felted material from an open tophus the needle-shaped crystals of sodium biurate are characteristic. Garrod's uric acid thread test may be tried. In a watch glass, 15 c.c. of blood-serum, which may be obtained by blistering, are treated with 0.25 of acetic acid. A fine thread immersed in it may show in a few hours crystals of uric acid. The result is often negative in cases in which there is no question as to the clinical diagnosis. An excess of uric acid in the circulating blood occurs also in leukæmia and chlorosis.

THE DIFFERENTIAL DIAGNOSIS between chronic gout and arthritis deformans demands some words of consideration. This necessity arises more from the misleading influence of the term "rheumatic gout" than from any real resemblance between the diseases. In the rare cases in which the deformities of chronic gout arise insidiously, in the absence of acute attacks, the following points are important: Arthritis deformans is common in women and among the poor and poorly nourished; spontaneous pain in the affected joints is less common and less urgent; the deformities, especially those of the hands, are more uniform and symmetrical; and tophaceous deposits form no part of the pathology of the disease.

THE DIAGNOSIS OF IRREGULAR GOUT is based upon the family and personal history of the patient, and the general experience of clinicians that gouty individuals more frequently manifest these particular derangements of health than others. The diagnosis of visceral gout depends, likewise, upon the anamnesis and the fact that the onset of the symptoms, indicating disease of a particular organ, corresponds in time to the subsidence or disappearance of the familiar symptoms of the acute attack of gout. The diagnosis of irregular, retrocedent, and visceral gout should be made with some reserve and only in the case of a distinct hereditary predisposition, or of an individual who has had acute attacks, or who shows tophi or characteristic deformities, and in whom, in the absence of such signs, other etiological factors can be excluded.

II. ARTHRITIS DEFORMANS.

Definition.—A chronic disease of the joints, of undetermined causation, characterized anatomically by lesions of the synovial membrane with hypertrophy of its fringes, atrophic changes in the cartilages and bones, irregular hypertrophy of the bones, and wasting in the periarticular structures; and clinically by characteristic deformities.

Etiology.—Predisposing Influences.—The synonyms *rheumatic gout* and *rheumatoid arthritis* indicate the prevalent belief that arthritis deformans has some relationship to those affections—a belief that finds little support in fact. In less than one-third of the cases the family history shows a tendency to joint disease—gout or rheumatism. Arthritis deformans in successive generations is unusual. Two or more cases have been noted in a family. Children are sometimes affected; young girls frequently; the greater number of cases begin in early adult and middle life. Women are affected more frequently than men. The disease very often first shows itself about the time of the menopause, and is more common among women who suffer from diseases of the reproductive organs. Habitual exposure to cold and damp, hardship and privation, sudden mental shock, and depressing emotions, appear to bear a causal relation to the disease.

Exciting Cause.—There are two theories: first, that the joint affection is secondary to some disease of the nervous system; second, that it is a chronic infection. The latter is now generally accepted. A variety of micro-organisms have been found in the lesions, but none that is uniformly present. The fact that, in a considerable proportion of the cases, there is a history of gonorrhœa, does not, in view of the wide prevalence of that disease, indicate a causal relation on the part of the gonococcus.

Morbid Anatomy.—All the tissues which enter into the structures of the affected joints are involved in the morbid process, but in which of them the process starts cannot be affirmed with certainty. It is probable, however, that the cartilages are first affected. The lesions consist of fibrillation and atrophy of the cartilages; in the bones, eburnation, abrasion, and osteophyte formation; in the synovial membranes, thickening and hypertrophy of the fringes and atrophy of the periarticular tissues. Osteophytes developing at the margins of the bones may interfere with movement. Bony ankylosis is rare in the joints of the extremities, but common in the spine, which sometimes becomes rigid and immovable. A late condition is contracture, with fixation of the joints in flexion. On the other hand, the muscles are atrophied and the ligaments relaxed, so that subluxation is common, especially in the knees and fingers. The hands are greatly deformed and the fingers, under the influence of gravity, show deflection to the ulnar side. Some of the joints are the seat of an effusion. Neuritis occasionally occurs. The most striking feature of the arthropathy is its symmetry. The changes in the cartilages and bones are well shown in radiographs.

Clinical Varieties.—Heberden's nodes; the progressive polyarticular form; the monarticular form; the vertebral form; and arthritis deformans in children.

(a) Heberden's Nodes.—"Tumors attaining to the size of a pea, which are sometimes developed near the third joints of the fingers. They have certainly nothing in common with arthritis (gout), since they are met with in many persons to whom that disease is unknown. They remain throughout life, are devoid of all pain, and show no tendency to ulceration. The deformity is more conspicuous than the inconvenience they cause, though the movement of the fingers is somewhat impeded by them." They occur much more commonly in women than in men, and about middle life. The nodules may become tender and red. Tophi do not appear. The larger joints are not involved. They are not influenced by treatment.

(b) The Progressive Polyarticular Form.—The *acute variety* in the initial attack bears a close resemblance to rheumatic fever, for which it is very often mistaken. It occurs frequently in young women among the working classes, and especially in mill girls. It is common after child-bearing and during lactation. It begins in some of the cases at the menopause. The joints become swollen, tender, and painful upon movement; there is fever and the patients become anæmic and weak, and rapidly lose flesh. The attack passes over, leaving the affected joints slightly deformed. From time to time similar attacks recur, each leaving, as it subsides, some increase of deformity and further impairment of health, until at length the patient becomes completely broken down and crippled. The small joints of the hands and feet first and chiefly suffer, but the ankles, knees, wrists, elbows, shoulders, and spine frequently become involved. *Chronic Form.*—This variety is most common. Acute attacks may occur. Only one or two joints may be at first affected; usually the hands are first involved, then the knees or feet. Gradually new joints suffer until, in the severe cases, scarcely an articulation escapes. As the disease progresses the joints are symmetrically involved. The earliest symptoms are pain on movement and slight swelling, which may be intra- or periarticular. The pain varies greatly in intensity. There are cases in which a high degree of deformity gradually comes to pass, without pain; others in which there is pain only at the time of outbreaks of the arthritis, or at night; and a few in which pain is intense and persistent.

The deformities are progressive. The joints are enlarged in part by the outgrowth of osteophytes, in part by thickening of the capsular ligaments, and in part by subluxation. While they remain movable, crepitation may be felt. The periarticular tissues, and especially the muscles, undergo atrophy, and at length the function of the joints is wholly lost. Osteophytes, adhesions, and infiltration of the tissues prevent movement, and the joints become fixed, usually in strong flexion, so that in extreme cases the patient lies completely helpless, unable to move any part of the body except the eyes. Trophic changes, atrophy of the muscles, glossy skin, pigmentation, and onychia occur, and numbness and tingling are common. In many of the severe cases the joints of the hands and wrists suffer to a less extent, and the ability to sew or write is in part retained. A considerable proportion of the cripples caused by this form of arthritis deformans maintain fair general health and a cheerful disposition.

(c) The Monarticular Form.—This form is usually seen in elderly persons. The knee, hip, and shoulder are commonly involved. They very often follow traumatism. In other cases they develop insidiously. Exposure to damp cold appears to exert a predisposing influence, and aged fishermen, oystermen, and hunters often suffer. The joint lesions are the same as in the polyarticular forms; the muscles rapidly waste, adhesions limit the movements of the parts, and motion is extremely painful.

(d) THE VERTEBRAL FORM.—Two varieties are recognized. In one, the spine alone is involved. The disease begins with obscure meningeal symptoms, with evidences of compression of nerve-roots, pain, anæsthesia, loss of function, and wasting of spinal muscles, atrophy of the disks, and progressive ankylosis of the vertebræ (Von Bechterew). In the second variety, the hips and shoulders are also ankylosed—spondylose rhizomélique—(Strumpell-Marie). Spondylitis deformans is more common in males than in females. It may result from spinal injury. The early manifestations are sometimes confined to the cervical or to the lumbar region. In other cases the entire spine gradually becomes rigid and immobile. There may be marked kyphosis, with a rigid and immobile thorax and with diaphragmatic breathing.

(e) ARTHRITIS DEFORMANS IN CHILDREN.—The disease occurs in early childhood. Girls are more commonly affected than boys. The disease may present the same features as in adult life. In some cases direct inheritance has been noted. Cold, privation, and unsanitary surroundings are predisposing influences. The onset may be acute, with the symptoms of infection, fever, profuse sweating, enlargement of the spleen and superficial lymph-glands—*Still's disease.* One or two joints may be at first affected, and others later. There is loss of function together with muscular atrophy. The prognosis is more favorable than in adult life.

Diagnosis. — The DIRECT DIAGNOSIS depends upon the subacute exacerbations of the joint affection, the fact that after each outbreak the deformity is increased, the remarkable symmetry of the lesions, the persistence of the process in the affected joints, the relaxations of the ligaments, the atrophy of the muscles, and the progressive character of the disease. The initial attack often presents a remarkable resemblance to subacute rheumatic fever.

DIFFERENTIAL DIAGNOSIS.—From rheumatic fever the diagnosis may be made by the permanence of the arthritis in particular joints, the persistence of the lesions, the extreme infrequency of endo- or pericarditis, and the incurability of the affection; and from gout by the history, the fact that gout is mostly a disease of men and middle life, and the absence of tophi. The monarticular forms are usually regarded as cases of chronic rheumatism.

III. THE RHEUMATOID AFFECTIONS.

Most of the cases of so-called chronic rheumatism are forms of arthritis deformans, especially those in which a single joint is involved. A majority of the cases of so-called muscular rheumatism are purely myalgic. Nevertheless, out of deference to an almost universal custom, chronic rheumatism and muscular rheumatism are permitted to retain their nosological position. It is important to note the entire absence of etiological and clinical relationship to rheumatic fever.

A. Chronic Rheumatism.

Definition.—A chronic joint affection of elderly persons, characterized anatomically by synovial inflammation, capsular thickening, and wasting of the periarticular tissues and the related muscles, and clinically by stiffness, pain, and impairment of motion.

Etiology.—Predisposing influences are advanced age, occupations which expose the individual to cold and damp—as in the case of washerwomen, ditch-diggers, and fishermen—poverty, and hardship. The exciting cause is sometimes an injury, which may be slight. In many cases the disease develops insidiously.

Morbid Anatomy.—The synovial inflammation is unattended by effusion. There is thickening of the ligaments, especially of the capsular ligament and the sheaths of the tendons. Erosion of the cartilages may be found. There is marked atrophy of the muscles related to the affected joint. Subluxation and other deformities are usually not marked until late in the course of the disease.

Symptoms.—Pain, which is more marked in the morning and when the weather changes; tenderness, which subsides after gentle massage; stiffness, which is usually relieved to some extent by exercise, are the chief symptoms. Swelling is commonly present, but not redness. A single joint is often affected, as a knee, hip, or shoulder; in many cases several joints are involved, more commonly the large than the small joints. Ankylosis and more or less deformity may gradually come to pass. The subjects are often broken down and anæmic. The prognosis as regards cure is not hopeful.

B. Myalgia.

Muscular Rheumatism.

Definition.—An affection of the voluntary muscles and their fibrous structures, of undetermined pathology, characterized by pain upon movement and pressure. The disease is local and is designated by various names, as lumbago, torticollis, pleurodynia, according to the parts involved.

Etiology.—Predisposing Influences.—The rheumatic and gouty habit of body, laborious occupations, and those involving exposure to cold and damp are important factors. Men suffer more frequently than women. It is an affection of middle and late life.

Exciting Causes.—The attacks follow cold and exposure, especially when heated. A draught of air may bring it on. Overuse of a group of muscles is a frequent cause. The muscular pains and soreness after a first horseback ride are myalgic.

Whether the pain and tenderness arise from some nutritional disturbance of the muscle substance acting upon the sensory nerves of the muscles, or these symptoms are due to a neuralgia of such nerves, has not been positively settled. Myalgia is usually acute; it may be subacute and is sometimes chronic.

Symptoms.—The disease is local. Constitutional derangements are rare and due to pre-existing or accidental conditions. They consist of loss of appetite, languor, and slight rise of temperature. Pain is the chief symptom. It is rarely constant, but is acute, even agonizing. when the affected muscles are contracted. It is sharp and cramp-like upon movement, but dull and sore, or absent altogether, when the muscles are in repose. Firm pressure causes soreness, as may be seen in lumbago—a sign of diagnostic value.

According to the seat of the affection the following principal varieties are described: LUMBAGO.—The erector muscles of the spine and their attachments are affected. There is pain upon rising and turning. The patient can lean over to lace his shoes, but cannot straighten his back without pain. There is marked tenderness upon firm pressure over the affected muscles and their attachments. The attack is of sudden onset and often completely disabling. TORTICOLLIS—WRY-NECK.—The sternocleidomastoid and adjacent muscles are affected. In some instances the posterior cervical muscles are also involved. This form is very common. Young persons frequently suffer. The head is held rigidly and cannot be rotated from side to side. PLEURODYNIA.—The intercostal muscles and, in some cases, other chest muscles are painful and tender upon pressure. The left side is more commonly affected than the right. A deep breath, coughing, sneezing, even laughter may cause the patient to cry out with pain. It is to be distinguished from pleurisy by the absence of friction râles and from intercostal neuralgia by the absence of painful points along the course of the nerves, and the fact that in the latter affection the pain is more paroxysmal. The epigastric pain often seen in measles is myalgic. Other forms are cephalodynia, in which the muscles of the scalp are involved; dorsodynia, scapulodynia, and so on.

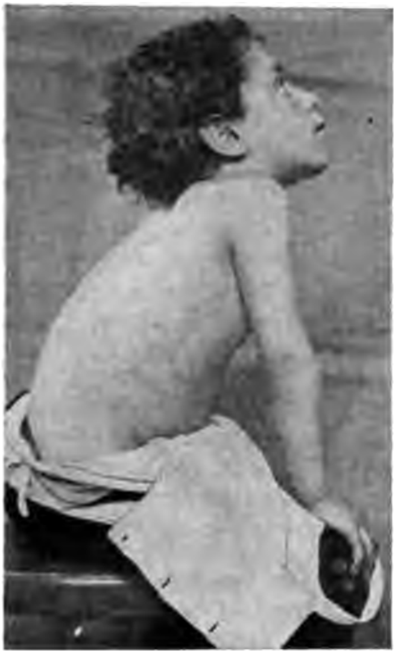


FIG. 307.—Acute rheumatic torticollis.—Rotch.

IV. DIABETES.

Diabetes mellitus and diabetes insipidus have little in common except a persistent increase in the amount of urine secreted. Glycosuria, a symptomatic condition characterized by the transient presence of sugar in the urine, corresponds to polyuria, a symptomatic condition in which the urinary output is greatly increased for a short time. These conditions are to be distinguished from diabetes mellitus and diabetes insipidus, which are substantive diseases.

A. Diabetes Mellitus.

Definition.—A chronic nutritional disease due to diminished capacity for the combustion of carbohydrates, and characterized by the persistent excretion of grape-sugar in the urine when moderate amounts of carbohydrates are ingested, or even none at all in certain cases. Polyuria, polydipsia, polyphagia, and emaciation are prominent but not constant symptoms.

Etiology.—PREDISPOSING INFLUENCES.—Diabetes mellitus prevails in every part of the world, but more extensively in some countries than in others. Southern Italy and India suffer to an especial degree. The disease is about as prevalent in the United States as in Europe and appears to be increasing upon both sides of the Atlantic. In all countries it is more common among those living in affluence than among the poor. The Semitic race manifests an especial predisposition to the disease. This racial peculiarity has been noted by competent observers in various countries. In the United States the negroes suffer to a less extent than the whites.

The predisposition is very commonly inherited. The remarkable prevalence of diabetes among the Hebrews is in many of the cases due to this tendency. Not only is the disease observed in successive generations in the direct, but also in collateral, lines, and it occasionally occurs at an early age in two or more children of the same family. The descendants of gouty or obese persons show an especial liability to diabetes. R. Schmitz first directed attention to the possibility of the transmission of the disease from one person to another. A long and intimate association, as in the case of a wife taking care of a husband suffering from the disease, has, in rare instances, been followed by the development of the same symptoms. Previous good health on the part of the second individual, with absence of hereditary predisposition, has been established in those cases. They are extremely rare and the transmissibility of the disease appears highly improbable.

Diabetes mellitus is more common in men than in women, the ratio, according to available statistics, being about three to two. The disease may occur at any period of life. Infants at the breast are sometimes affected, but such cases are extremely rare; they also run a rapid and fatal course. Hereditary influences are usually in evidence, and several of the children in one family may be affected. But diabetes mellitus is essentially an affection of adult life. A majority of the cases come under observation between the third and the sixth decades. The disease is often discovered upon routine medical examination for life insurance or other purposes, and has already existed in many cases for a considerable time. Those who live luxurious, aimless, and idle lives are peculiarly liable to the disease. The wear and tear of a strenuous intellectual life, especially when coupled with great anxiety and mental excitement, contribute a predisposing influence of great importance. Absorbing application to business, excesses at table, and a sedentary life are important factors in producing the disease. Those who dwell in cities suffer in greater proportion than countrymen. It is, however, to be noted in this connection that the disease is less apt to be recognized in the latter class. Neurotic persons are more commonly affected than those of a phlegmatic temperament, a fact to be considered in the matter of the great relative frequency of the disease among the Jews. Gout, syphilis, and malaria have been regarded as predisposing influences. It was at one time thought that the children of phthisical parents were especially liable to diabetes. The disease frequently develops during the course of chronic nervous affections; still more frequently forms of nervous disease, particularly neuralgia, neuritis, and neurasthenia, are dependent upon the diabetes, and arise as intercurrent affections during its course. Diabetes occurs among the insane, but not, according to the statistics of large institutions, in greater proportion than in general hospitals. Obesity is frequently associated with diabetes. In a majority of such cases the obesity precedes the diabetes often by a period of years. Under these circumstances the disease commonly runs a favorable course, the glycosuria diminishing, even disappearing under a moderately strict regimen, and reappearing when the rules are neglected. Much less favorable are the cases in which obesity and diabetes are simultaneously developed in early life. The form that develops consecutively to obesity has been designated "lipogenous diabetes." Von Noorden, who believes that in the obese cases the burning up of sugar is interfered with, and not its conversion into fat, proposes for this form the term "diabetogenous obesity."

EXCITING CAUSES.—*Psychical.*—Mental shock, intense nervous strain, worry, and violent depressing emotions are frequently followed by diabetes. *Physical.*—Disease or injury of the brain or spinal cord, an irritative lesion of the diabetic centre, and epilepsy may also give rise to the disease. The infectious febrile diseases, especially enteric fever, influenza, diphtheria, rheumatic fever, and syphilis, appear in some instances to have been the starting point of diabetes, the symptoms of which have shown themselves either during or directly after the attack. Under all these circumstances the causal importance of the particular event or condition depends upon the known absence of glycosuria prior to its occurrence. In the vast majority of cases diabetes mellitus develops insidiously, without discoverable cause.

Pancreatic Diabetes.—It has long been known that diabetes and disease of the pancreas are occasionally associated, and Lancereaux described, in 1877, a special form of diabetes under the name *diabète pancreatique.* The discoveries of Minkowski and von Mering, in 1899, aroused intense interest in this subject. The facts are, first, that experimental extirpation of the pancreas is followed by glycosuria; second, that if a portion of the gland is allowed to remain, glycosuria does not occur; third, that in a considerable proportion of the cases of diabetes, lesions of the pancreas have been found—sclerosis, chronic interstitial inflammation, hyaline degeneration of the islands of Langerhans; fourth, that the glycosuria is secondary to the lesions of the pancreas. The theory of an internal secretion containing a glycolytic body necessary to the proper combustion of glucose in the muscles supplies the key to the above facts. Pancreatic disease causes diabetes by arresting the formation of the internal secretion of the organ.

Carbohydrate Metabolism.—In health the carbohydrates of the food are stored in the liver and muscles in the form of glycogen. This substance is also formed from the proteids of the food, and under certain conditions glucose is formed from the proteids of the tissues of the body. The glycogen is again converted into glucose and given up gradually to the blood, in which it circulates in a 0.1 to 0.2 per cent. solution, to be distributed to the muscles, where it undergoes combustion, with the production of heat and energy. According to the investigations of the younger Cohnheim this is brought about by the action of the glycolytic bodies, one derived from the muscles, the other from the pancreas.

Whenever the glucose in the blood is in excess of 0.2 per cent. glycosuria results. This may occur in the absence or in the excess of the glycolytic body; by the sudden ingestion of an excess of carbohydrates—more than 180 to 250 grammes, fasting—*alimentary glycosuria;* and by derangements of circulatory disturbances or instability of the glycogen-storing mechanism.

Symptoms.—In a large proportion of the cases the onset is insidious and not attended by symptoms which attract the attention of the patient. In some instances, in physicians and others who have examined their urine at intervals, the disease has been preceded by an intermittent glycosuria, which, after a period of months or years, has become persistent. There are other cases in which, under treatment, glycosuria has disappeared, to recur when the strict regimen has been relaxed. The existence of the disease is usually recognized by the occurrence of conspicuous symptoms, as polyuria, polydipsia, polyphagia, emaciation, or pudendal pruritus, or by the discovery of sugar in the urine upon examination as a matter of routine or for life insurance.

In rare instances it follows an injury, profound depressing emotion, or a chill. There are cases in which thirst is not inordinate and the amount of urine not excessive. The tongue in established cases is usually red, dry, and denuded of epithelium; the saliva scanty; and the gums swollen and spongy. There are constipation and lumbar pain. The skin is dry and harsh and perspiration is scanty or absent altogether. In women pruritus vulvæ is a common and distressing symptom, and general pruritus is of frequent occurrence in both sexes. The pulse-frequency is high and there is increased arterial tension. The temperature is commonly slightly subnormal. Emaciation is common and rapid in young subjects, but older persons may preserve their weight for long periods.

THE URINE.—Under a rigorous regimen the quantity may not be greatly increased, and it may be reduced to normal during an intercurrent febrile disease. It is, however, commonly increased to three or four litres in cases of moderate severity, and may reach as much as twenty litres in twenty-four hours in grave cases. The specific gravity ranges from 1.030 to 1.045, but in exceptional cases may be low, 1.015 to 1.020, a fact to be borne in mind in the diagnosis. It is pale in color, with a faint greenish tinge, and has a mawkish, sweetish odor, and is said to have a sweetish taste. Sugar is present in amounts varying from 1.5 to 5 or even 10 per cent. The total quantity excreted in twenty-four hours ranges from 300 to 750 grammes or more.

Tests for Glucose.—The most satisfactory tests for clinical purposes are Fehling's, Trommer's, the bismuth test, the fermentation test, and polariscopy. If close results are desired the chemical tests may be controlled by fermentation or the polariscope. The urea and calcium salts are increased, the uric acid does not show important changes, and the phosphates may be much increased.

Phosphatic Diabetes.—This term has been applied to cases in which there is an excessive excretion of phosphates, with symptoms similar to those of diabetes mellitus but with inconstant glycosuria.

Acetone and diacetic acid are often present. The presence of these substances in the urine is conclusive evidence that β-oxybutyric acid, of which they are derivatives, is being produced within the organism. There are three stages in the excretion of the acetone bodies: (a) acetone alone in the urine—this substance is exhaled with the expired air; (b) diacetic acid is also present in the urine; and (c) β-oxybutyric acid is present in the urine in addition to acetone and diacetic acid. Much of the acetone fails of excretion by the urine, since, being highly volatile, it passes off by the respiratory surfaces and lends its characteristic odor to the atmosphere about the patient. The presence in the urine of these bodies is of the highest clinical importance, since β-oxybutyric acid is the cause of diabetic coma.

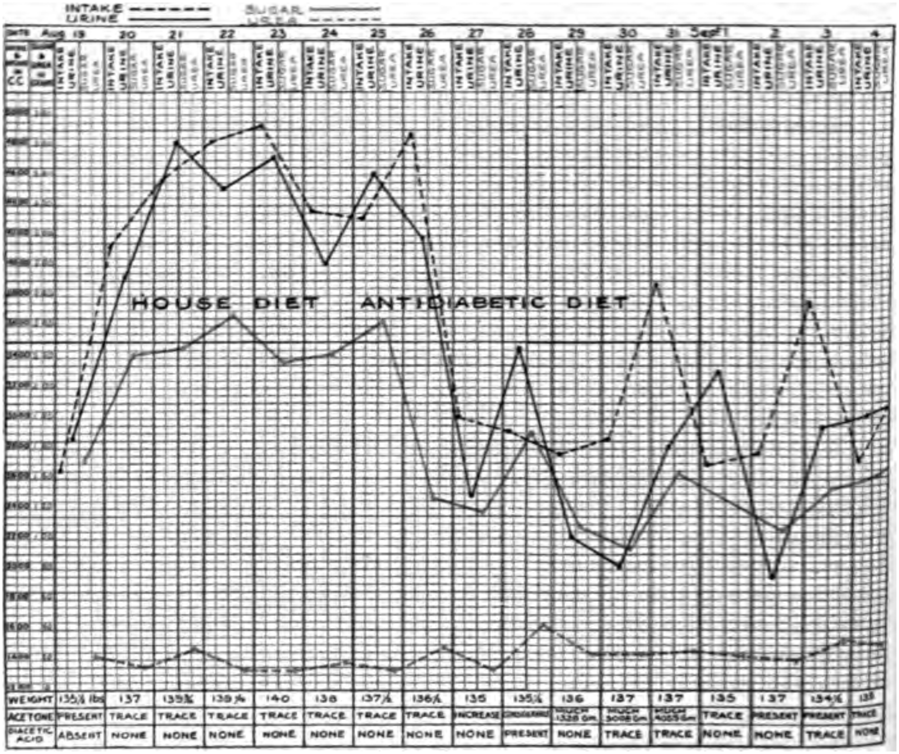


FIG. 308.—Chronic diabetes mellitus; male, age 44. Chart showing daily variations in the intake of fluid, the amount of urine, the quantity of sugar and urea, the specific gravity, and body weight.—Jefferson Hospital.

Glycogen has been found in the urine. Albumin is common, especially in the advanced stages of the disease. *Pneumaturia.*—Gas in rare instances of diabetes passes from the urethra with a bubbling sound. Its presence is the result of fermentative processes in the urine within the bladder. An associated cystitis is common.

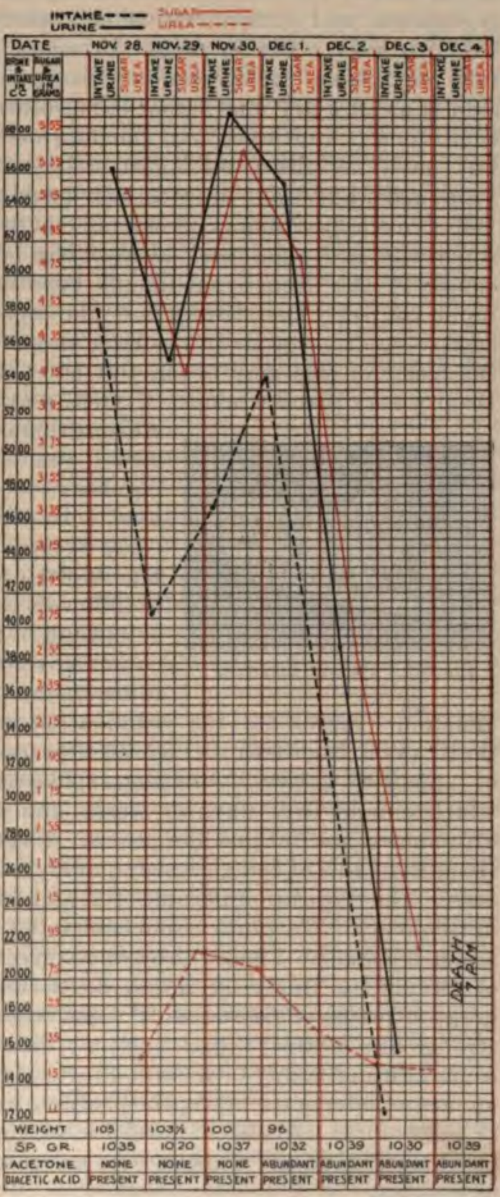


FIG. 309.—Acute diabetes mellitus; male, age 27. Chart showing daily variations in the intake of fluid, the amount of urine, the quantity of sugar and urea, the specific gravity, and the body weight.—Jefferson Hospital.

THE BLOOD.—Hyperglycæmia is constant. An increase in the cellular elements—polycythæmia—may occur, the erythrocytes reaching 6,000,000 or 7,000,000 per cubic millimetre. In coma there is leucocytosis, and β-oxybutyric acid is present. *Lipæmia.*—Large quantities of fat are often present in the blood. The plasma presents a milky appearance, and if the blood is allowed to stand in a glass a thick creamy layer forms at the top, the nature of which is apparent upon testing with ether, osmic acid, and other substances which possess characteristic reactions with fats. A similar fatty layer is obtained by centrifugalization. The fat particles may be readily seen upon microscopical examination of the fresh blood. The fat is identical with that of the chyle and is therefore probably derived directly from the food. The methylene blue reaction—Williamson, Bremer—is of diagnostic value.

Complications.—The diabetic has less power of resistance against pathogenic influences than others, and suffers in a remarkable manner from complications. Among these the following are especially important: SKIN.—Local inflammations, and in particular boils and carbuncles, are common. These lesions have their origin in cracks or fissures of the skin by way of which infection occurs. Wounds and injuries heal slowly and granulation tissue has an especial tendency to slough. Gangrene is common and is due to arteriosclerosis. In rare instances perforating ulcer of the foot has been observed. I have recently seen symmetrical perforating ulcer of the ball of the foot in a woman aged 52. Eczema may occur. Pruritus is common, and pruritus vulvæ is an early and harassing complication. A balanitis may occur in men. Bronzing of the skin—*diabète bronzé*—is a rare cutaneous manifestation associated with hæmochromatosis. The skin is usually dry and harsh, but profuse sweating may occur in terminal conditions. Xanthoma and purpura occur as in other chronic affections, but have no direct etiological relation with diabetes. THE LUNGS.—Pulmonary affections are common terminal events. Croupous pneumonia and bronchopneumonia occur as acute complications. Gangrene may supervene and pulmonary abscess has been observed. Pulmonary tuberculosis of the bronchopneumonic type is common. It runs, as a rule, a rapid course. THE KIDNEYS.—The signs of nephritis, albuminuria, and casts very often occur in the later stages of diabetes mellitus. Œdema of the feet and legs may occur, but the polyuria prevents general anasarca. Dropsy may precede coma. Cystitis may occur. THE SEXUAL ORGANS.—Amenorrhœa may come on early, or the menstrual function may continue more or less regularly to an advanced stage of the disease. Impotence is common and often an early symptom. Conception is not unusual, but is often followed by abortion. Pregnancy is followed by an aggravation of the diabetic symptoms. THE NERVOUS SYSTEM.—Diabetic coma is the most important of the complications on the part of the nervous system. This affection "represents the culmination of the specific diabetic intoxication" (Von Noorden). It is often preceded by gastric symptoms, as loss of appetite, nausea, vomiting, epigastric tenderness, and constipation, which extend over days or weeks and are followed by mental dulness, drowsiness, and fatigue symptoms. The dulness increases and the patient, though capable of being roused, quickly falls back into stupor, like a drunken man. The respiration becomes full and deep, without being much increased in frequency. The pulse is quickened, but remains strong and full. Cyanosis is not constant. Finally drowsiness gives way to coma, and in the course of twenty to thirty-six hours death terminates the scene. In another group of cases coma suddenly supervenes in persons who, if not well and strong, are in their usual health. This fatal complication often follows some unusual bodily or mental effort. Again, coma diabeticum may follow an acute infectious or septic process, or alcoholic intoxication, or ether or chloroform narcosis induced for surgical purposes. The diagnosis may be obscure when the diabetic becomes comatose in consequence of apoplexy or uræmia.

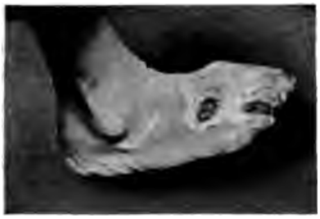


FIG. 310.—Diabetic gangrene.

Minor forms of diabetic intoxication may explain the numbness and tingling, which are common symptoms, and the neuralgias, which sometimes affect the upper or lower extremities, sometimes a single nerve-trunk, as the third or the sciatic. Herpes zoster is encountered in diabetes, but not more frequently than in the non-diabetic.

Tabetic symptoms, lightning pains, loss of knee-jerks, and extensor palsies, manifested by a steppage gait, are mostly the manifestations of a peripheral neuritis, though changes in the posterior columns have been described. Paraplegic symptoms are due to neuritis.

THE ORGANS OF SPECIAL SENSE.—Cataract, almost always double, is a late development and occurs in cases in which the glycosuria is of high grade. It develops with great rapidity in young diabetics, but more slowly in the elderly, in which it cannot be differentiated from the senile form. Retinitis, due to associated renal disease, or the ordinary hemorrhagic form, may occur, and optic neuritis followed by atrophy. Sudden amaurosis without ophthalmoscopic changes may come on in the early stages of diabetes. Changes in the organs of hearing, smell, and taste are not common. Otitis media may occur and be followed by mastoid disease.

Diagnosis.—DIRECT DIAGNOSIS.—The presence of grape-sugar in the urine, continuing for weeks, months, or years, even upon a diet containing carbohydrates in moderate amounts, is the fundamental diagnostic criterion. Of secondary importance in the diagnosis because they are not constant are inordinate thirst, excessive appetite, the excretion of abnormally large quantities of urine, and loss of weight. Very often all these symptoms are present and give rise to a characteristic clinical picture. The occasional temporary absence of sugar under a diet which does not contain carbohydrates, or during an acute illness, does not constitute an objection to this diagnosis, since such a diet cannot be indefinitely continued, and sugar reappears in the urine when carbohydrates are added, and also reappears upon convalescence from an intercurrent febrile disease.

In the examination of the urine for sugar the total quantity for twenty-four hours is collected, that of the day and of the night separately. Both the percentage and the total quantity of sugar are ascertained. The presence or absence of acetone and oxybutyric acid is also noted and tests for albumin are made. The specific gravity and reaction are recorded.

The quantitative estimation of the ammonia is of great importance, since it permits conclusions in regard to the approximate amount of oxybutyric acid which is excreted at the same time. When more than 2 grammes of ammonia are excreted in twenty-four hours, there is danger of coma.

It is to be remembered that the glycuronates, which sometimes precede the excretion of sugar and are frequently associated with sugar in the urine, reduce alkaline copper solutions but do not ferment, and that the homogentisic acid of alkaptonuria reacts to the copper tests but not to Nylander's bismuth test, nor to fermentation. The fermentation test is the most reliable single test, but must, in doubtful cases, be controlled by other tests, since the yeast sometimes contains sugar.

DIFFERENTIAL DIAGNOSIS. — *Non-diabetic Glycosuria.* (a) *Transient Glycosuria.*—In rare instances this condition follows concussion of the brain, cerebral apoplexy, severe neuralgias, and profound depressing emotion. The sugar is present usually for a few hours, in some instances for a few days, and shows no tendency to recur.

Under this heading must also be grouped the acute forms of glycosuria, lasting but a few hours, which accompany poisoning by morphine, amyl nitrite, carbonic oxide, chloralamide, and nitrobenzole; the rare glycosuria noted in biliary colic, and that observed in hydrocyanic poisoning. *Phloridzin Poisoning; Renal Glycosuria.*—The sugar is present so long as the administration of phloridzin is continued, alike when carbohydrates have been ingested, or the animal is fed upon a proteid diet or is fasting.

(b) *Intermittent Glycosuria.*—This condition occasionally occurs in gouty persons and in the non-gouty as the precursor of diabetes. The assimilation limit for carbohydrates may be determined by the administration of 100 grammes of glucose in solution, two hours after a breakfast of a roll and butter with coffee, during a period in which glycosuria is absent. This amount of sugar should not, in a healthy person, cause glycosuria. The excretion of sugar indicates a fault in the storage or metabolism of the carbohydrates. Many cases of diabetes mellitus begin as intermittent glycosuria.

(c) *Alimentary Glycosuria.*—The limit of assimilation for glucose in the healthy subject varies from 120 to 200 grammes in a single dose. If this be exceeded glycosuria occurs, but only a portion of the quantity ingested appears in the urine, the remainder being stored as glycogen in the liver and muscles for future use. The limit for cane sugar is about the same, that for milk sugar much lower, and for maltose in many individuals very low. It is stated by Von Noorden that in some persons half a litre of beer is sufficient to give rise to sugar in the urine and that this fact should be known to avoid the danger of mistaking a harmless symptom for a serious disease. In rare instances glycosuria follows excesses in alcohol.

(d) *Malingering.*—Persons have been known to feign diabetes by dissolving sugar in the urine. The specific gravity is high and the reactions those of cane sugar; but very well-informed patients may use glucose for this purpose. The fraud is, however, easy of detection.

(e) *Glycosuria in Pregnancy and the Lying-in State.*—Sugar occurs under two circumstances: First, the pregnant woman may have been diabetic before conception or may have become so during pregnancy. The reactions are those of grape-sugar and the significance is unfavorable; secondly, milk sugar may be resorbed from the breasts and excreted by the kidneys. This occurs when there is a hypersecretion of milk or, for some reason, such as fissure of the nipple or the removal of the child, there is an interruption of its withdrawal. The reactions are those of lactose and the prognosis is favorable, the sugar disappearing as the secretion of milk is arrested. The condition is not glycosuria but puerperal lactosuria.

Prognosis.—The dangers of diabetes consist in lowered nutrition, diminished powers of resistance to intercurrent diseases, inflammatory and chronic degenerative processes, and, in the severe cases, an abnormal production of acids.

Favorable prognostic indications are onset or recognition of the disease at an advanced period of life, absence of emaciation, the gouty habit of body, the occurrence in the patient's family of other cases running a mild course, slight glycosuria, and tolerance for moderate amounts of carbohydrates. The prognosis is unfavorable when the disease begins at an early age and when other cases of severe type have occurred in the patient's family. Rapid emaciation, grave intercurrent affections or complications, intense glycosuria, and intolerance for carbohydrates are of ominous prognosis. The patient's circumstances and ability to avail himself of favorable personal influences, such as the avoidance of overwork and worry, are very important. Finally, the excretion of ammonia in large amounts and the presence of the acetone bodies in the urine are of immediate gravity, because they are the common heralds of coma.

B. Diabetes Insipidus.

Definition.—A chronic disease characterized by great thirst and the habitual discharge of excessive quantities of urine of low specific gravity.

This affection is a primary or idiopathic disease and is to be differentiated from the transient or persistent hypersecretion of urine, which is symptomatic of certain forms of chronic Bright's disease and some affections of the nervous system.

Etiology.—PREDISPOSING INFLUENCES.—Heredity exerts an important influence. Cases have been known to occur in four generations. The disease is sometimes congenital. There is very often a history of chronic disease on the part of the parents of the patient. Diabetes, renal affections, pulmonary phthisis, gout, and rheumatism have been noted. Diabetes insipidus most commonly develops in early life. It is relatively frequent in young children and rarely begins after thirty. It is more common in males than in females.

EXCITING CAUSE.—The actual cause is unknown. The disease usually develops insidiously without assignable cause. It has in some instances been attributed to excessive quantities of water or beer, and has followed an acute infectious disease, especially influenza. In young children malnutrition arising from neglect, insufficient food, and constitutional taint have been assigned as a cause; in older persons acquired syphilis, alcoholism, worry, anxiety, and prolonged exposure to cold.

Symptoms.—The two symptoms which are characteristic are an excessive quantity of urine without sugar, and intense thirst.

THE URINE.—The quantity voided is enormous. It often reaches twenty or thirty pints in twenty-four hours. Fifty-six pints have been noted. It may even at times exceed the fluid ingested, the difference being made up of fluid withdrawn from the tissues of the body and the food. It is pale in color and limpid, the specific gravity varying between 1.001 and 1.007. The reaction is faintly acid or neutral. The total urinary solids are increased by one-fourth or one-third. *Inosite—muscle sugar*—is occasionally present in small amounts. Albumin is usually absent until late and grape-sugar is occasionally present toward the close, when the symptoms of diabetes mellitus sometimes occur. THIRST.—This symptom is proportionate to the quantity of urine. As much as fifty pints of fluid have been consumed by a patient in the course of a day. There is usually a remarkable thirst for alcoholic beverages. BULIMIA.—The appetite is usually enormous and the digestion well performed. These conditions fail, however, in the terminal dyscrasia, when there may be complete anorexia, flatulence, and unmanageable diarrhœa. The patients are often well nourished and healthy-looking for years, the main troubles being unquenchable thirst and frequent micturition. Emaciation is an early symptom and becomes, toward the last, extreme. The saliva is scanty, the mouth dry, perspiration slight, and the skin dry and harsh.

Diagnosis.—DIRECT.—Extreme thirst, excessive habitual secretion of non-saccharine urine of low specific gravity, and emaciation justify the diagnosis of diabetes insipidus.

DIFFERENTIAL.—The distinction between the idiopathic disease and symptomatic polyuria is extremely important. The following forms of the latter are to be considered: *Diabetes Mellitus.*—Persistent glycosuria and high specific gravity are important. If sugar appears in diabetes insipidus it is usually in faint traces and transitory. To this statement an exception must be made in regard to the cases in which diabetes mellitus constitutes a terminal condition. *Diseases of the Nervous System.*—Polyuria is a conspicuous symptom in certain cases of brain tumor, lesions of the medulla, and intracranial hemorrhage. It occurs also with some frequency in cerebral syphilis and has been observed in lesions of the cord. *Hysterical Polyuria.*—Copious, limpid urine of low specific gravity may simulate diabetes insipidus. The condition is transitory and the characteristic features of hysteria render the diagnosis a simple matter. *Contracted Kidney.*—There is frequently a large amount of urine of low specific gravity. Albumin is at times absent. It is, however, much more commonly present in association with granular and hyaline casts. The signs of arteriosclerosis and cardiac hypertrophy are important. *Polyuria in Abdominal Diseases.*—This symptom is sometimes prominent in tuberculous peritonitis, aneurism of the abdominal aorta or iliac arteries, tumors of various kinds, and especially malignant new growths. *Hydronephrosis.*—The periodical discharge of large quantities of urine in connection with the subsidence of fluctuating abdominal tumor, which slowly reforms during the intervals, is of positive diagnostic significance. *Intercurrent Polyuria in Enteric Fever.*—I have reported a case of excessive urinary discharge, reaching a maximum of six litres in twenty-four hours, with slight increase of total urinary solids, occurring during the course of an otherwise mild attack of enteric fever, with disappearance of the polyuria upon convalescence. Other similar cases have been observed. *Malingering.*—Water may be added to the urine, but the absence of thirst, bulimia, and emaciation would lead to a suspicion of fraud which may be readily exposed upon investigation.

Prognosis.—The course of diabetes insipidus is extremely variable. It may be acute and rapid, or continue for many years with but slight deterioration of the general health. Recovery may occur and spontaneous cures have been noted. Death commonly results from some intercurrent affection.

V. NUTRITIONAL DISEASES.

A. Scurvy.

Scorbutus.

Definition.—A nutritional disease, due mainly to improper food, and characterized by anæmia, great debility, swollen and ulcerated gums, and subcutaneous, submucous, and subperiosteal hemorrhages.

Etiology.—PREDISPOSING INFLUENCES.—Scurvy is a disease of unsanitary conditions and has occurred in all parts of the world in which such conditions prevail. It has been known from the earliest historical periods and has been the scourge of armies in the field, soldiers and sailors on long voyages, and the peoples of beleaguered cities. This disease may occur in any climate, but is more common in northern climates and cold countries. It is more frequent in cold weather than in warm, in rigorous winters than in mild, and in periods of famine. Among adults males suffer more frequently than females, and more severely. In besieged cities where the conditions affecting the sexes have been very similar, the proportion of men affected has exceeded that of women. It is most common in adult life, but affects children and the aged. A special form is known as *infantile scurvy.* Malaria, syphilis, dysentery, alcoholism, defective teeth, and gastro-intestinal disorders constitute predisposing influences of recognized importance. Anxiety, prolonged fear, nervous depression, and nostalgia play an important part in the predisposition to the disease, but are incapable of causing it.

PATHOGENESIS.—There are four hypotheses as to the essential cause of the disease:

1. That it is due to the absence in the food of certain substances present in fresh vegetables. The nature of these substances has not been positively determined. They have been thought to be various organic salts present in fruits and vegetables; or the potassium salts (Garrod). According to Ralfe the lacking substances are the malates, citrates, and lactates, from which are derived the carbonates upon which the alkalinity of the blood depends. This view appears to be disproved by the fact that scurvy does not prevail among the Esquimaux, who live exclusively upon animal foods and fats which are often tainted, and that of Nansen and other Arctic explorers who, living for months upon a similar diet in most unhygienic surroundings, escaped the disease.
2. That it is due to toxic substances of unknown character, products of decomposition, in the food.
3. That it is essentially a toxæmia, resulting from the absorption of poisonous substances from the gastro-intestinal tract, produced by microörganisms in the intestinal contents.
4. That it is an infection depending upon an unknown specific microorganism, for which depressing influences, unsanitary conditions, and improper food prepare the soil.

It is to be noted that prolonged insufficiency of food—starvation—on the one hand does not necessarily result in scurvy, and that, upon the other, the disease may occur with an abundant diet of improper and monotonous food.

Symptoms.—Scurvy occurs as an epidemic, endemic, and sporadic disease. Sporadic cases are often encountered in prisons, almshouses, hospitals, and other similar institutions, and occasionally among well-to-do persons in private life, who, as a matter of fancy, or for some other reason, have lived for a long period upon a restricted and unvaried diet. In the United States, where scurvy is at present a rare disease, these sporadic cases are often unrecognized. The disease is insidious in its development. It may be subacute or chronic. In very rare cases the onset is abrupt.

The course in ordinary cases is progressive and attended by fluctuations in the intensity of the symptoms. The early manifestations consist of weakness, pallor, and loss of weight. The gums become swollen, spongy, and ulcerated. Sometimes they show the continuous oozing of blood. The teeth are loosened and frequently fall out. The tongue is enlarged, red, and frequently heavily coated. Submucous hemorrhages in the mouth occur and the breath is disgustingly fetid. The skin becomes dry and harsh and petechiæ appear in and around the hair-follicles, at first upon the legs and later upon the arms and trunk. Subperiosteal extravasations upon the legs often give rise to painful nodes, which sometimes break down and form deep ulcerations. Subcutaneous hemorrhages occur at points of injury or pressure, and brawny indurations occur in the subcutaneous tissues, with discoloration of the overlying skin. Epistaxis is common and free bleeding from other mucous surfaces takes place in the graver cases. Hemorrhagic infarcts in the lungs and spleen with characteristic symptoms may be noted. Feeble action of the heart with arrhythmia and palpitation are frequent and there is often a basic systolic murmur. There is anorexia and inability to masticate food owing to the condition of the gums. Constipation is the rule. Arthritis has been noted. The urine is albuminous. Urea is diminished. The other constituents show inconstant changes. Mental depression and languor are common. Delirium and coma occur as terminal events. Subconjunctival and intra-ocular hemorrhages are of common occurrence and hemeralopia and nyctalopia are occasional symptoms. Fever is not usual, but pyrexia of irregular type may occur in the presence of complications, such as pleurisy, pericarditis, or abscess formation.

Complications and Sequels.—Abscesses, inflammation of the serous sacs with hemorrhagic effusion, croupous pneumonia and bronchopneumonia, pulmonary gangrene, and pulmonary œdema as terminal events constitute the most serious complications. Gastro-intestinal complications are common. Ankylosis of joints that have been inflamed, particularly the elbow, knee, and ankle, may cause permanent deformity,

Diagnosis.—The DIRECT DIAGNOSIS in the epidemic and endemic disease is unattended with difficulty. The surroundings and circumstances of the patient, the condition of the gums, the petechiæ and ecchymoses, the languor and anæmia, the nodes due to subperiosteal bleeding, and the results of dietetic and hygienic treatment establish the nature of the disease.

DIFFERENTIAL DIAGNOSIS.—The diagnosis in sporadic cases may be difficult, especially in persons living in affluence in whom errors of diet and unhygienic surroundings are unsuspected. The foregoing diagnostic criteria are important. The lesions of the gums are absent in early infancy prior to dentition, and in aged persons who have lost their teeth. Pericarditis and pleural effusion of scorbutic origin may, in the absence of the lesions of the gums and subcutaneous hemorrhages, give rise to serious diagnostic difficulty. The anamnesis, the evidences of deep-seated hemorrhages, and the results of treatment are important. *Purpura.*—The various forms of purpura differ from scurvy in their causal relations, the absence of the peculiar lesions of the gums, and the absence of the deeper-seated hemorrhages.

THERAPEUTIC DIAGNOSIS.—An abundance of fresh vegetables and meat, such as constitutes an ordinary wholesome mixed diet, is usually followed by a remarkable disappearance of the symptoms, even in cases of great severity. The articles of diet which have the reputation of being especially valuable comprise potatoes, lettuce, cabbage, spinach, and fresh fruits and fruit juices, as lemon and lime juice. From the time that the regulations of the Board of Trade have required that a sufficient quantity of such articles be included in ships' supplies, the occurrence of scurvy among sailors has become a rare event.

Prognosis.—The cases are apt to recover unless the causal conditions persist or they are far advanced when treatment is begun. This disease is now infrequent and the mortality low. Death results from progressive inanition, sudden syncope, large serous effusions, pneumonia, pulmonary œdema, meningeal hemorrhage, or sepsis.

B. Infantile Scurvy.

Barlow's Disease.

Definition.—A nutritional disease of young infants, due to improper food and characterized by subperiosteal hemorrhages, particularly in the lower extremities, a form of pseudoparalysis, and a cachectic condition. Our knowledge of this disease, which was formerly confounded with rickets and infantile syphilis, is of comparatively recent origin (1881–83).

Etiology. — PREDISPOSING INFLUENCES. — Infantile scurvy is more common among the well-to-do than among the poor, a condition to be explained, first, by the fact that the children of the former are more commonly fed upon artificial foods, and second, that among the latter the child has at an early age a more varied diet, into which enter to some extent articles of ordinary table food quite unknown to children of the same period among the affluent. Scurvy begins most commonly between the sixth and eighteenth months. It is rare earlier, but has been observed as late as the fifth year. Rheumatism, syphilis, and rickets have been supposed to bear a causal relation to infantile scurvy. Of these the first two have nothing to do with the disease in question and the last, though sometimes associated with it, is wholly different in its etiology, pathology, and symptomatology, and when present in the same individual persists when the scorbutic symptoms have disappeared.

THE IMMEDIATE CAUSE.—Improper diet is the cause of this disease. The various commercial foods, including condensed milk (especially when prepared with water), sterilized milk, and other artificial foods (particularly when administered in unvarying monotony), are found to have constituted the diet in almost every case for a considerable period prior to the manifestation of the symptoms.

Symptoms. — The disease shows itself as an insidiously developing cachexia. The child is fretful and peevish. It lies quiet when undisturbed, with its thighs and legs strongly flexed, but screams when any attempt is made to extend them. Obscure swellings due to subperiosteal hemorrhages may be observed on both lower extremities but they are not symmetrical. These ill-defined, tumor-like prominences occupy the lower ends of the femurs, the tibiæ, and less frequently the bones of the forearms. They are most marked just above the epiphyseal junction and extend along the shafts of the bones. Similar swellings may sometimes be found upon the scapulæ. The overlying tissues are boggy and slightly œdematous and the skin is somewhat tense. As the disease progresses the whole limb becomes thickened. Presently the limbs assume a different position, being no longer drawn up, but everted and motionless—*pseudoparalysis*. The joints are not involved. Separation of the epiphyses and fracture may occur in severe cases, these lesions being manifest by crepitus and further deformity. Barlow described a remarkable depression of the sternum and costal cartilages. Proptosis of the eyeballs, more marked upon one side than the other, with œdema and slight discoloration of the eyelids, may occur in advanced cases. Petechiæ occur, but are much less conspicuous than in the scurvy of adults, but hemorrhages from the mucous surfaces are common. Anæmia—3,000,000 to 2,000,000 or lower—is proportionate to the severity of the case. The white corpuscles show no constant changes. The color of the skin is pallid and earthy; emaciation is not a marked feature; asthenia is extreme. The temperature may be normal or slightly subnormal, with occasional transient rises to 102° or more, these usually accompanying the signs of fresh subperiosteal hemorrhages. If the teeth have appeared the gums may be swollen and spongy.

Diagnosis.—Direct.—It is a matter of surprise that the true character of infantile scurvy is so often overlooked. Few diseases of infants present a more characteristic symptom-complex or a more obvious etiology. The attitude, the behavior of the child upon being handled, the anæmia, the elongated, subperiosteal nodes and thickening of the limbs, the immobile, forced flexion of the limbs in the early, and the pseudoparalysis in the later, course of the affection are diagnostic. Proptosis and œdema of the eyeballs are significant.

Differential.—*Rickets.*—The gastro-intestinal symptoms are more prominent than in scurvy. The rachitic rosary and the lesions of the bones are characteristic. The boggy swellings, protrusion of the eyeballs, petechiæ, and spongy gums when the teeth are present do not occur in rickets. The two diseases may, however, be associated. The forms of *purpura* resemble scurvy only in the presence of petechial and other hemorrhages, but the distribution of these lesions and the absence of all else characteristic of the latter disease render the diagnosis an easy matter. *Infantile Paralysis.*—The pseudoparalysis may suggest this affection, but the history of the case, the sudden onset, the absence of pain, tenderness, and the localized swellings are diagnostic. *Syphilitic Pseudoparalysis; Parrot's Disease.*—Sudden loss of motion in the lower or upper limbs, or both, with great pain on passive movement, and crepitus due to a separation of the epiphyses may present a superficial resemblance to infantile scurvy This resemblance ceases upon a proper consideration of the anamnesis and the lesions. The diagnostic criteria of congenital syphilis are usually unmistakable. In any doubtful case an etiological diagnosis based upon the nature of the diet will be helpful.

Prognosis.—The outlook is favorable in cases early recognized. Complete recovery, with the disappearance of the lesions, often takes place in the course of two to four weeks after the institution of a proper diet. More advanced cases recover more slowly.

C. Rickets.

Rhachitis.

Definition.—A disease of infants due to improper diet and characterized by impaired nutrition of the tissues of the body and specific alterations of the skeleton.

Etiology.—PREDISPOSING INFLUENCES.—All those conditions which involve neglect of hygiene, and especially of alimentary hygiene, favor the development of rickets. The geographical distribution of this disease is wide. It abounds in great cities and crowded industrial centres. It is more common in Europe than America. In this country it is especially prevalent among the children of recent immigrants. The great frequency of rickets among the children in the Italian and negro colonies of American cities is due not to racial but to social conditions. Rickets affects male and female children to the same extent and degree. If we except the rare condition known as fetal rickets—*achondroplasia, chondrodystrophia fœtalis*—and the late form described by Jenner—the *osteomalacia of puberty*—rickets is a disease of the first two years of life, a period corresponding to the first dentition. It rarely begins before the sixth month or after the third year. Rickets is especially a disease of poverty and all that poverty entails—want of sunlight and want of fresh air, neglect, filth, and insufficient and improper food. Rickets, like scurvy, occasionally occurs as a sporadic disease in the families of the well-to-do. Rickets has been looked upon as a manifestation of congenital syphilis, but this view has been abandoned alike upon etiological and pathological grounds. A syphilitic child is not rhachitic, though it may acquire rickets, and the two conditions frequently coexist. There is no evidence that rickets is hereditary. The endemic and epidemic prevalence of the disease, under certain social conditions, is neither evidence of its hereditary nature nor of its contagiousness, as has been assumed.

THE CAUSE.—An improper diet is the essential cause of the disease. Prolonged lactation and suckling the child after pregnancy has occurred bring the milk of the nursing mother within the category of improper food. Cow's milk, foods rich in starches, condensed milk, and the various commercial infant's foods are responsible for a large proportion of the cases. Deficiency in fat and proteids, and failure in the assimilation of the lime salts, constitute the chief alimentary defects.

THE PATHOGENESIS.—The following are the chief hypotheses: (1) Rickets is a manifestation of congenital syphilis (Parrot), (2) a trophic disease of the bones, resulting from nervous derangements; (3) an infection; (4) a disease of nutrition due to faulty alimentary hygiene. Of these, the last, in various modified forms, is the view now generally maintained.

Symptoms.—Rickets is a chronic disease of insidious onset, beginning during the first dentition and usually before the child begins to walk. It is preceded by digestive disorders of varying degree, and impaired nutrition, but not necessarily by emaciation. The child is often pallid, plump, and soft. Slight fever, irritability, and poor sleep are suggestive symptoms. He is feeble and unsteady on his feet and disinclined to walk. There is diffuse tenderness of the tissues and unwillingness to be handled or touched. Free sweating, especially about the head and neck, is common. The weakness of the muscles, and especially in the legs, is suggestive of partial paralysis—*pseudoparalysis* of rickets. The skeletal changes appear early in the course of the disease and are characteristic. They consist of: 1. The "RHACHITIC ROSARY," composed of nodular enlargements of the ribs at their juncture with the cartilages on both sides. These enlargements may be readily felt upon palpation and in thin children may be recognized upon inspection. They appear early and gradually increase in size until

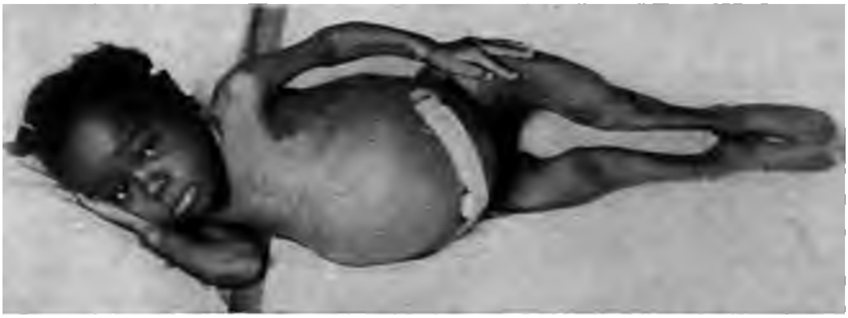


FIG. 311.—Rickets. Showing the epiphyseal enlargements, the rosary, the distended belly and the deformities as a whole.—Pennsylvania Hospital.

some time in the second year, after which they gradually disappear. 2. CHANGES IN THE THORAX.—Shallow furrows, corresponding to the junction of the cartilages with the ribs, pass obliquely downward and outward. A similar transverse depression extends from the level of the ensiform cartilage toward the infra-axillary space—*Harrison's groove*. The sternum projects, particularly in its lower half, giving rise to the prominent deformity known as *chicken-* or *pigeon-breast*. These changes in the contour of the chest are not peculiar to rickets and may occur in any condition habitually interfering with inspiration in early life. 3. CHANGES IN THE HEAD.—As a rule the head appears large, the frontal and parietal eminences are exaggerated, and the fontanelles remain open for a long time. The forehead is prominent, the top of the skull flattened, and in some cases the head viewed from above appears square— [illegible] —There are circumscribed [illegible] occipital, parietal, and [illegible] in consequence of [illegible] the skull [illegible] of the finger, giving rise [illegible] observed also in syphilis. A systolic [illegible] over the anterior fontanelle and in the temporal region. This auscultatory phenomenon is sometimes heard in healthy children. The bones of the face also show changes, especially in the maxillæ, which are small and angular. The normal course of dentition is deranged and retarded. The first teeth may not appear until some time in the second year and undergo cariės at the time of their eruption. The cephalic changes of rickets are very often first in the point of time. 4. CHANGES IN THE PELVIS.—The changes in the pelvis are of especial importance in female children, since they lead to deformities with narrowing, which interfere with natural labor and frequently render it impossible. 5. CHANGES IN THE EXTREMITIES.—The scapulæ are not usually affected. The clavicles are often thickened at the sternal ends and at the point of insertion of the sternocleido muscles; their curves are exaggerated and they are shortened. The rhachitic deformities are most conspicuous in the long bones. They consist of enlargements in the region of the junction of the shaft and epiphysis, and curvatures, which in the lower extremities cause a corresponding diminution in the height of the individual. The enlargements in the upper extremities are most marked at the distal ends of the radius and ulna. To a less degree the lower end of the humerus may be affected. *The Rhachitic Hand.*—Koplik has described a deformity of the hands which occurs in rickets, consisting in thickening and bowing of the phalanges of the fingers, associated with laxity of the ligamentous structures of the phalangeal joints. The changes give rise to a somewhat characteristic appearance of elongation of the fingers and plumpness of the whole hand. They have been observed in connection with the ordinary lesions and deformities of well-marked rickets. In cases attended by pains in the bones "the rhachitic hand" may suggest syphilis, but the association of the lesions of infantile syphilis renders the differential diagnosis easy. In the lower extremities the lower end of the tibia, of the fibula, and of the femur show progressive enlargements proportionate to the severity of the case. If the child walks, the femurs are curved forward and the bones of the legs forward and outward. Exceptionally the curves may cause the deformity known as knock-knee. These abnormal curvatures are due to the muscular traction and the weight of the body upon the decalcified and softened bone.

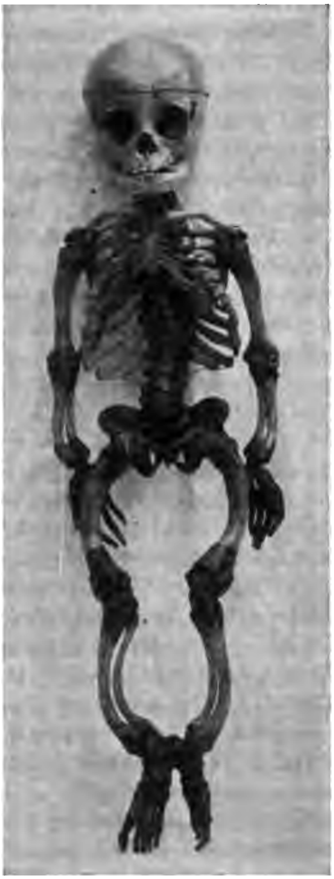


FIG. 312.—The skeleton of the body shown in Fig. 311.

The liver is enlarged; the spleen enlarged and palpable. There is usually more or less flatulent distention. These conditions combine to render the belly large and protuberant, a condition made more conspicuous by the relatively small size of the thorax.

The urine shows no constant changes. There is slight anæmia; the hæmoglobin is decreased; leucocytosis may or may not be present. The nervous symptoms increase with the severity of the other symptoms. Convulsions are common. Tetany and laryngismus stridulus are occasional intercurrent affections. The growth of the child is greatly retarded and many dwarfs are rhachitic.

Diagnosis.—DIRECT.—Many cases are so slight as to escape recognition. Weakness, fretfulness, pallor, diffuse soreness, profuse sweating of the head during sleep, an open fontanelle, and irregular evening fever justify a provisional diagnosis, especially when defects of hygiene and diet exist. When to these symptoms are added the skeletal changes above described, especially those which first appear, namely, the rosary and craniotabes, it becomes positive.

Prognosis.—The slighter forms are amenable to treatment and recovery takes place without deformity. The graver cases recover more slowly and with lasting skeletal changes. The disease is essentially chronic, and, though not in itself fatal, renders the patient peculiarly liable to intercurrent affections, while it at the same time diminishes the powers of resistance.

D. Obesity.

Definition.—An excessive development of fat.

The condition is not always pathological. It is better to be fat and enjoy a normal amount of health and vigor than to reduce the fat by an unwise and rigorous regimen and depleting drugs, and become an invalid.

Etiology.—PREDISPOSING INFLUENCES.—The hereditary tendency to obesity may be demonstrated in about fifty per cent. of the cases. This tendency may be manifest in childhood, in women after the first pregnancy, or not until middle life. It is more common in women than in men. Not alone in the hereditary cases, but also in those in which the tendency is acquired, is the condition more common in the female. There is a manifest relation between sexual inactivity and the tendency to corpulence. In males the tendency to accumulate excessive fat frequently begins in the fifth decade of life; in females at puberty, during the period of childbearing, and at the grand climacteric. The distribution of fat varies at different periods of life. In infancy and childhood the undue accumulation is chiefly subcutaneous; in middle life it is visceral as well as subcutaneous, while in the aged the subcutaneous fat may disappear and that in the omentum, mesentery, pericardium, mediastinum, and around the kidneys persist. Persons of phlegmatic temperament, given to repose and the pleasures of the table, are more disposed to obesity than those who are sanguine, active, and self-denying.

THE ACTUAL CAUSES.—In general, obesity is due to the ingestion of excessive and improper food and an indolent and inactive life, but to this statement there are many exceptions. There are fat persons who are small eaters and exercise constant care in the selection of their diet, and among the obese are to be found men of superior intelligence and energy.

Fats, starches, and sugars taken in excess cause obesity. The habitual ingestion of large quantities of fluids and the abuse of alcohol are important etiological factors.

Symptoms.—Oertel describes a plethoric and an anæmic form. The first is more common in men who are high livers and consume much beer. The face is flushed, the subcutaneous and visceral fat are increased, but the muscular power is preserved for a long time. The second is especially encountered in chlorotic girls and anæmic women. The face is pallid, the skin white, the subcutaneous fat especially abundant, and the muscular power feeble. The ankles are often slightly œdematous. The hands and feet long remain free from disfiguring fat. Both types are too familiar to require detailed description.

Diagnosis.—DIRECT.—The recognition of obesity is a matter of little difficulty. The contour of the body and the disproportion between the height and weight of the individual are diagnostic. More difficulty arises in determining the line at which normal corpulence proper to the age, habits, and hereditary peculiarities of the individual ends, and obesity with its inconveniences and dangers begins. This can only be done by a careful study of individual cases.

DIFFERENTIAL. — *Œdema.* — The irregular, doughy masses of subcutaneous fat, with the sharp folds of the skin and general distribution which characterize obesity, are in sharp contrast to the smooth, tense, glistening skin of anasarca, with its tendency to accumulate in the dependent tissues, where there is characteristic pitting upon pressure. *Myxœdema.* —The dense subcutaneous infiltration, symmetrical and of moderate extent, not pitting upon pressure, the implication of the hands, the pads, the mental state, and the prompt reaction to thyroid medication render the diagnosis clear. *Emphysema of the Subcutaneous Tissues.*—This rare condition may suggest obesity, but the history of the case, the circumscription of the swelling, and crackling upon palpation are characteristic.

Prognosis.—The outlook, varying with the causes, degree, symptoms, complications, and the disposition of the patient, ranges from favorable to positively ominous. It is less favorable in the hereditary than in the acquired form, in the anæmic than in the plethoric, and in the cases in which feeble action of the heart, arteriosclerosis, gout, albuminuria, or diabetes is present. Obese persons bear intercurrent febrile infections badly and usually make a tardy and unsatisfactory convalescence because of the slow regeneration of red blood-corpuscles and enfeebled recuperative powers.

Adiposis Tuberosa Simplex.—Under this term Anders has described a rare condition encountered in obese persons, characterized by the presence of circumscribed masses of fat in the subcutaneous tissues, particularly in the extremities and abdomen, and forming distinct, moderately dense, slightly movable, somewhat flattened tumors varying in size from a bean to a hen's egg and in number from six to twenty-four or more. These masses are not elevated above the surface and show no tendency to fuse together. They are sensitive to palpation and are sometimes, but not always, the seat of pain of variable intensity. The overlying skin is not adherent. Their etiology is not clear, but their relationship to corpulency is manifest from the fact that they disappear as that condition is reduced under treatment. Adiposis tuberosa simplex is to be distinguished from: (1) *Adiposis dolorosa—Dercum's disease*—which is not amenable to treatment, and in which definite changes in the thyroid gland and the pituitary body, together with extensive interstitial neuritis and degeneration of the columns of Goll, have been found post mortem. (2) *Lipomata.*—Fatty tumors which are painless, soft and doughy, globular in shape, often lobulated, usually distinctly elevated above the surface, and which occur independently of general obesity and remain uninfluenced by treatment. (3) *Adenolipomatosis*, in which fatty accumulations develop in relation with the lymph-nodes of the neck, axillæ, or groins. These fat masses are symmetrical in distribution and occur in various chronic constitutional diseases, and only rarely are associated with general obesity. In fact, they may persist when, in consequence of the progress of the associated malady, emaciation has occurred or cachexia developed.

Adiposis Dolorosa.—DEFINITION.—Dercum first called attention to "a disorder characterized by irregular, symmetrical deposits of fatty masses in various portions of the body, preceded by or attended with pain."

The disease occurs chiefly but not exclusively in women at middle life. Neuralgic pains precede and accompany the disorder. Irregular hyperæsthesia and paræsthesia occur. Fatty masses, sometimes of enormous size, lumpy, soft, and pendulous, form at various points of the body, in association with a general great increase of the subcutaneous fat. The face, hands, and feet are not affected. Atrophy of the thyroid body has been noted in some of the cases, and the administration of thyroid extract has been followed by relief of the neuralgia and diminution of the fat. Lesions of the pituitary body with interstitial neuritis and degeneration of the columns of Goll have also been found post mortem. The essential nature of the trouble is unknown. This disease differs from other forms of obesity in its unknown etiology, the distribution of the fat in masses, and the presence of marked nervous symptoms, especially pain.

VI. AMYLOID DISEASE.

Lardaceous Disease; Waxy or Bacony Infiltration; Amyloidosis.

Definition. — A secondary affection in suppuration and syphilis, characterized by the formation and deposition of amyloid material or lardacein in the walls of the arteries and the viscera.

Pathologically, lardaceous disease is regarded as a degenerative change involving certain elements in the blood and an infiltration in the tissues of the organs. The process is general or constitutional. It affects no particular organ locally, but many organs and tissues at the same time, though not to the same degree. The organs commonly affected are, in the order of frequency, the kidney, the spleen, the liver, the intestines, the adrenals, and the lymph-glands. The pancreas, thyroids, testis, œsophagus, and endocardium are less frequently involved. The amyloid material is deposited at first in the arterioles, and in certain anatomical structures or regions as the intermediate or hepatic artery zone of the liver lobule, the

Malpighian tufts and the cortex generally in the kidney, the Malpighian bodies in the spleen, and the arterioles in the mucous membranes. In many of the cases the material is distributed throughout the whole of the organ, with the result that the solid viscera are increased in bulk, sometimes to an enormous extent. This increase is sometimes, especially in the kidney, followed by contraction.

Etiology.—Suppuration, chronic or recent, with or without discharge, is present in the great majority of the cases. Pulmonary tuberculosis and disease of bone are the most frequent causes of suppuration antecedent to amyloid disease. Tuberculosis without suppuration does not appear to be a factor. On the other hand, suppuration, in the absence of tuberculosis or other specific constitutional infection, is a very common antecedent. Syphilis without purulent lesions must be recognized as a cause. Malaria is a possible cause, but its agency is still in question. As a rule, the suppuration has been prolonged, but there are exceptions to this rule. The amyloid process develops during the suppurative process, but may not cause recognizable clinical manifestations until after suppuration has continued for years, or not until after it has ceased. Males are more liable than females, not because of any differences incident to sex, but because they are more exposed to injuries and diseases attended by purulent lesions, and to syphilis. The predisposition associated with age is shown by the rarity of amyloid disease before ten and after fifty. It is most common between twenty and forty.

Symptoms.—There are general manifestations of amyloid disease irrespective of the visceral changes. The suppurative primary diseases have almost always produced changes that are characteristic or at least suggestive. These are manifest in the signs of advanced phthisis, the deformities of old empyema or bone disease, especially those forms which involve the spine and joints. Since such processes are attended with wasting, the pinched features, emaciated frame, and shrunken extremities are highly suggestive, especially as they are associated with a prominent or enormously distended abdomen due to the overgrown size of the amyloid viscera. A muddy pallor of the skin, dropsical effusions in the dependent parts, diarrhœa, polyuria, thirst, albuminuria, and great weakness complete the picture. The onset is insidious.

It is customary to describe the clinical manifestations of amyloid disease in the organs in connection with the various diseases of each; it seems, however, more appropriate and more useful for the purposes of the diagnostician to consider them here.

1. **Amyloid Kidney.**—The process is associated with wide-spread amyloid degeneration in other viscera due to suppurative diseases or syphilis. It has been attributed also in some instances to leukæmia, chronic lead intoxication, and gout. It is frequently associated with the chronic form of parenchymatous nephritis. The kidney is usually much increased in size; in exceptional cases it does not exceed the normal kidney in this respect. The surface is smooth and the stellate veins are conspicuous. The organ is firm. Upon section the cortex is thickened, the glomeruli distinct, and the pyramids of a deep red color. The iodine test shows a deep mahogany color most marked in the Malpighian tufts and straight vessels.

SYMPTOMS.—There are urinary features of importance. The quantity is increased, the color pale and transparent, the specific gravity low. Albumin is, as a rule, abundant; exceptionally there is a mere trace or it may be absent. Hyaline, fatty, and granular tube-casts are present, and occasionally the amyloid color-reaction may be obtained. Dropsy is usually present, but there are cases in which it does not occur. Diarrhœa is common. Increased arterial tension, cardiac hypertrophy, retinal lesions, and uræmia do not occur except in cases of amyloid degeneration affecting the small granular kidney.

DIAGNOSIS.—The renal symptoms alone have little diagnostic value. Their development in connection with prolonged suppuration or syphilis, and in association with an enlarged liver and spleen, and persistent diarrhœa, is highly suggestive.

2. **Amyloid Liver.**—The etiological relations of this condition are the same. It constitutes an important visceral manifestation of amyloid disease. The organ is large and may attain an enormous size. It is firm, dense, and resistant. Upon section the surface is pale and presents at the edges a slightly translucent appearance. It responds to the iodine test by the development of a mahogany-brown color in the affected areas. The capsule is smooth and the borders of the enlarged organ are rounded and blunt. Exceptionally the margins are sharp and well-defined. The enlargement is commonly uniform.

SYMPTOMS.—There are no characteristic hepatic features. Jaundice does not occur. The stools are sometimes light but not clay-colored. There are no signs of portal obstruction. The spleen is often enlarged.

DIAGNOSIS.—The history taken in connection with progressive enlargement of the liver which, upon palpation, yields the above signs, together with enlarged spleen, polyuria with or without albumin, and diarrhœa, constitutes positive evidence of the presence of amyloid disease.

3. **Amyloid Spleen.**—The organ is not usually greatly enlarged but can be readily recognized upon palpation. Its edges are thick and rounded and its consistence dense. Upon section the lardaceous infiltration is seen to affect especially the Malpighian bodies, which are prominent and glistening, giving rise to the appearance described as "sago spleen." In some cases the intervening tissue is more or less diffusely affected.

There are no special SYMPTOMS.

DIAGNOSIS.—A history of suppuration or syphilis, a cachectic state, emaciation, prominent abdomen due to coincident enlargement of the liver, urinary changes, and diarrhœa when present justify the assumption that an enlarged spleen is amyloid.

4. **Amyloid Disease of the Intestines.**—The blood-vessels of the entire digestive tract may be affected. More commonly the small intestine, especially the ileum, or the colon is the seat of the disease.

SYMPTOMS.—When slight in intensity or of limited extent the disease presents no features by which it can be recognized clinically. The one symptom of advanced or extensive amyloid degeneration in the intestines is persistent diarrhœa. The stools are variable in consistency and number. They are usually thin and liquid but without distinctive characters. Especially are they not bloody. They are not attended by colic or tenesmus and the abdomen is not sensitive to pressure.

DIAGNOSIS.—The recognition of the disease is difficult and uncertain, since diarrhœa without pain and tenderness and equally intractable may occur in various other intestinal diseases. The association of this symptom with the above-described clinical manifestations in a person suffering, or who has suffered, from prolonged suppuration, or who has syphilis, renders it in the highest degree probable that there is amyloid disease of the gut.

Prognosis in Amyloid Disease.—The outlook is doubly unfavorable. The antecedent disease is a frequent, the amyloid disease a common, cause of death. The highest mortality among the visceral forms relates to the kidneys, the next to the intestines. Extensive lardaceous degeneration of the liver and spleen may occur without special symptoms of importance, and without great impairment of health in addition to that caused by the primary disease.

IX.

THE DIAGNOSIS OF THE DISEASES OF THE DIGESTIVE SYSTEM.

(DISEASES OF THE MOUTH, TONGUE, GUMS, SALIVARY GLANDS, PHARYNX, TONSILS, AND ŒSOPHAGUS ARE CONSIDERED IN PART III.)

I. DISEASES OF THE STOMACH.

i. Acute Gastritis.

The following forms are recognized: toxic, phlegmonous, diphtheritic, parasitic, and dietetic.

1. **Toxic Gastritis.**—An intense form of inflammation produced by various irritant and corrosive poisons. It varies in degree according to the nature, concentration, and quantity of the poison, and the length of time it has remained in contact with the gastric mucosa.

Symptoms.—Sudden pain, nausea, retching, and vomiting occur. The vomitus in severe cases consists of blood-stained food remnants, mucus, shreds of mucous membrane, and the poison itself. Thirst and dysphagia are distressing. Later there is usually diarrhœa. Collapse comes on rapidly; the temperature, at first subnormal, rises later; and jaundice is not uncommon. Epigastric and abdominal tenderness may be followed by the signs of general peritonitis. Death occurs from the intoxication, or from exhaustion, convulsions, or suffocation. When recovery takes place, ulceration with stenoses or chronic gastritis supervene. The direct diagnosis depends upon the anamnesis, the evidences of the corroding poison upon the lips, mouth, and pharynx, the odor of the breath in certain cases, the presence of the vial or package which contained the poison, the analysis of the vomitus, the analysis of the urine, and the foregoing associated symptoms. The prognosis is, in the main, unfavorable.

It depends, however, upon the nature of the poison, its amount, the time elapsing before its removal or the administration of antidotes, the direct damage to the stomach itself, the intensity of the collapse, and the occurrence of peritonitis.

2. **Phlegmonous Gastritis.**—Acute diffuse or circumscribed suppurative inflammation of the gastric submucosa.

Etiology.—This is a very rare affection. The cases are primary, in which alcoholism appears to be a predisposing influence, and trauma, faulty diet, and various irritant poisons the exciting causes; and secondary, in which the various general febrile infections, sepsis, and peptic or carcinomatous ulceration constitute the primary disease. Streptococcus infection is most common. The colon bacillus may be present. Cases have been reported at every period of life between ten and ninety. Phlegmonous gastritis is four times as common in males as in females.

Symptoms.—In the circumscribed variety the symptoms are obscure. Pain may be absent. There may be a circumscribed tumor in the epigastrium, and vomiting of pus and blood. In the diffuse form the onset is sudden, with a rigor, severe prostration, and a rise of temperature to 104°–105° F. (40°–40.5° C.). Gastric symptoms speedily supervene. They consist of the urgent and continuous vomiting of pus, mucus, and bile, epigastric pain and tenderness, and, in rare instances, the signs of a fluctuating tumor. In the course of a brief period general abdominal tenderness, meteorism, and other signs of peritonitis usually appear.

The diagnosis has rarely been made *intra vitam.* The occurrence of the above symptoms in the course of a severe general infection or sepsis would be suggestive. The differential diagnosis concerns perigastritis following peptic ulcer, circumscribed peritonitis, acute pancreatitis, cholecystitis, and toxic gastritis. The prognosis is in the highest degree unfavorable. Of the reported cases 95 per cent. have terminated fatally.

3. **Diphtheritic Gastritis.**—Pseudomembranous inflammation of the gastric mucosa occurs as a true diphtheria of the stomach in cases of invasion by the Klebs-Löffler bacillus in diphtheria of the œsophagus, throat, or upper respiratory passages, but it is a very rare complication. More commonly it occurs as a complication of other infectious diseases, as enteric fever, pneumonia, the exanthemata, and sepsis. In children tuberculosis is often the primary infectious disease. A condition closely resembling diphtheritic gastritis may be produced by corrosive poisons. The characteristic lesion is the presence of the pseudomembrane, which may be diffuse, patchy, or arranged in irregular streaks extending from the cardia to the pylorus. The superficial layer is formed by a coagulation necrosis. The bacteriological findings vary. Streptococci, tubercle bacilli, and Klebs-Löffler bacilli have been more commonly isolated. The symptoms are not characteristic. Vomited membrane may have been dislodged from the upper air-passages, or the pharynx or œsophagus. The diagnosis, with a very few exceptions, has not been made during life.

4. **Parasitic Gastritis.**—Inflammation of the stomach as the result of infection by various pathogenic organisms by way of the blood and lymph stream—*infectious gastritis*—is of common occurrence. This form occurs in various septic conditions, enteric fever, pneumonia, and the exanthemata.

The presence of moulds and yeasts in lesions of the gastric mucosa renders it probable that, under certain circumstances, those agencies may cause or aggravate such conditions as inflammation, erosion, and ulceration. Among the growths obtained by lavage or observed post mortem are mucor mycelia, forms of leptothrix, thread fungi, Oidium lactis and albicans, favus, and Penicillium glaucum. The symptoms are not characteristic and their dependence upon the presence of fungi is uncertain. The subject is of anatomical rather than of clinical importance.

5. **Dietetic gastritis** is of common occurrence and great clinical importance.

Etiology.—Individual and family predisposition are common. Gouty subjects are peculiarly liable to subacute and acute gastritis.

The exciting causes comprise excesses at table, highly seasoned foods, unwholesome, indigestible, or tainted articles of food, large amounts of unduly cold or hot fluid, and alcohol.

Symptoms.—Habitual sensations of weight and gastric distress characterize the subacute forms. The more acute variety is associated with colicky pain, distention, fulness, and vomiting, which are often relieved by removing the offending material. In very acute cases the inflammation persists with the pain, colic, and hypersecretion of mucus and gastric fluid. Under such circumstances vomiting does not always afford relief. Bile frequently appears in the vomited matter. The early vomitus may contain undigested food, hydrochloric, lactic, and butyric acids, and have a butyric odor; the subsequent vomitings consist principally of water and mucus and are of a light green color. Diarrhœa may occur. Only in very severe cases is there fever. The tongue is coated, the pulse and general condition are usually good. Improvement commonly takes place in a few hours: occasionally an attack lasts two or three days. Slight fulness in the epigastrium and tenderness on pressure over the stomach are common in severe cases. Two or three days of persistent vomiting may be followed by retraction of the abdomen.

Diagnosis.—DIRECT.—Epigastric pain, nausea, and vomiting, following or referable to some indiscretion of the diet, justify a diagnosis of simple acute gastritis, particularly in the absence of fever or marked general symptoms. Fever, rapid pulse, and prostration, with persistent vomiting, should arouse the suspicion of an acute infection, cholecystitis, or intestinal or pyloric obstruction.

DIFFERENTIAL.—If associated with fever at the onset, acute gastritis must be differentiated from, (a) *various infections*, as scarlet fever, meningitis. In acute gastritis the constitutional symptoms are less severe, there is absence of local or other phenomena peculiar to the specific infections, less intense pyrexia, and, as a rule, early improvement. (b) *Mild or Abortive Enteric Fever.*—Acute gastritis has a more rapid onset, an abrupt rise of temperature rather than the step-like rise, and rose spots, bronchitis, enlarged spleen, and diarrhœa do not occur. (c) Severe pain may suggest *gall-stone colic*, but the pain is usually less severe; the vomiting and pain of gastritis are more continuous; chilliness or a chill does not occur; and the general symptoms are less pronounced. Marked icterus is absent, though the possibility of an associated duodenitis with catarrhal jaundice [illegible] Gastric Crises of Locomotor Ataxia.—The Argyll-Robertson pupil, ataxia, and loss of knee-jerks are distinctive. If associated with persistent vomiting, acute gastritis must be differentiated from [illegible]. In both of these conditions the other signs are apt to be marked, while general symptoms are present, and the history of the condition is different.

ii. Chronic Gastritis.

Chronic Gastric Catarrh.

Definition.—*Chronic inflammation of the gastric mucosa, giving rise to mucus in excess and alterations in the gastric juice, associated with marked disturbance of the digestion and weakening of the muscular coat.*

Etiology.—In many families there is a predisposition to chronic gastritis—chronic dyspepsia. Many constitutional diseases act as predisposing factors. Careless habits of eating and drinking, and the persistent use of gastric irritants are the chief causes. Chronic congestion of the stomach, the result of heart disease or hepatic cirrhosis, commonly ends in chronic gastritis, and most of the local gastric diseases, as cancer and ulcer, bring about the same condition.

Symptoms.—Headache, drowsiness, dizziness, inaptitude for work, and sallow complexion are common general symptoms. A coated tongue, bad taste in the mouth, aphthous stomatitis, chronic pharyngitis are usually present. A variable and capricious appetite, occasional repugnance for food, burning sensations in the œsophagus and at the cardiac end of the stomach—heart-burn—are early symptoms. Distress and weight in the stomach, oppression, distention, and actual pain (more particularly after meals), belching of gas, and eructations of bitter fluid soon occur if the condition persists. Nausea may be an early symptom. Vomiting usually appears late and occurs soon after eating or in the morning before food. That which occurs after a meal contains mucus in excess. Undigested food, indicating retention, fermentation of the carbohydrates, diminished amount of free HCl and ferments (or none at all), and traces of lactic and butyric acids are characteristic of delayed vomiting in rare cases. The vomitus has a sour odor. That occurring early in the day is composed of small amounts of thick mucus.

The Ewald test-breakfast may be below the normal amount, or, where dilatation has occurred, it may be in excess. At times it is brought up with difficulty owing to the thick grayish mucus. The toast or bread may have passed out of the stomach completely, or it may remain in varying amounts mixed with mucus and poorly minced. Early in the disease the free HCl may be normal in amount or even slightly increased; later it is diminished or absent. Lactic acid is not usually present. Both peptic digestion and the milk-curdling reaction for rennin may be absent. Further proof of muscular weakness and excess of mucus follows the washing of the stomach after the removal of the test-meal. The water used must often be removed by suction or siphonage instead of gushing back as is the case when poured into a normal stomach. It contains numerous mucus flakes, which accumulate in a stringy mass on the surface. Inflation often shows dilatation at the lower border, reaching the level of the umbilicus.

Differential Diagnosis.—1. *Ulcer of the Stomach.*—In chronic gastritis the pain is less intense and more continuous, less aggravated after food, and more diffuse. The decline in general health is less marked and rapid, and there is absence of hæmatemesis or blood in the stools. A coated tongue is common in chronic gastritis, while a clean red tongue is usually present in the hyperacid conditions in which ulcer occurs. There is no point of extreme local tenderness in chronic gastritis and no evidence of obstruction and muscular hypertrophy such as are sometimes demonstrable in chronic ulcer.

The results of gastric analysis in chronic gastritis and peptic ulcer are in strong contrast (see Gastric Ulcer). The chemical and physical conditions of the vomitus are, however, much modified when gastritis and ulcer are associated, as not rarely happens.

2. *Cancer of the Stomach.*—The differential diagnosis in the absence of tumor is at times almost impossible. The loss of flesh and strength in chronic gastritis is rarely so rapid as in cancer. A protracted course is in favor of gastritis. Pain and vomiting are less marked, less persistent, and more amenable to treatment in chronic gastritis than in carcinoma ventriculi. For differences in the results of gastric analysis see Cancer of the Stomach.

3. *Pernicious Anæmia with Gastric Symptoms.*—The gastric condition is usually a chronic anacid gastritis. The differentiation rests upon the comparatively rapid and extreme deterioration of health and the blood examination.

4. *Gastric Neuroses.*—The conditions are frequently associated. The irregular dietetic habits and despondency characteristic of neurasthenia often cause chronic gastritis. Chronic gastritis is more amenable to dietetic treatment. Articles of food, such as tea, coffee, alcohol, and hot stimulating drinks, which aggravate the symptoms of gastritis often allay the subjective symptoms of a neurosis. Regulation and restriction of the diet is usually beneficial in gastritis; not so, as a rule, in the neuroses. Fermentation and consequent flatulence and belching are more pronounced in gastritis; the flatulence of the neuroses is largely due to air swallowed or worked into the stomach. Anæmia is more marked in chronic gastritis than in the gastric neuroses.

The analysis of the stomach contents in chronic gastritis shows mucus in excess, lack of free acid, and fermentative changes; in the neuroses a normal or excessive acidity without mucus.

Prognosis.—Cases of chronic gastritis seen early and systematically treated get well. Advanced cases are comfortable only on a non-irritating diet and require continuous therapeutic management.

iii. Dilatation of the Stomach.

Gastrectasis.

Enlargement of the stomach, usually attended with weakening and thinning of the various coats, and supersecretion. Acute and chronic dilatations are recognized.

Acute Dilatation.—The **etiology** is not well understood. The condition is that of paralytic distention of the organ. Among the assigned causes are local and general debilitating conditions, trauma, general anæsthesia and other forms of narcotism, and dietetic errors, especially excesses in beer. Post-mortem findings point to acute dilatation as a terminal condition in acute illness, especially pneumonia and cardiac disease.

Symptoms.—Sudden collapse symptoms, the vomiting of enormous amounts of fluid, moderate pain, and pressure symptoms from distention, as dyspnœa and cardiac oppression, constitute the symptom-complex. The fluid reaccumulates as fast as vomited, the pylorus remaining in a condition of spasmodic closure.

Physical Signs.—The physical signs of enormous distention of the stomach are present. The enlargement is often such that its true outline is lost. Peristalsis is not observed. The removal of large amounts of fluid, as much as eight or nine pints, by the stomach-tube and subsidence of the distention are, as a rule, followed by only transient relief of symptoms. This fluid is dark brown or clear and contains traces of blood and bile. Its odor is foul but not fecal. HCl is present in diminished but variable amounts, and there are in some cases traces of lactic acid. Constipation, oliguria, and torturing thirst not easily allayed are among the symptoms.

Diagnosis.—DIRECT.—Acute symptoms of collapse and oppression, epigastric pain, profuse vomiting of dark or clear fluid, marked distention of the stomach (the signs indicating fluid rather than gas), and the rapid reaccumulation of fluid after removal are the diagnostic criteria. The condition is not always recognized during life.

DIFFERENTIAL.—*Acute Obstruction of the Pylorus and Upper Duodenum.*—Active peristalsis, moderate distention, and a relatively small accumulation of fluid, not immediately returning after withdrawal by vomiting or the tube, point to pyloric obstruction.

Acute Obstruction of the Duodenum below the Entrance of the Bile and Pancreatic Ducts or of the Intestine Still Lower Down.—The presence of considerable quantities of bile and pancreatic fluid in the vomitus or material removed would be significant. A fecal odor in the vomitus would suggest obstruction lower down.

Prognosis. The condition is almost always fatal.

Chronic Dilatation.—**Etiology.**—There may be a family tendency. General and gastric debilitating conditions constitute predisposing influences. Habitual overdistention from excesses in eating or drinking may induce dilatation without pyloric obstruction being present. The majority of cases arise from obstruction at the pylorus, from cancer, ulcer with cicatricial contraction, adhesions to the gall-bladder, kinking of the duodenum, or stretching of the gastric walls already weakened by chronic gastritis.

Symptoms. The condition may exist for an indefinite period without symptoms. In cases occurring independently of cancer or ulcer the loss of weight and strength is less marked and anæmia and cachexia may not be present. Thirst, constipation, headache, torpor, and dizziness are common symptoms. Persistent dyspepsia, flatulency, belching, eructations, nausea, and eventually recurrent vomiting of large amounts of fermented magested food characterize the course of the affection. The dyspeptic symptoms resemble those of chronic gastritis, and comprise pain and weight immediately or shortly after eating, or hyperacidity four or five hours after the meal. Retention vomiting is common. Pain, in the absence of hyperacidity, is not usual except in carcinoma or ulcer.

Physical Signs.—Thinning of the abdominal wall is usually present with the general wasting. The skin is often dry and harsh. *Inspection.*—The abdomen may show general fulness, or the epigastrium, hypochondria, or supra-umbilical region may be especially prominent. The stomach may be distinctly outlined, its greater curvature sometimes reaching into the pelvis. Displacement of the pylorus and lesser curvature may outline the whole stomach as a dilated sac lying in great part below the umbilicus. Peristalsis from left to right is easily distinguished. In obstructive cases it ends in the hypertrophied pylorus or tumor mass. On palpation "clapotage" or splashing of fluid is readily obtained when the dilatation reaches the umbilicus. The cushiony condition of the air-distended stomach is readily felt. Percussion yields tympany. In the erect posture the lower limit of the stomach can be determined by the dulness of the contained fluid. Auscultation reveals nothing important.

Inflation with air through the tube or by CO_2 shows distention reaching to the umbilicus or below it. The whole stomach can often be outlined. Usually, however, the outline obtained does not include the lesser curvature. *Auscultation.*—The cask-like tympany produced in the distended stomach by quickly compressing or relaxing the bulb of the tube, or by percussing with the finger, is readily heard with the stethoscope and can be followed over the whole stomach, being lost on passing the stethoscope's bell away from the immediate area of the stomach. Inflation with water has no practical value.

Large bismuth meals are of service in photographing the outline of the stomach by the Röntgen rays. Both recumbent and erect positions must be employed. Transillumination can at best only give us the lower border of the stomach.

The vomitus is variable. That of dilatation consequent upon chronic gastritis shows mucus, usually absence of free HCl, marked fermentation; that associated with cancer frequently has an odor of putrefaction in addition to that of fermentation; the vomitus in which much free HCl is present is rarely foul, the HCl acting as an antiseptic. The amount may be enormous.

The test-meal removed in an hour may come away with several hundred c.c. additional fluid. Much mucus is present in chronic gastritis. The toast is poorly minced and almost wholly returned. Free HCl may be present in excess, even in very advanced cases, or may be absent, as in cases of cancer or gastritis. Lactic acid may be persistently present in cancer. It tends, however, to disappear under systematic lavage. The weakness of the gastric muscle is shown by the necessity of using suction and siphonage to remove the contents of the stomach.

Diagnosis.—DIRECT.—Persistent dyspepsia, flatulence, eructations, and vomiting of large amounts of long-retained fermented food remains are suggestive. Distention, the outlining of an enlarged displaced stomach visible peristalsis, and an abnormally large amount of gastric contents after a test-meal are conclusive. Inspection alone may make the diagnosis. A pyloric tumor with its associated signs suggests gastrectasis.

DIFFERENTIAL.—The anamnesis distinguishes dilatation of the stomach from cystic conditions of the mesentery, gall-bladder, and ovary, and from chronic dilatations of the colon. The last is not associated with "retention vomiting;" it shows the intestinal outlines and peristalsis from right to left, and can be reduced by passage of the rectal tube. The use of the stomach-tube is of great diagnostic importance.

Prognosis.—Dilatation of the stomach, when recent and unattended by obstruction at the pylorus, and not excessive, may permanently subside. Many cases require systematic lavage. Operative measures are required for the relief of marked dilatation with pyloric obstruction.

iv. Gastric Ulcer.

Definition.—Ulceration of the gastric mucosa in any part of its extent, due to nutritional disturbance in a circumscribed region and the action of the gastric juice. The necrotic areas may occur in the lower end of the œsophagus and in the duodenum as low as the papilla of Vater. They involve the various coats of the stomach and sometimes perforate. They are usually round or oval, with clean-cut edges in the acute and irregular indurated borders in the chronic cases.

Etiology.—Anæmia and chlorosis predispose to the affection. The disease is more common in the working classes. Heredity has some influence. Hyperacidity of the gastric juice is usually present. Trauma or large superficial burns may be direct causes. Septic cases occur.

Symptoms.—General symptoms are often absent. Loss of weight and strength, and progressive anæmia, often of extreme degree, are common. Constipation is usual. Simple dyspepsia, distress and fulness after eating, flatulence, and belching are common symptoms. In such cases there is often normal acidity. In the more severe cases the dyspepsia is more intense; nausea and vomiting occurring three or four hours after eating, or severe agonizing pain on taking food and lasting two or three hours or more, or not beginning until the height of gastric acidity is reached, two or three hours after the meal, are frequent symptoms. At times the pain is gnawing in character, more marked when the stomach is empty, and relieved by food. Cases of this type usually show a high degree of acidity. The pain is referred to the epigastrium, often radiating to the back and sides. Vomiting may give relief. Hemorrhage is common and may be the first symptom. The blood may be passed either by the mouth or bowel. Concealed hemorrhage may occur with characteristic symptoms. Recurrent hemorrhage may end fatally. I have seen a single profuse hemorrhage followed by death in a man in apparent health. Small continued hemorrhages may only be recognized by blood tests. The testing for occult blood in the stools is most important in such cases.

The symptoms may continue for years. Perforation of the stomach and peritonitis may be the first clear indications of an ulcer. The local symptoms in old cases with pyloric obstruction are those of gastrectasis, flatulence, dyspepsia, nausea, and retention vomiting. In early cases, in the absence of obstruction there may be nothing to indicate disease of the stomach. In others tenderness in the epigastrium, mostly acutely localized, may be the only symptom. During the inactive stage of an ulcer near the pylorus or in the duodenum temporary signs of pyloric obstruction may develop. These are probably due to spasmodic closure of the pylorus and the infiltration of the neighboring tissue. Fulness of the epigastrium, the outline of the enlarged stomach, and visible peristalsis may be evident. The thickened and hypertrophied pylorus can sometimes be felt during contraction. Tenderness over the pylorus is often marked. In chronic cases the physical signs indicate tumor formation due to scarring or puckering of the ulcerated area, thickening and muscular hypertrophy, and hour-glass contraction. These are often much more manifest upon inflation.

Gastric Analysis.—The vomitus or gastric contents from the fasting stomach, usually thin, watery, and light green in color, frequently contain large amounts of free HCl. The gastric contents removed after the ordinary test-meals are usually in excess of the normal and generally contain some mucus and a small amount of well-mixed food residue. They show a high total acidity, a high degree of free HCl, pepsin and rennin, no lactic or butyric acid. Traces of blood by chemical tests are common. Under the microscope small round cells and pus-cells can be demonstrated in most cases when the ulceration is active. The red blood-cells are, as a rule, disintegrated by the high acidity. In the older cases, the same high percentage of free HCl and hypersecretion is found, together with mucus from the coexisting chronic gastritis. The vomitus in cases with obstruction is sometimes of enormous quantity, containing free HCl and mucus. It is acid in odor but rarely foul. The high total acidity and free HCl are occasionally absent in well-marked cases of gastric ulcer.

Diagnosis.—Direct.—The diagnosis rests upon persistent dyspepsia, anæmia with loss of weight and strength, marked circumscribed tenderness in the epigastrium, a high degree of gastric acidity, and occult blood in the fæces, gastric contents, or vomitus. In marked cases intense pain after eating, vomiting, nausea, the sudden appearance of hæmatemesis or tarry stools, with rapidly developing weakness and anæmia occur. In long-standing cases the symptoms and signs of pyloric obstruction or gastric malformation are significant.

Differential.—1. *Chronic Gastritis.*—In ulcer the high degree of acidity of the gastric juice, the presence of occult blood in the gastric contents and fæces, the more marked tenderness or local pain are distinctive. A well-defined hæmatemesis or the passage of blood per rectum is conclusive. 2. *Gastric Superacidity.*—Loss of weight is common to both; anæmia is more common in ulcer. Localized pain is more marked in ulcer; nausea and vomiting, pain after eating, hæmatemesis, tarry stools, occult blood in the fæces or gastric contents are not symptoms of superacidity. Relief of pain on taking food is suggestive of simple hyperacidity, in which signs of obstruction and marked retention do not, as a rule, occur. 3. *Carcinoma of the Stomach.*—Sudden onset with hæmatemesis, hemorrhage from the bowels, and perforation are suggestive of ulcer. The history of many years' duration is against cancer. Cachexia develops rapidly in cancer, is unusual in ulcer. Hæmatemesis or hemorrhage from the bowels is common in ulcer, as is hyperacidity of the gastric juice, while the presence of lactic and butyric acids is unusual. Absent or diminished free HCl is common in cancer, the presence of lactic and butyric acids frequent.

The signs of a tumor are, as a rule, present early in cancer, but late if at all, and associated with scar formation, in ulcer. Pyloric obstruction is an early condition in cancer.

v. Cancer of the Stomach.

Carcinoma Ventriculi.

Definition.—Cancerous infiltration of the stomach walls, occurring most frequently toward the pylorus and about the lesser curvature.

Etiology.—The predisposition has been thought to be hereditary. A chronic gastric ulcer not infrequently becomes the seat of a carcinoma. Middle and advanced life is the time of common occurrence, but there is no "age of cancer." Irritation of the stomach by improper diet, injury, or pressure from without have all been considered as exciting causes.

Symptoms.—Rapid anæmia, loss of strength and weight, and early developing cachexia in a middle-aged person are suggestive. Persistent distress in the stomach after eating or even when the stomach is empty, often amounting to intense pain, is an early symptom. Nausea and anorexia soon follow. Vomiting may be an early symptom. If there is marked obstruction at the pylorus with consequent dilatation, the vomitus may be of large volume and foul odor. The vomiting of altered blood ("coffee-ground vomit") and passage of small amounts of blood in the stools occur. Constipation is often present.

Physical Signs.—Inspection may show nothing, especially in early cases. Metastasis may be seen in the supraclavicular fossa or beneath the skin of the abdomen. Fulness of the epigastrium and left hypochondrium, visible outline of the stomach, displaced and distended visible peristalsis (usually from left to right), and distinct contracting tumor may be observed. Inflation will often produce peristalsis and bring a tumor mass, not otherwise recognizable, into evidence. Inflation will often show the abnormal size and position of the stomach. Palpation also may reveal nothing at first, especially if the tumor is posterior or adherent. Careful and repeated palpation, aided by "dipping" and slapping the abdomen, may reveal the presence of a new growth. Inflation aids palpation at times by bringing the tumor into the field of examination. The movements of the tumor, with respiration, with movements of the body, with the aortic pulsation, and from the muscular contraction, should be noted.

GASTRIC ANALYSIS.—Vomitus or a test-meal containing traces of blood and showing no free hydrochloric acid and a lowered total acidity must be regarded as suspicious in all cases. Lactic acid is usually to be found in the vomitus after a mixed meal if there is any degree of stagnation, and in the test-meals given in late cases. After a few days' lavage it diminishes and may disappear. Butyric acid will be found in the vomitus under the same conditions as lactic acid. Pepsin and rennin are usually to be found.

Pieces of cancer tissue should be looked for. Oppler-Boas bacilli are found more frequently in the vomitus of cancer than in any other condition. They disappear quickly under lavage. Pus-cells, sarcinæ, yeast cells, and many bacteria may be seen. Free HCl is not always absent, and may even be present in excess. With obstruction at the pylorus the amount of material obtained after a test-meal will be excessive.

Diagnosis.—DIRECT.—Persistent dyspepsia, pain, loss of weight and strength, anæmia, a tumor in the gastric area, signs of displacement and dilatation of the stomach, absence of free HCl, presence of lactic acid, and Oppler-Boas bacilli in the test-meal or vomitus, an excessive amount of vomitus, and gastric contents with evidences of stagnation are the main points in the diagnosis.

DIFFERENTIAL.—*Chronic Gastritis* (see Chronic Gastritis). *Pernicious Anæmia.*—Gastric symptoms with absence of free HCl often occur, but this condition may be distinguished by the blood examination. The anæmia of cancer rarely falls as low as in pernicious anæmia. The color index is low in cancer. There may be a slight leucocytosis, and the presence of megaloblasts is rarely noted. In general the blood picture is that of a secondary anæmia. Hæmatemesis, tarry stools, the reaction for occult blood in the fæces and gastric contents are indicative of a gastric lesion. *Severe Secondary Anæmia with Gastric Symptoms.*—The presence of lactic acid in the test-meal, the absence of free HCl, the presence of a tumor, and occult or gross blood in the gastric contents and fæces point to cancer.

Prognosis.—Unless an early diagnosis makes a total extirpation of the tumor possible the course is steadily downward. Gastro-enterostomy may be of service. Temporary improvement usually follows the institution of lavage in cases that have marked obstruction at the pylorus.

vi. Hypertrophic Stenosis of the Pylorus.

Definition.—Obstruction of the pylorus from hypertrophy of the musculature and the submucous tissue.

Etiology.—Most of the reported cases are in infants and are congenital; even those reported in adults are considered to be late developing cases. The same conditions that induce pyloric spasm, namely, nervous and direct irritation, are possible exciting causes.

Symptoms.—The symptoms and physical signs are those of obstruction at the pylorus.

Diagnosis.—DIRECT.—Symptoms of obstruction at the pylorus, visible peristalsis, and palpable pyloric tumor occurring in infants are diagnostic of the condition. In adults the condition can hardly be distinguished from simple pyloric spasm.

DIFFERENTIAL.—There is no other condition which can be confused with hypertrophic stenosis in infants except pyloric spasm. Relief following general and dietetic treatment would justify the diagnosis of the latter affection. True hypertrophic stenosis in adults cannot always be distinguished from other forms of pyloric tumor or obstruction. It may be differentiated from spasm of the pylorus by the fact that the latter is not followed by permanent lesions, as tumor or dilatation. Its occurrence in early infancy is against its being confounded with either cancer or ulcer. In adult cases the absence of anæmia and wasting, cachexia, hemorrhage, and occult blood are of diagnostic value.

vii. Tuberculosis of the Stomach.

Tuberculous ulceration of the stomach is discussed under Tuberculosis.

viii. The Gastric Neuroses.

The gastric neuroses, nervous disorders of the stomach, "nervous dyspepsia," include a host of conditions many of which are explained and described by their names. With a few exceptions, which comprise the nervous disorders of secretion, they are characterized by the predominance of symptoms over physical signs. Disorders of sensation, secretion, and of the motor function are recognized.

1. **Disorders of Sensation.**—(a) **Gastric Hyperæsthesia.**—A condition of oversensitiveness of the gastric mucous membrane. ETIOLOGY.—The general nervous state is the predisposing influence. Any article of food may be the exciting cause. Overacidity of the gastric juice in an empty stomach is a frequent cause. SYMPTOMS.—Neurasthenia or hysterical symptoms are common; wasting or anæmia is infrequent. Gastric distress, gnawing sensations, burning in the stomach immediately or shortly after taking food or when the stomach is empty are the main features. The sensation seems to be that of feeling the stomach doing its work. Stimulants or condiments often relieve the symptoms. There is often tenderness over the stomach. The gastric contents after a test-meal usually yield normal results. DIAGNOSIS.—*Direct.*—Hysterical or neurasthenic manifestations, gastric symptoms occurring before there has been time for acid to accumulate or for fermentation to begin, negative physical examination and negative results of the gastric analysis are suggestive. *Differential.*—*Gastric Hyperacidity.*—The gastric analysis sometimes shows slight hyperacidity; as a rule, the analysis serves to distinguish simple hyperæsthesia from hyperacidity. The symptoms in hyperacidity develop later—three to four hours after a meal. *Chronic Gastritis.*—The sensory symptoms in some forms of chronic gastritis are practically the same but they are definitely related to certain articles of diet. The gastric analysis of chronic gastritis shows distinct differences (see Chronic Gastritis). *Gastric Ulcer.*—Hyperæsthesia rarely produces anæmia or wasting. There is no vomiting, no hæmatemesis or tarry stools, no signs of pyloric obstruction, no occult blood in fæces or gastric contents, and usually no hyperacidity of the gastric juice. *Gastric Cancer.*—Absence of anæmia and cachexia, absence of vomiting, hemorrhages, gross and occult, absence of a tumor or signs of dilatation and obstruction, a normal condition of the gastric juice favor the diagnosis of hyperæsthesia.

PROGNOSIS.—Recovery depends upon the course of the general nervous condition. Persistence of the symptoms may reduce the patient to an extreme degree.

Anorexia nervosa is considered in the sections on hysteria and neurasthenia. Excessive hunger, bulimia, absence of the sense of repletion—acoria—are often merely symptoms of hyperacidity. They may occur, however, with a normal condition of the gastric juice.

(b) **Gastralgia; Gastrodynia.**—Severe paroxysmal pain in the stomach, often periodic, not referable to ulcer, cancer, or organic nervous disease (gastric crises of tabes). This is often symptomatic of excessive acid secretion. ETIOLOGY.—The predisposing influence is always neurotic. An exciting cause cannot always be found, the gastralgic attacks occurring more or less independently of food ingestion. SYMPTOMS.—There are almost always general nervous symptoms. Acute grinding pain in the epigastrium and gastric region, which radiates to the back, is the main feature. Vomiting is unusual. Pressure may or may not relieve the pain. Tenderness on deep pressure is the rule. DIAGNOSIS.—Acute periodic painful attacks with no evidence of gastritis, ulcer, hyperacidity, or of locomotor ataxia. Marked general nervous symptoms are suggestive. The *differential diagnosis* concerns cancer, peptic ulcer, the gastric crisis of tabes, and biliary colic. *Cancer and Ulcer.*—The nervous symptoms and the general condition of the patient in gastralgia are important diagnostic points, since many cases of gastralgia show absence or even excess of HCl in the gastric juice. Negative occult blood-tests of the fæces and gastric material, absence of anæmia and cachexia, of tumor mass and physical signs are in favor of gastralgia. *Gastric Crisis of Tabes.*—Absence of the knee-jerk and other tabetic phenomena are conclusive. *Gall-stone Colic.*—The distinction is sometimes extremely difficult, since the pain of gall-stone colic may be referred to the epigastrium, subsequent jaundice may not occur, and the calculus may be masked in the fæces or not passed from the duct. PROGNOSIS.—The prognosis in gastralgia is usually favorable. It depends upon the course of the general nervous condition.

2. **Disorders of Secretion.**—Three main varieties are recognized: supersecretion, hyperacidity of the gastric juice, and hypoacidity and anacidity.

Either hyper- or hypoacidity may be associated with supersecretion; hyperacidity and supersecretion is the more frequent combination.

(a) **Supersecretion; Gastrosuccorrhœa.**—A condition in which excess of gastric juice is secreted continuously,—*Reichmann's disease.*—or periodically,—*Rossbach's disease; nervous gastroxynsis,*—usually in association with a certain degree of dilatation of the stomach from a general relaxation of its muscular tone. ETIOLOGY.—General neurasthenia is the main predisposing cause. Stimulants and tobacco are occasionally exciting causes. SYMPTOMS.—Neurasthenic symptoms are almost always present. In the periodic form there occurs suddenly the accumulation of large amounts of fluid, associated usually with gnawing distress or even pain, eructations or vomiting of a clear watery fluid ensue and may persist for several days. The secretion is enormous and independent of food, the symptoms often occurring in the early morning. If hyperacidity is present the irritation of œsophagus and pharynx may occasion great distress. The continuous form ends frequently in dilatation of the stomach from the persistent pyloric spasm so often present and the weight of the accumulating fluid upon the relaxed muscles. PHYSICAL SIGNS.—Dipping palpation may give clapotage. Percussion in the erect position may show a level of fluid in a position lower than normal. The stomach-tube, passed in the morning or at other times when the stomach is presumably empty, may bring away as much as 200 to 300 c.c. of fluid, often highly acid from free HCl. Inflation shows a moderate degree of dilatation. Spasm of the pylorus and peristalsis may be seen and felt. The test-meal may be returned after an hour with an accumulation of several hundred c.c. of a high or normal degree of total acidity and with a large or normal amount of free HCl. Mucus is not in excess; lactic acid is not present. In late cases of continuous supersecretion the condition is practically that of dilatation. DIAGNOSIS.—*Direct.*—The continuous or periodical presence in the stomach of an excess of fluid having the above characters and its accumulation independently of the stimulation of food are the main characteristics of the condition. *Differential.*—*Acute Gastritis.*—Acute gastritis usually has a distinct and recognizable etiology. The fluid vomited is smaller in quantity, usually anacid, and contains mucus. *Acute Dilatation.*—In acute dilatation the general symptoms are marked from the outset. The condition is much more serious, and neither vomiting nor lavage has much effect upon the course of the attack. *Chronic Dilatation.*—Supersecretion often ends in dilatation. Simple supersecretion in its early stages, however, has a different history. Retention vomiting and the physical signs of marked dilatation do not occur. PROGNOSIS.—This depends upon the nervous state; as a rule it is good.

(b) **Hyperacidity; Hyperchlorhydria.**—Excess of free HCl in the gastric juice. ETIOLOGY.—Hysteria and general neurasthenia predispose to the condition. Irregular habits of eating, stimulants, tobacco, and an excess of proteid nourishment are common exciting causes. SYMPTOMS.—The general nervous symptoms of the underlying condition are present. Headache, hunger, and constipation are common. Gastric hyperæsthesia is frequently present. Supersecretion often coexists. Gnawing distress, burning, or severe pain developing two to four hours after eating, relieved in turn by eating proteid food, are local symptoms. When vomiting occurs the symptoms are usually relieved. The tongue is commonly clean, red, and moist, and there is epigastric tenderness. The test-meal is usually expelled vigorously and in excessive quantity, as some degree of supersecretion is almost always present. The digestion of the starch is retarded. The total acidity, instead of a normal 40, may reach 120 or 150. Free HCl may be as high as 90 to 110. If the condition has been persistent mucus may be present. Lactic acid is absent. Many round epithelial cells showing mitosis may at times be seen under the microscope. The vomitus on account of its high HCl percentage does not readily ferment. Bacteria are not present in any large numbers. Meat digestion can be shown to be rapid. DIAGNOSIS.—*Direct.*—Distress two to four hours after eating [illegible] relieved by taking food, and a high degree of free acid in the vomitus [illegible] chemical examination of the gastric contents constitute the basis of the diagnosis. *Differential.*—*Ulcer.*—Hyperacidity [illegible] present [illegible] in ulcer. Pain immediately after eating is usual in ulcer and vomiting is more common. Local tenderness or pain, a history of hæmatemesis or tarry stool, loss of weight, anæmia, occult blood in gastric contents and fæces are important. The two conditions often coexist, the ulcer being unrecognized. Laboratory methods are imperatively required in doubtful cases. *Cancer,* with normally acid or hyperacid gastric juice. The general symptoms, as loss of weight, cachexia, anæmia, the persistence of local symptoms, as vomiting and pain, are distinctive of cancer, while occult blood in the fæces or gastric contents, or gross hemorrhages are against the diagnosis of simple hyperacidity. The diagnosis may, in rare cases, be impossible until a palpable tumor is detected or a dense shadow appears in the skiagram. PROGNOSIS.—The condition may last for years. Relief under appropriate treatment is the rule.

(c) **Hypoacidity ; Anacidity ; Hypochlorhydria.**—Conditions of the gastric juice in which the free HCl is of low value, or lacking. ETIOLOGY.—Hysteria and depressed nervous states are predisposing causes or even the exciting cause. It is to be remembered that the Ewald test-meal may fail to provoke much secretion. Low HCl in organic disease occurs as follows: in subacute and chronic gastritis, early carcinoma, dilatation of the stomach, and various chronic diseases of the abdominal viscera. SYMPTOMS.—Symptoms are frequently absent. Fermentation, flatulence, a feeling of fulness, or other manifestations of indigestion occur. Diarrhœa, anæmia, and pallor are more common than in the other secretory disorders. Local signs are absent. A coated tongue is usual. Inflation reveals nothing. The test-meal is brought away either as normal amount, or thick and pasty from lack of fluid. Starch digestion has progressed. There is persistently a low degree of free HCl, and the total acidity is also low, or there may be no reaction for either acidity or free acid. Lactic acid is absent. Mucus is not in excess Pepsin and rennin are present. When the gastric secretion is completely absent, as occurs in rare instances, the condition is designated "achylia gastrica."

DIAGNOSIS.—*Direct.*—Persistent absence of free HCl, or its presence in an abnormally low percentage half an hour or one and a half hours after the test-meal, is suggestive. General nervous symptoms and the absence of localizing gastric phenomena are important. *Differential.*—*Chronic Gastritis.*—The relation of symptoms to food is more marked in gastritis. Dyspeptic symptoms are more marked, vomiting and nausea more pronounced in gastritis, and mucus in excess is common. Nervous symptoms are not especially prominent in gastritis. *Cancer.*—In cancer constitutional symptoms are more marked. Pain, vomiting, nausea, occult and gross hemorrhages, anæmia, wasting, physical signs of tumor and dilatation may be present. The test-meal may show no distinguishing feature. The presence of lactic acid and blood is in favor of cancer. *The Anacidity Seen in Pernicious Anæmia.*—The blood picture is the only means of differentiation; the gastric conditions are undistinguishable.

PROGNOSIS.—The condition often persists unrelieved. Occasionally the secretion of free HCl may be restored.

3. Disorders of the Motor Functions.—With one or two exceptions disorders of the motor functions of the stomach are the expression of hysteria or are cultivated habits. They are usually not associated with gastric distress and show no physical signs or changes in the gastric juice.

Some of them, as rumination, peristaltic unrest, gurgling, and singultus, need no special description here. Nervous vomiting, relaxation of the cardiac orifice is unattended by nausea or symptoms of irritation. The ease with which the gastric contents are regurgitated and the absence of the signs of fermentation, hyperacidity, etc., are of diagnostic importance.

Spasm of the cardiac orifice may be differentiated from œsophageal stricture by the use of œsophageal sounds and bougies. The regurgitation of unaltered food, without the chemical reaction of gastric juice, is characteristic of stricture or diverticula of the œsophagus.

Spasm of the pylorus is frequently associated with hyperacidity. Gastric distention, flatulence, belching, colicky pain, are common associated symptoms. In thin subjects the contracted pylorus can be seen and felt. Visible peristalsis may occur. The diagnosis from organic disease may be difficult. The good general condition of the patient, the absence of severe local signs, a normal or excessive acidity of the gastric juice are in favor of a neurosis

Relaxation of the pylorus occasionally occurs, the stomach emptying itself almost at once. The condition is usually discovered accidentally during attempts at removing a test-meal.

Gastric Atony.—Atony of the gastric muscles is usually part of general muscular relaxation seen in nervous and exhausted states. It is practically a condition of moderate dilatation without pyloric obstruction. The coexistence of neurotic symptoms or other disease is important in its recognition.

The prognosis of the motor neuroses of the stomach depends upon the underlying nervous condition and is usually good.

ix. Gastroptosis.

Definition.—A prolapse of the stomach from its natural position, due to stretching of its ligamentous attachments, usually associated with ptosis of other organs, especially the kidneys, liver, and large intestine.

Etiology.—The predisposing influences are neurasthenic states with weakened and relaxed musculature. Possibly the bad carriage and slouching forward of many neurasthenics who always require "straightening up" may be an exciting cause. Repeated pregnancies, recurring ascites, stretching, relaxing, and diastasis of the abdominal muscles are frequent causes.

Symptoms.—Cases in which the neurotic element is absent may show no symptoms, even with the stomach far out of place. As the stomach drags downward, however, kinking at the duodenum is likely to occur, since duodenal ptosis does not follow to any marked degree, and symptoms of mild obstruction may develop. In the markedly neurotic, gastroptosis once established seems to give rise to or keep in continuance many general symptoms, such as faintness, weakness, continued exhaustion, headache, depression, dragging pain in the back and abdomen, loss of weight and strength, sallowness, and slight anæmia. Flatulence from slight obstructive kinking at the duodenum, various forms of nervous dyspepsia, constipation, colicky abdominal pain are common symptoms.

Persistence of the ptosis and obstruction, eventually causing chronic gastritis and distinct dilatation as well, will give rise to the symptoms characteristic of these conditions. Symptoms due to ptosis of the liver, kidneys, and transverse colon are usually coexistent.

Physical Signs.—Marked relaxation and thinning of the abdominal walls may be evident. If the patient lifts his head and shoulders from the couch, "diastasis recti" can often be easily made out. Peristalsis of the stomach or intestine is readily seen between the two flat band-like recti muscles. If much flatulence is present the stomach can be seen outlined and occupying the umbilical region or even reaching into the pelvis, the lesser curvature falling as low as the umbilicus. Palpation shows whether or not other organs, as the liver, kidneys, or spleen, are displaced, and on "dipping" whether or not clapotage is present. Palpable peristalsis is not as evident as in true obstructions, though the pylorus in contraction can frequently be made out. The relaxation and thinning of the abdominal walls is also readily appreciated by the touch. Auscultatory percussion yields gastric tympany practically normal in outline but completely displaced downward. Inflation gives characteristic results. The stomach is entirely displaced downward; the cardiac portion stretched downward; the lesser curvature clearly outlined about the umbilicus in more or less natural relationship to the greater. The stomach may show but little or no evidence of dilatation. The test-meal may show normal acidity of the gastric juice and normal amount of free HCl or hyperacid, hypoacid, or anacid juice. There is usually some degree of retention and evidence of poor action of the gastric muscles, the bread or material used not being well minced or digested. Mucus may or not be present. The passage of food from the stomach, and absorption are delayed, as can be demonstrated by the potassium iodide test.

The X-ray examination is an important aid in the diagnosis of gastroptosis.

Diagnosis.—Direct.—Gastric and neurasthenic symptoms, displacement of liver and kidneys, relaxed abdominal walls, presence of the stomach in an abnormally low area, the organ remaining more or less normal in size and shape, the lesser curvature in the usual relation to the greater, are characteristic of the condition.

Differential.—*Dilatation of the Stomach.*—Gastrectasis and gastroptosis often coexist. Symptoms such as nausea, copious vomiting, thirst, and wasting are significant of dilatation. So also are visible and palpable peristalsis and the signs of hypertrophied musculature. In dilatation the enlargement principally displaces the greater curvature downward, the lesser curvature remaining more or less fixed except in the case of movable tumor involving the pyloric extremity. The gastric contents removed in cases of dilatation are usually excessive, 300–600 c.c., whereas in gastroptosis there may be but a few c.c. more than normal.

Prognosis.—A markedly prolapsed stomach probably never returns to its former position. Untreated cases usually terminate in dilatation or become complicated with chronic gastritis. In many cases a surgical procedure is necessary to maintain the stomach in a position to properly empty itself.

II. DISEASES OF THE INTESTINES.

i. Enteritis.

Inflammation of the intestines. Any part or the whole of the gut may be involved. There are important clinical and pathological distinctions between inflammation of the large and of the small intestine.

Catarrhal Enteritis.—Definition.—A disordered condition of the small intestine associated with increased secretion and frequent watery or softened stools. Abdominal pain, mucous stools, and evidences of disordered intestinal digestion occur. Acute and chronic forms are described.

Etiology.—PREDISPOSING INFLUENCES.—Certain individuals are especially liable to catarrh of the bowels as a result of either dietetic or climatic conditions. In women and children intestinal catarrh occurs very readily. Habitual dietetic errors and chronic disease predispose to the development of catarrhal enteritis. EXCITING CAUSE.—Frequent attacks of acute enteritis may lead to a chronic catarrhal condition. Unwholesome food, toxic food products, certain poisons, as arsenic and mercury, nervous irritability, gastric disorders, particularly hyperacidity, intestinal affections, tubercular ulceration, enteric fever, excessive use of purgatives, and sudden changes from a warm to a cold temperature are exciting causes. Variations in the composition of the intestinal juices, arising independently or due to lack of proper stimulus on the part of the gastric juice, may play an important part.

Symptoms.—General symptoms, such as depression, exhaustion, thirst, anorexia, nausea, are common to acute and chronic enteritis. Wasting occurs very rapidly in the former, and may be accompanied by fever.

Abdominal colic is more common in acute than in chronic enteritis. It is apt to occur shortly after eating and is usually referred to the mid-abdomen. Abdominal tenderness is commonly present. In the chronic cases pain is not a conspicuous symptom.

Diarrhœa is the main feature of the disorder. It may exist as, (a) frequent, watery, brownish colored, unoffensive acid movements, well mixed with brownish sago-like soft mucus. The absence of fetor is largely due to the fact that time for fermentation and decomposition is lacking. Microscopical examination shows excess of undigested food remnants. On standing such stools show gas formation and develop an intense putrefactive odor. This particular form of diarrhœa usually attends the acute cases. (b) Less frequent soft mushy stools, often distinctly pale and putty-like (pultaceous stools), very offensive when passed. At times no undue frequency is to be observed. There is an intimate mixture of fine sago-like mucus. The pallor is due to undigested fat; the mucus and gas bubbles to fermented carbohydrates. Microscopically, undigested food particles are abundant. Fat droplets, fatty acid crystals, some starch, meat fibres are in excess. Bile-stained epithelium and mucus can be seen. The reaction is intensely acid. Gas production occurs on standing. Neither occult nor gross blood is to be detected in uncomplicated catarrhs.

Fetor of the breath and a pasty tongue are common. In thin subjects visible peristalsis may be seen. The pulse is usually slow. The urine is diminished in amount and contains indican, very frequently also albumin and casts.

Diagnosis.—Abdominal pain and tenderness, diarrhœa, the presence in the movements of fine particles of bile-stained mucus and excess of undigested food are significant. Fermentation, acidity and pallor of the stool are indicative of intestinal catarrh. Occult or gross blood would indicate ulcerative or hemorrhagic conditions. Large flakes or masses of mucus occur in membranous enteritis. Time and the Widal reaction will distinguish the cases with fever from typhus abdominalis.

Prognosis.—In the acute cases the outlook is mostly favorable. Repeated attacks of acute enteritis may end in the chronic form. Even protracted cases occasionally terminate in recovery.

Phlegmonous Enteritis.—Acute suppurative inflammation of the submucous tissue of the small intestine, occurring anywhere in its course, either as a primary disease or as a secondary affection in various intestinal accidents.

I. **Primary Phlegmonous Enteritis.**—A disease of very rare occurrence. ETIOLOGY.—*Predisposing Influences.*—We know nothing of the conditions likely to induce primary phlegmonous enteritis. *The Exciting Cause.*—Acute bacterial infection by pyogenic organisms, usually the Streptococcus pyogenes or the Bacillus coli communis. SYMPTOMS.—The symptoms are those of acute peritonitis. There is no definite classical picture of the disease. Diarrhœa is not a necessary accompaniment. DIAGNOSIS.—The diagnosis has not been made during life. PROGNOSIS.—The disease is invariably fatal.

II. **Secondary Phlegmonous Enteritis.** — A condition occurring in connection with various intestinal disorders. Embolism, carcinomatous and tuberculous ulceration, intussusception, strangulation may be complicated by phlegmonous enteritis. ETIOLOGY.—*Predisposing Influences.*—The above conditions predispose. *Exciting Cause.*—Infection by pyogenic organisms as the result of an infected embolus, thrombosis, extensive ulceration, or complete obstruction of the intestine from any cause. SYMPTOMS.—The symptoms are those of the primary disorder plus those of peritonitis. There are no distinguishing features of the disease. DIAGNOSIS.—The symptoms of peritonitis superadded to those of the existing intestinal condition may suggest phlegmon formation. The differentiation between developing phlegmonous enteritis and peritonitis is not possible. The PROGNOSIS is lethal. Recovery from phlegmonous inflammation does not occur.

Diphtheritic Enteritis.—**Definition.**—An inflammatory disorder of the intestine, usually secondary, associated with necrosis, ulceration, and the formation of pseudomembrane. **Etiology.**—PREDISPOSING INFLUENCES.—Chronic diseases predispose to the affection. Cancer, Bright's disease, cirrhosis of the liver may be especially mentioned. Diphtheritic enteritis is frequently a terminal infection. EXCITING CAUSES.—Acute infections, as enteric fever and pneumonia, and certain poisons, as mercury, lead, and arsenic, are among the exciting causes.

Symptoms.—The condition may exist without symptoms. Thirst, fetor of the breath, loss of appetite, diarrhœa, ill-defined pain, dryness of the skin, and wasting are symptoms when the lesions are in the small intestine, tenesmus and diarrhœa when the lower bowel is involved. The toxic cases usually present the very acute symptoms. The clinical picture of the primary affection may completely mask the intestinal condition. Fever may or may not be present. The disease may last for many weeks. Indicanuria, increased sulphate excretion, and albuminuria are common. The fæces are not characteristic. Poor digestion of all elements is evident and the stools may be pale from undigested fat, frothy and fermenting from the starch remnants, and intensely fetid from decomposing proteids. Unaltered blood and pus may be present. Occult blood can usually be detected.

Differential Diagnosis.—*Catarrhal Enteritis.*—The symptoms in diphtheritic enteritis are more urgent. Severe pain, bloody and purulent mucoid stools suggest diphtheritic enteritis. The primary infection or the history of chronic or acute poisoning should arouse suspicion as to the nature of the intestinal affection. *Membranous or Mucous Enterocolitis.*—Diphtheritic enteritis is an inflammatory necrosing disease with a necrotic fibrinous membrane formation. Mucous enterocolitis is a neurosis associated with but transient if any inflammation and an increased mucus formation. Diphtheritic enteritis is usually a grave disorder associated with wasting and serious symptoms; mucous enterocolitis often allows progressive increase of weight and strength. The stools of the latter condition are usually normal fecal material plus mucus. Save in an acute attack blood is not present.

Prognosis.—Occurring as a terminal infection in chronic disorders diphtheritic enteritis usually ends fatally. In the acute infections the primary disease is the important element in prognosis. The diphtheritic enteritis following the administration of poisons is always serious and often fatal.

ii. Diarrhœal Disorders of Children.

Definition.—Acute and chronic disturbances of the gastro-intestinal tract in infants, associated with diarrhœa and various clinical and pathological conditions. They are usually the result of, (1) disordered digestion, (2) absorption of toxic products, (3) acute infection.

Etiology.—Predisposing Influences.—Feeble and poorly developed infants, those suffering from illness, and those nursed by ailing mothers are especially predisposed to intestinal disorders. The change from breast to artificial feeding, particularly in the summer months, exposes the intestinal tract to infection and favors the absorption of toxins.

Exciting Causes.—Improper food, proteid or carbohydrate excess, the ingestion of milk contaminated by bacteria and their products, and, finally, a specific bacterial dysenteric infection through the milk, water, or other means are the exciting causes. The dysentery due to the Shiga bacillus and allied organisms includes a great number of cases formerly considered due to intestinal decomposition and toxemia—probably the majority of the so-called summer diarrhœas.

Symptoms.—Several forms of infantile diarrhœa are recognized: (1) dietetic, (2) toxic and bacterial, (3) inflammatory, (4) chronic.

1. **Dietetic. Acute Gastro-enteritis.** — Fever, rapid pulse, anorexia, restlessness, crying, are initial phenomena. Convulsions may occur. Vomiting and diarrhœa soon ensue. The abdomen is distended, and there are evidences of abdominal pain. The vomitus is not characteristic. The stools, six to twelve or more in the twenty-four hours, are brown, watery at first, and in the mild cases becoming greenish, offensive, with green-brown mucus and fragments of undigested milk or food. In the more severe cases there is an increase of fever, together with prostration and diarrhœa and persistent offensive green acid stools. The common organisms of the intestine are always present in abundance.

2. **Toxic and Bacterial Diarrhœas.**—Infection or toxæmia may be superadded to the dietetic gastro-enteritis. More commonly the toxic and bacterial diarrhœas occur independently, following the ingestion of infected milk, or without evident cause. *Cholera Infantum.*—The onset is abrupt and characterized by convulsions, restlessness or stupor, marked prostrat on, rapid wasting, and all the evidences of a severe toxæmia or infection. The temperature rises rapidly to 104°–105° F. (40°–55° C.); thirst is pronounced, and the skin is clammy and turgid, or inelastic and shrivelled in consequence of the loss of fluids by the bowel. Death may ensue in twenty-four to forty-eight hours. The bowel movements, brownish or greenish at first, but soon becoming gray and watery with abundant mucus and flocculi, are incessant. There is little or no odor and the reaction is alkaline. Blood and pus are unusual. The Shiga and allied dysentery bacilli, together with other organisms, are present in these cases.

3. **Inflammatory Diarrhœas.**—Either of the preceding forms may be followed by a localized ulcerative dysenteric ileocolitis, or the condition may be inflammatory from the start. Even though digestion and absorption may be little affected, the inflammatory and ulcerated condition of the lower ileum and colon gives rise to fever, pain, tenesmus, and frequent bloody or mucopurulent stools. Free pus or blood, usually from the lower part of the colon, may be passed. The odor of these stools is not necessarily offensive and the fecal remnants may show a fair degree of digestion. The various dysentery bacilli and the Streptococcus pyogenes are frequently found in these cases. Diphtheritic or membranous enterocolitis is a common sequel.

4. **Chronic Diarrhœa** —Repeated attacks of dietetic diarrhœa may lead to chronic catarrhal enteritis. The inflammatory ileocolitis may persist for months. Failure to gain weight, loss of appetite, a dry wrinkled skin, nervousness, and general evidences of failing nutrition are evident. Persistent catarrh of the upper bowel is characterized by five or six loose greenish daily movements, offensive, and with excess of mucus. Undigested food is easily detected. Fermentation from the carbohydrates may be demonstrable. The putrid odor of albuminous decomposition may be recognized if the proteids are poorly digested. In chronic ileocolitis pain frequently occurs upon defecation. Mucus persists in large quantities. Blood and pus may be absent. The stools may have resumed their normal brown yellow color. Dysentery bacilli are found in some of these cases.

Diagnosis.—In most instances the diagnosis is evident. Cases with persistent fever may require a Widal reaction to distinguish them from typhoid. In the chronic cases the tuberculin test may help to exclude intestinal tuberculosis. Bacteriological examination of the stools is necessary to differentiate the various forms. Serum diagnosis has not yielded positive results.

Prognosis.—In the dietetic forms with careful treatment the prognosis is good. The acute toxic and infectious cases have always a grave prognosis; only the strong infants survive. The prognosis in the inflammatory diarrhœas is likewise grave. If the infant survive the acute attack there is always the probability of an ensuing chronic ileocolitis.

In the chronic diarrhœas only exceptional cases recover, and often only after months. Many cases ultimately die of inanition or some acute terminal infection.

iii. Ulceration of the Intestines.

1. **Ulceration Restricted to the Small Intestine.**—(a) **Duodenal Ulcer; Peptic Ulcer** —Non-malignant ulcer of the duodenum above the papilla of Vater and of the same nature as gastric ulcer.

Etiology.—Irregular habits in regard to eating, over-indulgence in alcohol, and chronic gastritis with hyperacidity are important predisposing factors. The immediate cause is obscure. The etiology of duodenal ulcer is the same as that of gastric ulcer, and the two conditions are frequently associated.

The ulcers may be acute or chronic. The former are circular or punched-out and more or less superficial; the latter often funnel-shaped, with thickened, indurated, and sloping borders. Duodenal ulcers are mostly near the pylorus and frequently upon the anterior wall of the gut. Gradual cicatrization with stenosis and dilatation of the stomach, obstruction of the biliary and pancreatic ducts, hemorrhage from erosion of large arterial trunks, and perforation are common.

(b) **Peptic Ulcer of the Jejunum.**—This condition occurs after gastrojejunostomy, the common site being near the point of attachment to the stomach. The causes are the same as those which lead to duodenal peptic ulcer. Hyperacidity plays an important rôle. Perforation may occur with circumscribed or general peritonitis.

Symptoms.—In both its general and local symptoms duodenal ulcer may be indistinguishable from gastric ulcer. Pain is less common in duodenal ulcer, and is apt to be late, occurring three to five hours after eating. It is referred to the right hypochondrium. Vomiting and dyspeptic symptoms are less frequent. Hæmatemesis may never occur, the blood being passed entirely by the bowel. Such hemorrhages and the resultant anæmia may be the only symptoms at first of deep ulceration. There may be an acutely localized painful area as in gastric ulcer. The test-meal shows nothing more than in gastric ulcer. Inflation and other modes of examination show nothing distinctive.

Diagnosis.—Sudden hemorrhage from the bowel (tarry stools) or persistent evidence of blood in the stools by chemical test (occult blood).

rapid or slowly progressive anæmia, pain in the right hypochondrium, occurring some hours after eating, justify the suspicion of duodenal ulcer. A positive diagnosis may be impossible.

DIFFERENTIAL DIAGNOSIS.—Gastric hyperacidity with pyloric spasm and gastralgia are to be considered. The occurrence of hemorrhage, gross or occult, anæmia, and the subsequent changes due to narrowing of the duodenum are important. In gall-stone disease, jaundice, the absence of blood in the fæces, and the paroxysmal character of the pain are suggestive.

(c) **Duodenal Ulceration Following Extensive Burns of the Skin.**—This form of intestinal ulceration occurs in about 6 per cent. of all fatal burns. The ulcers may be single or multiple, but rarely exceed six in number. The usual site is in the horizontal portion of the duodenum. They are long, narrow, and irregular in outline and commonly superficial. They have been found as early as the second day and as late as the third week, but most frequently about the end of the first week. They are more common in burns of the trunk than of the limbs, and in females than males.

Various hypotheses have been advanced to explain the restriction of this form of ulceration to the duodenum. Hunter assumes that, as a result of the impairment of function of the skin, toxic substances capable of causing ulceration of the duodenal mucous membrane are excreted with the bile. Embolism of the duodenal arteries and destruction of the antiferments in the mucous cells, with impairment of resistance to the digestive power of the gastric juice, are other explanations.

2. **Ulceration Peculiar to the Large Intestine.**—(a) Stercoral ulceration of the sigmoid flexure or colon, due to pressure of impacted fæces. (b) Dysenteric ulceration.

3. **Ulcerative Conditions Occurring in both Small and Large Intestines** include tuberculous, malignant, syphilitic, thrombotic, embolic, and simple follicular ulceration.

The ulceration of enteric fever is fully considered in the section on the Infectious Diseases.

Etiology.—PREDISPOSING INFLUENCES.—Any exhausting condition and old age favor the development of simple ulceration. Tuberculous or malignant ulceration may be primary or secondary.

EXCITING CAUSE.—Pressure of the impacted fæces is the exciting cause in stercoral ulcer. The swallowing of tubercle bacilli in the sputum or food is the cause of primary tuberculous ulceration. The local determining cause of syphilitic, malignant, and many thrombotic ulcerations is unknown. Embolic ulceration follows the blocking of arterial twigs in the intestinal wall. The exciting cause of simple follicular ulceration is often an acute toxic enterocolitis, an unchecked diarrhœa, or persistent gastro-intestinal abuse. Syphilitic and tuberculous ulceration are considered in the section on the diagnosis of the specific infectious diseases.

Symptoms.—General symptoms are wasting, anæmia, thirst, and, in tuberculous and syphilitic ulceration, slight fever. Subnormal temperature is more frequent. The symptoms of an associated neoplasm or obstruction are to be considered.

A prominent symptom is persistent diarrhœa, usually painless if the ulceration is in the small intestine, attended with pain and tenesmus when it involves the large bowel. Abdominal tenderness is generally present. Mucus, pus, and visible blood may be present in large quantities when the affection is in the colon. Thin watery movements are suggestive of small intestinal ulceration. Occult blood and excess of undigested food are usually demonstrable in the stools. Normal digestion of food substances may be but little interfered with even when the process is in the small bowel, and the examination of the fæces may fail to show excess of food remnants. Ulceration in the ileum may be unattended by diarrhœa.

Physical Signs.—A scaphoid abdomen, palpable thickening of the transverse colon, visible peristalsis, tenderness or pain on pressure, particularly along the course of the large bowel, in some cases complete absence of tympany may be observed. The physical signs of a neoplasm may coexist. Direct inspection of the sigmoid and descending colon may show the ulcerating area and is often the only positive proof of the existence of colonic ulcer.

Diagnosis.—DIRECT.—No combination of symptoms is conclusive evidence of ulceration. Detection by means of the Kelly tubes is of course final. The distinction of the various forms of ulceration may be impossible without direct inspection of the ulcerated area. Clinically the course of stercoral and follicular ulcers is usually favorable after the removal of the cause; that of malignant and tuberculous ulcerations unfavorable. Stercoral ulcers are large, few in number or single, clean cut, show little or no induration or inflammatory reaction. Follicular ulcerations are apt to be numerous, small, and distinctly outlined. Considerable inflammation of surrounding tissue is usually present. Malignant ulceration is usually attended by considerable infiltration of adjacent tissues. Scrapings from the surface may show characteristic microscopical findings. Tuberculous ulceration of the lower bowel is unusual. The tuberculous ulcer is distinctly marked; tubercles may be seen. Scrapings may show tubercle bacilli, giant cells, or characteristic microscopical tubercles. Amœbæ coli are present in the fæces in the ulceration of amœbic dysentery.

DIFFERENTIAL.—*Acute and Chronic Intestinal Catarrh.*—The persistence of the symptoms, the more marked wasting, anæmia, and cachexia suggest ulceration. Direct examination by means of specula affords, in many cases, the only sure means of differentiation. The presence of small tissue fragments in the fæces is evidence of ulceration. Occult blood may be present in both conditions. *The Intestinal Neuroses.*—The more serious general symptoms, the presence of blood, mucus, and pus, and direct examination of the bowel are of diagnostic value.

Prognosis.—The outlook is usually favorable in stercoral and follicular ulceration. The course of malignant and tubercular disease of the bowel is usually uninfluenced by treatment. The prognosis in syphilitic, thrombotic, and embolic ulcerations is uncertain. The majority of such conditions are only recognized post mortem.

iv. Intestinal Stenosis and Obstruction.

Narrowing and occlusion of the lumen of the intestine due to a variety of causes, and occurring anywhere in the course of the bowel from the pylorus to the rectum.

1. **Stenosis or Incomplete Obstruction.**—**Etiology.**—PREDISPOSING INFLUENCES.—Previous acute inflammatory conditions and malignant disease are the most important. Women, for obvious reasons, are especially predisposed to intestinal stenosis; their greater liability to enteroptosis also increases their liability to stenosis from the kinking or twisting of misplaced intestines.

EXCITING CAUSES.—The direct causes are many and may be enumerated according to the location of the narrowing. In the small intestine the cicatrices of duodenal ulcers, gall-bladder and common bile-duct disease, diseases of the head of the pancreas, cancer of the duodenum, jejunum, or ileum, omental and peritoneal adhesions, and accidents, as hernia, adhesions or compression caused by new growths, and involvement of the gut in inflammatory diseases of the appendix or pelvic organs are the most common causes. In the large intestine peritoneal and appendicular adhesion, adhesions to the gall-bladder, adhesions to and compression by pelvic tumors are common causes of stenosis. Cicatricial narrowing following dysenteric and stercoraceous ulceration is an occasional cause. Tuberculous ulceration of the large bowel is less often followed by stenosis, while syphilis of the rectum, with ulceration and resulting narrowing, is a frequent cause. Cancerous invasion of the rectum and sigmoid flexure is one of the most common causes of intestinal stenosis.

Symptoms.—GENERAL.—General symptoms in intestinal stenosis may depend upon the cause more than upon the narrowing of the bowel. Anæmia, wasting, and loss of appetite occur early in cancerous stricture and in tuberculous and syphilitic disease, whether the stenosis is of extreme grade or not. In the mechanical stenosis due to pressure and adhesions, unless they are near enough to the stomach to cause early dilatation and vomiting, wasting may not occur and anæmia may be long absent. Mental and physical depression are usually marked in persistent stenosis. Thirst is a common symptom; oliguria occurs.

LOCAL SYMPTOMS.—The situation is important. The fluid contents of the upper bowel may be easily forced through an opening which would be occluded or readily obstructed by the solid fæces of the lower bowel. Stenosis above the ileum sooner or later produces a dilatation of the stomach with marked local gastric symptoms. Stenosis in the colon is likely to be attended by constipation, a symptom not common in narrowing at a higher level. Finally, tenesmus and intensely painful muscular contractions are limited to stenoses of the large bowel.

Stenosis of the Duodenum and Jejunum.—Distention after eating, eructations, gradually increasing and persistent nausea, and finally vomiting are common local symptoms. Persistent biliary vomiting suggests stenosis below the papilla of Vater, while symptoms of disturbed hepatic and pancreatic function occur when the common and pancreatic ducts are involved in the lesion causing obstruction. Painful contractions of the intestine are not usual in stenosis of the upper parts of the small intestine. The vomitus is that of gastric dilatation; it is not fecal. Bile is present under the conditions just mentioned. Pancreatic ferments may be recognized. Blood tests may be positive if ulceration exists. The fæces are not distinctive. Associated occlusion of the bile and pancreatic ducts may show unabsorbed fat and undigested proteids, and carbohydrates in excess. Blood tests will be positive if there is ulceration.

Stenosis of the Ileum.—Distention following the intake of food is less common. The stomach is not usually dilated. Nausea and vomiting of gastric and intestinal contents occur, but not continuously. Stenosis situated low in the ileum may be associated with a slightly fecal smelling vomitus. Painful contractions of the bowel do not often occur, but in some cases persistent crampy pain with moderate distention is the only symptom. Many ileal stenoses never reveal themselves till a sudden occlusion produces an acute obstruction.

Stenosis of the Large Bowel.—Distention of extreme degree may be present, particularly if the stenosis is very low. Constipation is the rule, or constipation alternating with diarrhœa. Vomiting occurs occasionally, but is only fecal when it has persisted for some hours and acute obstructive symptoms have supervened. Tenesmus and painful muscular contractions of a periodic type are characteristic of lower bowel stenosis. With ulceration blood is present in the fæces. There is, however, no characteristic stool in stenosis of the lower bowel.

Physical Signs.—INSPECTION.—The existence of anæmia and cachexia, and wasting are to be noted. Marked distention is often apparent in stenosis of the large bowel. Stenosis of the small bowel is less apt to give rise to extreme distention. A distended stomach may be apparent in cases of duodenal stenosis. Inspection frequently shows a tense intestinal tube (intestinal rigidity) or several, one above the other (ladder pattern), and these rigid distended parts may further show energetic peristaltic movements running up to and ending in the obstruction and sometimes bringing a stenosing tumor into view. The colon or its sigmoid flexure may be clearly outlined. The latter may occupy the whole abdomen. Inspection may also reveal tumors, a protruding hernia, fulness in the hernial tracts, and the scars of abdominal operations which suggest adhesions or constricting bands. PALPATION.—The rigidity of an intestinal tube above a stenosis is easily appreciated. Peristalsis with muscular hypertrophy and a stenosing tumor of the bowel may be felt. Abdominal tumor, hernia, adherent scars, and the like can be readily examined. PERCUSSION is of limited value. AUSCULTATION, beyond allowing us to hear fluids trickling through an aperture and to conclude that it is still patent, does not afford any aid in diagnosis.

INFLATION of the stomach in the endeavor to determine duodenal stenosis gives no clear result. Inflation of the large bowel may, in thin subjects, reveal a stenosis in the upper part of the sigmoid flexure. Stenosis of the descending colon or transverse colon may become evident, but as a general rule the natural distention above the stenosis is more distinctive. RECTAL EXAMINATION in intestinal stenosis may reveal the occluding mass of a pelvic tumor, narrowing of the anus and lower rectum due to stricture, or the rough, hardened, ulcerating edges of a malignant growth. VAGINAL EXAMINATION may at times reveal palpable tumors or stenosing conditions in the pelvis or adjoining intestines. PROCTOSCOPIC AND SIGMOIDOSCOPIC EXAMINATION will show the presence of stenosing conditions, cicatrizing ulcers, syphilitic or fibroid stricture, or the narrowing of the intestinal tube from outside pressure. The X-RAYS after bismuth injection or ingestion may yield important diagnostic facts. A well-outlined sigmoid or colon may show acute kinking, a tumor, or constriction preventing the passage of the bismuth beyond a certain point even after many hours. Less accurate information must be expected in stenosis of the small intestine.

Diagnosis.—DIRECT.—Distention, abdominal pain, cramp-colic, tenesmus, constipation, or alternating diarrhœa suggest stenosis of the large bowel. Persistent gastric symptoms and gastrectasis, recurrent vomiting with no evidence of pyloric tumor or pyloric obstruction, and continuous biliary vomiting direct attention to the small bowel as the seat of trouble. The recognition of an intestinal tumor, the appearance of intestinal rigidity, intestinal patterns, visible peristalsis, and visible and palpable muscular hypertrophy make the diagnosis sure. Fecal vomiting, which usually indicates that the condition has passed from stenosis to complete obstruction, localizes the obstruction a very short distance either above or below the ileocæcal valve.

DIFFERENTIAL.—*Nervous Dyspepsia, Nervous Flatulence and Vomiting, Enteralgia.*—Gastric dilatation as seen in duodenal stenosis, intestinal rigidity, intestinal patterns, and visible and palpable intestinal peristalsis do not occur in the neuroses. Vomiting in the neuroses is easy, is apt to occur immediately after eating, and the vomitus is usually undigested, odorless food. General symptoms with the exception of wasting are less marked. *Lead Colic.*—The diagnosis may be impossible, since temporary stenosis undoubtedly takes place during the spasmodic contraction. Marked anæmia, with the early symptoms of intestinal disorder, and a blue line on the gums may be the only distinguishing features. *Persistent Vomiting of Alcoholism, Locomotor Ataxia, Gastroxynsis.*—In these conditions the general symptoms are practically absent. The history is different, and in tabes characteristic ocular and nervous phenomena are present. The vomitus in these conditions consists of mucus and a watery gastric secretion, and never contains food remnants or has a fecal odor, no matter how persistent and profuse it may be. *Distention of the Intestines (Paretic Distention) of Acute Fever; Idiopathic Dilatation of the Colon.*—In these conditions stenosis, pain, colic, complete constipation, and vomiting are absent, though faint visible peristaltic and intestinal patterns may sometimes be seen. Obstruction hypertrophy and palpable muscular contraction do not occur.

Prognosis.—The prognosis depends upon the cause of the stenosis. Cancerous stenosis, unless a diagnosis has been made in time to allow resection, is fatal. Any simple stenosis may terminate suddenly in a fatal obstruction, but the majority can be relieved by operation.

2. **Complete Obstruction.**—Many chronic stenoses end in acute obstruction. Various accidents, strangulation, twistings of the bowel, volvulus, etc., produce the same results—complete occlusion of the bowel, the retention of flatus and intestinal contents, and the sudden development of serious symptoms.

Etiology.—PREDISPOSING CAUSES.—*Chronic Stenosis.*—Bands of adhesion and open hernial canals constitute predisposing factors.

EXCITING CAUSES.—Excesses at table or accumulation of the residua of coarse food or of other material may suddenly block the narrowed gut. Twisting of the bowel, various hernial accidents, and intussusception are immediate causes. The settling of a large pelvic tumor is a not uncommon direct cause of acute obstruction. Foreign bodies, gall-stones, accumulation of parasites, and impaction of fæces are also direct causes. Almost any acute abdominal inflammation may give rise to acute obstruction.

Symptoms.—The general symptoms are marked and severe. In simple stenosis, so long as a narrowed opening remains patent, general symptoms may be absent. Large collections of fecal material may accumulate without causing marked discomfort, but the moment the obstruction becomes complete serious symptoms supervene. Rise in the pulse-rate, increased vascular tension, slight elevation of temperature followed by the signs of prostration and collapse, pallor, sweating, facies abdominalis, thready pulse, and shallow respiration constitute a symptom-complex at once alarming and significant. Thirst and oliguria are constant. Intense grinding pain, eructations, nausea, and persistent vomiting, at first of gastric juice, later of gastric fluid commingled with bile or intestinal contents and bile, and, when the obstruction is below the ileocæcal valve, of distinctly fecal material, speedily occur. The vomitus in obstruction of the lower ileum may have a faintly fecal odor. Distention is a constant phenomenon and is more marked the lower the obstruction. Volvulus and intussusception may be accompanied by the passage of blood, and in sigmoid obstruction tenesmus with bloody mucous discharges occur. Neither fæces nor flatus are passed per rectum. In unrelieved cases peritonitis rapidly develops with intestinal paresis, generalized pain, and marked meteorism.

Diagnosis. — DIRECT. — Grave general symptoms, abdominal pain with intense exacerbations, tenesmus, nausea, eructations, persistent vomiting eventually becoming of a fecal character, absolute failure to pass flatus and fæces, distention, intestinal patterns, rigidity, and tumefaction of the intestine as the contraction reaches the obstruction are the main diagnostic features. Changes in the urine such as the appearance of excess of sulphates (indican) are not diagnostic. Leucocytosis is not constant and therefore not an important diagnostic feature.

DIFFERENTIAL.—There are but few conditions which simulate acute obstruction. Thrombosis of the mesenteric vessels, acute pancreatitis, and acute enteritis with relaxation of the intestinal coils, pain, and vomiting, may be mentioned. The absence of intestinal patterns, rigidity, and visible and palpable peristalsis are of value in differentiating these conditions from acute obstruction in cases seen prior to the development of peritonitis and paralytic distention. Acute appendicitis with peritonitis may closely resemble acute obstruction. A history of attacks of pain in the region of the appendix is often obtained. The diagnosis may be difficult. Spontaneous relief is rare. Early surgical intervention is imperative. In neglected cases death ensues in the course of three to six days.

Obstruction of the large bowel from accumulation of hardened fæces is rarely complete. Fecal obstruction can be differentiated from stenosis due to other causes by the recognition of the accumulated masses, the comparative mildness of the general and local symptoms, the absence of marked visible and palpable intestinal peristalsis, and the relief afforded by judicious therapeutic measures.

v. Dilatation of the Intestines—Idiopathic Dilatation of the Colon.

Definition.—Chronic dilatation of the colon and sigmoid flexure, not due to stricture or accumulation of fæces.

Etiology.—Predisposing causes are unknown. A history of chronic constipation is usually obtained. The condition usually occurs in quite young persons and children. The pseudocyesis of middle-aged women depends largely upon dilatation of the colon and sigmoid flexure.

EXCITING CAUSES.—Fecal accumulation plus paretic distention of the bowel occurring repeatedly produces the condition. Spasmodic contraction of the rectum must coexist. Idiopathic dilatation due to structural abnormalities is a probable cause.

Symptoms.—The general health may be but little affected. Acute symptoms rarely occur. Extreme distention gives rise to respiratory and cardiac oppression. Constipation is the rule, but diarrhœa occurs. The most prominent symptom is distention. Pain, colic, and obstructive symptoms are absent. The distress that accompanies distention of the small intestine is not observed in idiopathic dilatation of the colon. Enormous distention of the abdomen, thinning of the abdominal walls, the presence of lineæ atropicæ, and glazing of the skin are seen. With marked wasting of the abdominal walls the outline of the sigmoid flexure rising from the pelvis and reaching to the costal margin, or the outline of the colon, can be made out. Peristalsis is not pronounced. Palpation shows the distention to be gaseous. No resistance or solidity is felt; no fluctuation wave obtained. Percussion gives a marked tympany everywhere, even in the loins up to the base of the lungs behind, and often shows an obliteration of the liver dulness in front. The passage of a soft rubber tube into the sigmoid flexure relieves distention by allowing the exit of air and shows what part of the bowel is affected. Reinflation through the tube produces a gradual ballooning and outlining of the sigmoid or colon. Examination by means of the speculum shows merely the relaxed condition of the colon when the air is expelled.

Diagnosis.—DIRECT.—The absence of serious local and general symptoms, constipation, extreme chronic distention with outlining of the sigmoid or parts of the colon, the disappearance of distention on passing the rectal tube, and the results of inflation are characteristic.

DIFFERENTIAL.—The general symptoms and the relief of the distention by the tube differentiate dilatation of the colon from *gastrectasis.* The shape and position of the stomach are radically different. *Distention of the Small Intestine due to Obstruction or Paretic Conditions of the Musculature.*—The general symptoms of disease of the small intestine are more marked. Intestinal patterns are smaller and more numerous; peristalsis (save in paretic distention) may be seen. *Distention of the Large Bowel due to Stricture, Malignant Growth, etc.*—The distention due to obstruction is associated with signs of associated muscular hypertrophy. Visible or palpable active peristalsis, pain, and colic are common. General symptoms rapidly develop. The obstruction can often be recognized by the speculum or examining finger, or upon palpation through the abdominal wall. *Fluid Accumulations.*—Bulging of the flanks, movable dulness on turning, fluctuation wave, flatness on percussion readily distinguish fluid accumulation from gaseous distention.

Prognosis.—The disease in itself is rarely fatal. In the idiopathic cases death commonly occurs early in life. General treatment has little effect. Removal of the distended sections of the colon and sigmoid flexure has been tried.

vi. Appendicitis.

Definition.—Inflammation of the vermiform appendix.

The conception of appendicitis is a modern one, dating from the studies of Reginald Fitz (1886). It includes and explains the facts relating to foreign bodies in the appendix; catarrhal, diffuse, purulent, and necrotic inflammation of that organ; ulceration, gangrene, cyst formation, and abscess of the appendix; chronic, recurrent, and obliterative inflammation; peri-appendicular abscess, typhlitis, perityphlitis, and iliac phlegmon; and local and general peritonitis having its starting-point in the ileocæcal region.

These conditions constitute phases in the evolution of a single process—appendicitis. The central fact is infection of the appendix.

The infection may be a local manifestation of a general infection, as, for example, influenza or pneumonia; or a purely local process, as in the case of foreign bodies, fecal concretions, the extensions of an inflammation from the cæcum, or when injury to the appendix results from strains or blows; and finally the infection may be associated with a specific local lesion in a general disease, as when typhoid ulceration involves the lymph tissue in the appendix. Kelynak (1903) suggested that acute appendicitis is a metastatic inflammation arising from a distant primary focus of infection. Other observers, notably Apolant and Kretz, have recently advanced the opinion that "appendicitis begins as a metastatic disease of the adenoid tissue, and that the lymphatic apparatus of the throat and nose is to be regarded as the most frequent primary localization and portal of entry of the infection." The recognition of the unity of the process under varying etiological conditions and in varying clinicopathological manifestations is the key to its diagnosis and treatment.

Etiology.—PREDISPOSING INFLUENCES.—Appendicitis is the most important and one of the most common of the acute diseases of the intestine. There are no especial causal relations connected with country, race, social conditions, or occupation, save that it has been held that those whose occupations involve habitual strain and heavy lifting suffer more commonly than others. About half the cases occur before the twentieth year. It is rare in infancy but common in childhood and adolescence. Cases have been observed as late as the seventh and eighth decades. It is equally common in the two sexes. The symptoms have occasionally followed a fall or blow upon the abdomen. Indiscretion in diet, especially overeating, and exposure to cold and fatigue are conditions frequently mentioned in the anamnesis. The acute infections, in particular influenza, pneumonia, and rheumatic fever, sometimes are attended with or followed by appendicitis. A majority of the cases, however, arise in ordinary health without any obvious or discernible determining cause. Two or three cases in the same house at or about the same time have occasionally been observed.

THE EXCITING CAUSE.—Various pyogenic organisms have been found in the early lesions, among which Bacterium coli communis and Streptococcus pyogenes are common. A lesion of the mucosa, caused by the presence of a foreign body, fecal concretions, retained secretions, or traumatism, probably constitutes the point of entrance for pathogenic bacteria.

Nature of the Pathological Process.—The character of the lesions is determined by the intensity of the infection and the reaction of the tissues involved. Broadly speaking, the lapse of time between the onset of the attack and the condition at any given hour has a most important bearing upon the anatomical diagnosis; that is to say, the early lesions are relatively simple and limited, the later complex and extensive. But to this rule there are many exceptions. In a large group of cases, the so-called catarrhal cases, the inflammation runs a favorable course, resolution takes place in a short time, and in a few days the patient is convalescent. But the recovery is by no means always complete in the sense of an anatomical *restitutio ad integram.* The inflammation subsides but the appendix remains infected and lesions of a chronic and progressive nature persist—infiltrations of the mucosa and submucosa, connective-tissue overgrowth, local atrophies involving especially the longitudinal and circular muscular fibres, stricture-like narrowings, retained secretions, cyst formation, angular kinking, adhesions, and other deformities. It is in such cases that the teasing pains known as appendicular colic occur, in which there is persistent discomfort and frequent tenderness in the right lower quadrant, in which inflammatory flare-ups recur, and in which at any time necrosis, perforation, abscess formation, or general peritonitis may suddenly arise. They constitute the cases of so-called recurrent appendicitis, an unfortunate and misleading term since this form of the disease is in point of fact essentially chronic with occasional exacerbations—a smouldering fire with now and then an ominous puff of flame. On the other hand there are cases in which the symptoms of onset are urgent, and necrosis and perforation follow in the course of a few hours. Many of the cases, however, make a substantive recovery and live on without subsequent attacks. Others, which constitute a large proportion of all cases, go on more or less rapidly from bad to worse, terminating in abscess formation and chronic invalidism, or general peritonitis and death.

The natural history of appendicitis is indicated in the following table.

The course of the attack may be interrupted and in a majority of the cases its more serious events and unfavorable terminations arrested by early surgical intervention.

Symptoms.—The significance of the clinical phenomena becomes more apparent from a careful consideration of the following pathological data:

APPENDICITIS.

I. ACUTE.

Pathological variety.	Onset.	Course.	Termination.
A. Catarrhal	Abrupt	Brief; a few days	(a) Recovery without subsequent attacks. (b) Recovery with subsequent attacks at long intervals. (c) Chronic forms: (1) Apparent recovery, with subsequent attacks at irregular short intervals, with uneasiness and pain in the intervening periods. (2) Recovery from acute symptoms with persistent local pain and tenderness, intestinal symptoms, and other indications of ill health. (3) Chronic thickening and hyperæmia, without symptoms. (4) Thickening with fibrotic changes, an involution process; the obliterative form. (d) Suppuration.
B. Suppurative	Abrupt. May follow the catarrhal form	Variable; 24 to 36 hours, several days	(a) Pus formation: when the lumen is obliterated the distal appendix may be converted into an abscess cavity; more commonly necrosis occurs. (b) Local peritoneal infection without actual perforation; local adhesive peritonitis and peri-appendicular abscess. (c) Necrosis.
C. Ulcerative	Gradual in the course of the catarrhal form from pressure of a foreign body or appendolith, or in the suppurative form from intense local action of pyogenic organisms	Variable; cannot be known	(a) Peri-appendicular adhesive inflammation. (b) Perforation and abscess formation. (c) Perforation and peritonitis.
D. Gangrenous	Abrupt, or may follow suppurative form, or be fulminant	Very variable; may occur after several days of suppuration or ulceration, or with urgent, even fatal, symptoms in the course of a few hours	(a) Appendicular abscess, circumscribed or extensive. (b) General peritonitis, rarely adhesive; commonly ichorous or purulent, and almost always fatal. (c) When necrosis occurs at the site of adhesions to neighboring organs, as the gut or bladder, a fistulous communication is established through which pus is discharged. (d) Hemorrhage.
II. CHRONIC.			
E. Primary fibrosis	Insidious	Prolonged; indefinite without marked clinical manifestations	(a) Thickening of appendix with progressive obliteration of lumen beginning at distal extremity. (b) General atrophy and sclerosis.
Secondary fibrosis	Obscure. May be insidious or a terminal condition following an acute form	Prolonged; condition may not be suspected during life	(a) Thickening and enlargement; elongation or kinking; narrowing of lumen. (b) Chronic adhesive inflammation to adjacent structures with thickening and signs of solid tumor.

Catarrhal inflammation of the appendix may be acute, subacute, or chronic. The mucosa is slightly involved, being swollen, injected, and the seat of an increased secretion. Inflammatory thickening may cause retention of the secretion and persistence of the infection. In the subacute form symptoms may be absent. In the acute form they are of variable intensity, consisting of pain and tenderness in the right lower quadrant of the abdomen, together with loss of appetite, nausea, and occasional fever, which is usually of slight intensity and brief duration. In the chronic form there is persistent tenderness with a sense of thickening upon deep palpation, vague and often intractable gastro-intestinal symptoms, neurasthenia, and in some instances an associated mucous colitis.

Acute diffuse inflammation is more common. The infection extends from the mucosa to the deeper structures. There is inflammatory thickening and hyperæmia which involves the serous investment. Erosion and ulceration of the mucosa are common. The symptoms are more severe than in the acute form and the duration of the attack is prolonged.

Acute suppurative inflammation may supervene. There may be purulent infiltration of the wall of the organ, or the lumen may be occluded so that the pus distal to the stricture forms an abscess cavity. The symptoms are now more severe and a mass may be felt through the abdominal wall. An extension of the purulent infection or a small perforation may be followed by peri-appendicular suppuration and cause a distinct fluctuating tumor. Septic phenomena are often present, as irregular fever, sweating, rapidly developing anæmia, and gastro-intestinal symptoms, especially nausea and vomiting. In many of the cases, however, these symptoms do not occur. Fever in particular may be wholly absent.

Ulceration may follow catarrhal or diffuse inflammation and cause more or less extensive subacute local peritonitis with adhesions. More rarely it may cause perforation with abscess formation. The lesion advances slowly and is far less likely than gangrene to cause general peritonitis. There are no special symptoms of ulceration other than those due to the subacute circumscribed adhesive peritonitis which it causes, namely, local pain, tenderness, rigidity, and a more or less well-defined tumor mass.

Gangrene is common. It may occur in the course of a few hours after the symptoms of onset. In a case in which pain was first felt after the bath at eight in the morning, the whole appendix was found necrotic upon operation at five in the afternoon. Instances of this kind are by no means exceptional. More commonly gangrene takes place, in unrecognized cases or those in which operation is refused, in two or three days or later.

The necrotic process may involve the tip, or a circumscribed patch, or the entire organ, or it may be confined to the region of its attachment to the cæcum. The appendix may slough off and be found loose in the pus of an appendicular abscess. When the necrotic tip has previously become adherent to a neighboring hollow organ, as the colon or bladder, a fistulous communication may be found. The appendix has in some instances perforated into a hernial sac. The necrosis has occasionally involved the wall of an artery, as one of the iliacs, to which adhesions have formed, and led to fatal hemorrhage. Perforation, which only occasionally occurs in the acute diffuse and suppurative forms, is the usual outcome of the gangrenous variety. The opening is commonly single, but multiple perforations may occur. When previously formed adhesions confine the escaping contents of the appendix, an abscess is found which varies in size and may, in the absence of operation, ultimately discharge into the bowel or bladder, vagina, or externally.

Chronic appendicitis constitutes a common condition when the acute symptoms subside without operation. The appendix may be thickened and deformed, adherent to the gut, or embedded in a mass of irregularly puckered peritoneal adhesions. Its lumen may be more or less completely obliterated, or it may be converted into a series of cysts separated by stricture-like occlusions, or, finally, it may contain foreign bodies or fecal concretions. There are cases in which no symptoms are present, but it is more common to find occasional pain, persistent tenderness, and intestinal symptoms. When acute exacerbations of these symptoms occur the condition is known as recurrent appendicitis, falsely so called.

In another group of cases chronic appendicitis takes the form of a progressive primary fibrosis, a progressive involution process, not characterized by symptoms and terminating in complete atrophy of the organ.

The bodies found in the lumen of the inflamed appendix are, as a rule, to which there are few exceptions, fecal in character. The pus varies in amount and consistence. It may be soft and easily expressed, or consist of dense, compact masses resembling date-stones or grape seeds. A series of three or even more such masses may occupy the canal of the appendix. Sometimes there is only one, which is short and rounded, or elongated and curved. Of foreign bodies the most common are pins.

The symptom-complex of acute appendicitis is most variable. Certain symptoms are, however, suggestive, their association significant. The chief of these are pain, tenderness, and gastro-intestinal disturbance. Of subordinate importance are fever, muscular rigidity, deep induration, and leucocytosis. We cannot be satisfied with an affirmative reply to the question: Has this patient appendicitis? We must ask ourselves further: What stage in its evolution has the process reached? Is the appendix alone affected? Are there signs of abscess formation? Is there local adhesive peritonitis without or with pus? Are there signs of a beginning general peritonitis? In other words, What are the lesions?

1. PAIN.—The pain is usually sudden, mostly severe. It is sometimes sharp and stitch-like; sometimes dull and aching. It is often colicky; almost always continuous with exacerbations. It is usually referred to the right lower quadrant of the abdomen, but may be diffuse or central. In the chronic cases a diffuse central pain is very often present for weeks or months before the pain in the right iliac fossa is felt. It may be referred to the region of the gall-bladder or to the right lumbar region, or extend in these directions; or it may extend in a similar way to the perineum or right testicle. Under these circumstances the pain of appendicitis has been mistaken for biliary or renal colic. These referred pains have been ascribed to the position of the appendix, which has been found in some instances elongated in the direction of the liver or kidney, in others downwards into the pelvis.

2. TENDERNESS.—Firm, deep pressure over the affected area usually causes pain. A circumscribed spot situated at the intersection of a line drawn from the umbilicus to the anterior superior spine of the ilium and a second line corresponding to the outer border of the right rectus muscle, first described by McBurney and known as McBurney's point, is in well-marked cases decidedly, even exquisitely, tender to the pressure of a single finger. Deep pressure with the open hand upon the left side of the abdomen frequently causes or aggravates the pain in the right iliac fossa. The patients usually assume the dorsal decubitus with the right knee flexed, and pain is elicited or increased by extending the limb.

3. GASTRO-INTESTINAL SYMPTOMS. — There are loss of appetite, thirst, nausea, and very often vomiting which ceases in the course of some hours. Constipation is the rule. Diarrhœa occasionally occurs in children. The patients are often able to recall the eating of an unusually hearty meal or some indigestible article of diet, or undue exertion or exposure after food, and attribute pain and tenderness to indigestion or colic or a bilious attack.

4. FEVER.—An initial chill or chilliness is very rare. It is said that fever is always present at the onset. If so, it is very often transient and has disappeared before the case comes under medical observation. Frequently the temperature is normal when the patient is first seen, and in favorable cases remains so. Many cases show subfebrile temperatures not reaching 101° F. (38.5° C.), and fever of irregular type—102°-3° F.—is not uncommon. There are cases in which, with abscess formation or general peritonitis, the temperature remains low, and others in which fever gradually subsides and the patient enters upon convalescence without serious symptoms. Too great reliance upon the thermometer may prove misleading.

5. MUSCULAR RIGIDITY.—Spastic tension of the abdominal wall upon the right side, especially over the rectus muscle, is common in severe cases. In many cases this symptom is not present. Its presence is suggestive of a beginning local peritonitis.

6. INDURATION.—Upon deep palpation a circumscribed sensation of resistance may frequently be detected. This sign is often well defined and situated at or near McBurney's point. In very rare instances the thickened appendix may be distinctly recognized upon palpation. In other cases there is a diffuse ill-defined boggyness with some impairment of resonance upon percussion. Upon the supervention of pus the signs of a solid or fluctuating tumor are characteristic. Necrosis and perforation may take place without either induration or a palpable tumor, especially in the early gangrenous cases.

7. LEUCOCYTOSIS.—In appendicitis unaccompanied by suppuration, gangrene, or serous inflammation, there is usually little increase in the leucocytes. In cases which have gone on to abscess formation, necrosis, and local or general peritonitis, leucocyte counts of 15,000 or more per cubic millimetre are the rule. Normal counts or slight leucocytosis may occur in cases characterized by early gangrene and perforation,—so-called fulminant appendicitis,—and in suppurative cases in which absorption does not take place from the abscess cavity.

8. INCONSTANT AND THEREFORE UNIMPORTANT SYMPTOMS are vesical irritability, oliguria, and albuminuria. As in many other acute inflammatory affections, there is moderate early anæmia of secondary type. In cases attended with prolonged suppuration, with sepsis, anæmia becomes pronounced with a reduction of hæmoglobin to 30 or 40 per cent. and an erythrocyte count of 3,000,000 per cubic millimetre or less.

The course of a case of simple appendicitis terminating favorably is usually as follows: The gastro-intestinal symptoms subside, the tongue cleans, and the constipation ceases spontaneously. The appetite and strength return. The pain gradually disappears, but tenderness outlasts it for some days or a week or two. Local induration progressively diminishes and convalescence is soon fully established. The persistence of a distinct circumscribed tumor is very liable to be followed by recurrent outbreaks.

Abscess and peritonitis are sequels of appendicitis.

Abscess.—The conditions revealed at operation shed much light upon the development and course of appendicular abscess. Perforation either as the result of ulceration or necrosis is the common cause of peri-appendicular suppuration. In exceptional cases it occurs in consequence of general suppurative inflammation of the appendix. Pus may be found in the course of two or three days, but more commonly towards the close of a week. The symptoms are aggravated. There are more or less pronounced signs of sepsis and fever of irregular type. There are, however, cases in which fever is wholly absent. The pain and tenderness are often increased. The abscess cavity is usually in relation with the appendix and the adjacent coils of intestine, which are adherent among themselves as the result of local plastic peritonitis. In some of the recurrent cases there are one or more small collections of pus in an irregular mass formed by dense adhesions and puckering of the wall of the gut. More commonly the abscess cavity is single and its size corresponds to the duration of the case. When small and deeply seated, especially if below the pelvic line, it may elude external palpation but be detected upon vaginal examination. As a rule, it forms a palpable or visible tumor of variable size in the right iliac fossa, which in some cases is distinctly fluctuating. The pus shows the usual tendency to burrow and may discharge into the bowel, vagina, or bladder, or externally.

Peritonitis.—General infection of the peritoneum may at once result from early necrosis, prior to the formation of circumscribing adhesions, or later from the rupture of already formed adhesions. In the fulminant cases the symptoms of general peritonitis may follow those of appendicitis so rapidly that it is not always possible to determine the nature of the primary process. Ordinarily the onset of peritonitis is attended by sudden aggravation of the previous symptoms. The pain, tenderness, and rigidity become more marked and extend over the entire abdomen. Nausea and vomiting are more severe. The pulse becomes small and rapid, the tongue dry, and the urine scanty or suppressed. After a time meteorism, absence of peristaltic movement, dorsal decubitus with flexed thighs, and the characteristic facies complete a well recognized clinical picture. Cessation of pain and a clear mind are the heralds of death.

Diagnosis.—DIRECT.—The recognition of acute appendicitis rests upon the association of sudden pain and tenderness in the right iliac region with nausea or vomiting. The presence of a circumscribed tumor or deep resistance and rigidity of the right rectus muscle are significant. The age of the patient is suggestive, since appendicitis is very common before and comparatively rare after thirty. Conditions having some resemblance to appendicitis are to be carefully excluded, as, for example, hepatic and renal colic, dysmenorrhœa, and tubo-ovarian disease. The diagnostic importance of the blood counting may be readily overestimated.

DIFFERENTIAL.—1. *Cholecystitis.*—When the inflamed appendix lies upwards, the pain and tenderness may suggest gall-bladder disease. In the latter careful physical examination will usually reveal increased dulness and circumscribed tenderness in the region of the gall-bladder, and a history of recurrent attacks without tendency to inflammatory tumor or abscess formation. 2. *Renal Colic.*—It is only in the rare cases in which the appendix extends backwards that uncertainty arises. The pain of renal colic is usually more severe and more distinctly paroxysmal than that of appendicitis. It arises in the lumbar region, extends forward and downward towards the groin, and is attended by retraction of the testicle. The diagnosis in some cases of persistent kidney colic with hydronephrosis is very difficult. The X-ray examination may be of service. Dietl's crises in floating kidney present remote resemblances to acute appendicitis. 3. *Dysmenorrhœa.*—Menstrual colic, especially in an hysterical girl, may suggest appendicitis, but the seat of the pain, the period in the month, recurrence, the absence of a tumor, or the stigmata of hysteria are significant. 4. *Disease of the Right Tube and Ovary.*—A careful vaginal examination is necessary. The recognition of salpingitis or an enlarged, tender, prolapsed, or adherent ovary, together with a history of menstrual derangements or previous pelvic pain would be of diagnostic importance. The fact that such conditions are frequently associated with an infected appendix is by no means to be overlooked. 5. *Mucous colitis* is sometimes associated with chronic appendicitis without distinctive signs of the latter affection. In such a case I have seen a diseased appendix removed with decided improvement in the mucous disease and general health. 6. *Local abscess* in the cæcal region associated with malignant disease of the gut cannot always be distinguished from appendicular abscess. The anamnesis is important. An exploratory incision may be necessary. The situation of the tumor and œdema in perinephric abscess are of diagnostic importance. 7. *Intussusception, volvulus, and other forms of intestinal obstruction* present in the beginning only remote resemblances to appendicitis. Stercoraceous vomiting, so significant in these conditions, does not occur in appendicitis, nor do the tenesmus and bloody stools common in intussusception, especially in children, nor the sausage-like tumor, mostly on the left side, nor the invaginated gut upon rectal examination. 8. *Enteric Fever.*—There is no real difficulty in well-defined cases. Both the pain and tenderness are milder, vomiting is rare, fever is higher and the temperature tends to run a typical course, and headache is a prominent symptom. Later splenic tumor, rose spots, disproportion between the pulse-frequency and temperature, and the results of laboratory methods are conclusive. The occasional occurrence of typhoid ulceration of the appendix and of appendicitis as an intercurrent affection in enteric fever is not to be overlooked. Perforation of the appendix may occur in the second or third week or during convalescence. 9. *General Peritonitis.*—In the absence of a history of the case the peritonitis which follows acute appendicitis cannot be differentiated from that due to other causes.

Prognosis.—The mortality is stated to be 2 per cent. of all cases, but the general statistics are without value since the death-rate is dependent upon extremely variable data relating to diagnosis and operation. The death-rate in early operations is low. The statistics vary from 2 to 10 per cent. according to the time of operation. The immediate recognition of the condition may be of vital importance to the patient. In cases characterized by recurrence, operation in a quiescent interval is attended with comparatively slight risk. The outlook in neglected cases is grave. The patient's general health may be undermined by prolonged suppuration and sepsis, or death may follow pylephlebitis or hemorrhage from an artery or vein. A very large proportion of cases in which early operation is neglected die of peritonitis. The fact that the course of the lesion in any given case can neither be foreseen from the beginning nor controlled by any other sure means, constitutes full justification for early operation.

vii. Enteroptosis.

Definition.—Falling forward or dragging downward of the intestines from stretching of their mesenteric attachments, practically always affecting the large intestine, and frequently associated with ptosis of the stomach, liver, and kidneys—*splanchnoptosis or Glénard's disease.*

Etiology.—See Gastroptosis.

Symptoms.—The general symptoms are often the only symptoms. The condition may exist for years without producing discomfort. The inherent weakness of the tissues which support the abdominal viscera is in turn aggravated by the gastro-enteroptosis. Persistent weakness, abdominal distress, pain in the back and loins, headache, torpor, dulness are common. The patients are always neurasthenic. Hyperchlorhydria or hypochlorhydria may occur. The local symptoms are varied. Abdominal discomfort, burning sensation, the weight of the prolapsed abdominal organs, colicky, irregularly recurring pains, nausea, anorexia, constipation, diarrhœa, abdominal distention are symptoms of enteroptosis. Obstruction from kinking is rare. Many of the so-called cases of intestinal indigestion belong here. Mucous diarrhœa and mucous stools are not uncommon.

Physical Signs.—These have been described under gastroptosis. In enteroptosis the transverse colon is the part of the intestine mostly affected and most readily demonstrated. Even on inspection the outline of the displaced bowel may be evident at and below the umbilicus. Peristalsis from right to left may be observed. Palpation will frequently reveal displacement of the liver and kidneys. At times the spasmodically contracted transverse colon can be clearly felt.

Diagnosis.—DIRECT.—The abnormal position of the colon, displacements of other organs, and diastasis recti are diagnostic features.

Inflation with the tube passed high up will often clearly distend the colon in its various positions. Simultaneous distention of the stomach prevents errors. The X-ray examination after bismuth injections may be of confirmatory value.

Differential.—*Gastroptosis.*—These conditions commonly occur together. The absence of gastric symptoms and the normal position of the stomach on inflation may be observed in exceptional cases. *Malignant Disease of the Bowel.*—The long history of enteroptosis, ptosis of other organs, and diastasis recti, absence of marked anæmia or cachexia, absence of a tumor mass with palpable muscular contraction, absence of occult blood in the stools are in favor of enteroptosis.

Prognosis.—The condition may never cause trouble. A suitable abdominal support may relieve the symptoms but cannot permanently restore a prolapsed intestine to its normal position.

viii. Intestinal Indigestion.

Definition.—Disorders of digestion in the intestine, usually due to deficiency of the pancreatic ferments and characterized by the excretion of abnormal amounts of the various food matters ingested.

Etiology. — Predisposing Influences. — Nervous disorders of the stomach or intestine predispose to intestinal indigestion.

Exciting Causes.—Anacidity of the gastric juice, obstruction of the bile-ducts, pancreatic ducts, chronic intestinal disorders, chronic appendicitis, and dietetic errors are causes of intestinal indigestion. The definite cause that in many instances apparently interferes with the "secretin" production and the pancreatic supply is unknown.

Symptoms.—The general symptoms may be those of a neurosis: headache, coated tongue, persistent loss of weight without obvious cause or without symptoms. Habitual inability to digest certain articles is frequent. Pain in various regions of the abdomen, flatulence, and burning sensations are common. Constipation, diarrhœa, or pultaceous stools may occur. Foul-smelling flatus and fæces that decompose and ferment on standing are usual.

Physical Signs.—Abdominal distention may occur as the sign of active intestinal fermentation. Tenderness can usually be elicited. Examination of the fæces shows excess of fat and fatty acids, and much undigested proteid, or carbohydrate (see Fæces). The various tests (bead test, desmoid test, absorption tests) show impaired digestion and absorption.

Diagnosis. — Direct. — Chronic abdominal distress and symptoms of disturbance of nutrition, evacuation of abnormal amounts of undigested material as proved by chemical and microscopical tests, absence of physical signs of neoplasm, absence of blood from the stools and of fever are important diagnostic features.

Differential. — *Intestinal Neuroses.* — The differentiation is often impossible as a neurosis may coexist with the intestinal indigestion. The condition of the fæces is important. Mucous stools must be distinguished from undigested material. *Neoplasm; Ulceration.*—Chronicity, absence of blood from the fæces, absence of physical signs of obstruction or tumor, the persistent presence of undigested material in the fæces indicate intestinal indigestion.

Prognosis.—If the cause can be removed resumption of normal digestive power may return. The withholding of certain forms of food,—fat, carbohydrate or proteid, as indicated by the fæces,—may be continually necessary. Many cases persist unrelieved by treatment.

ix. Intestinal Neuroses.

Definition.—Disorders of sensation and of the secretory and motor functions of the intestine.

Etiology.—PREDISPOSING INFLUENCES.—All conditions which predispose to functional nervous disorders may be considered as predisposing influences.

EXCITING CAUSES.—Shock or emotional outbreaks may precipitate an intestinal neurosis. The more common exciting causes are strain, worry, irregular habits of life, and dietetic errors.

Symptoms.—Symptoms common to all intestinal neuroses are nervous depression, exhaustion, and derangements of the normal function, sallowness of the complexion, and emaciation. When the digestive function of the intestines is not impaired a physical appearance of well-being is often seen. There is rarely any evidence in the fæces of impaired digestive activity. Common sensory symptoms are a feeling of weight or distention in the abdomen, the subjective perception of the intestine in peristalsis, persistent general discomfort or soreness in the intestines, and colicky pains which are often severe. These pains may simulate appendicitis, biliary, renal, or lead colic, or the tabetic crises. Motor disturbances give rise to nervous diarrhœa, intestinal rumblings and gurglings, or to spasmodic contractions of the bowel with constipation and pain. Explosive flatulence is a common motor neurosis. Retention of intestinal air or gases and distressing flatulent distention is frequently the result of intestinal spasm. Reverse peristalsis with fecal vomiting as a motor neurosis is rare. Few secretory neuroses are recognized. Diarrhœa and constipation are often due to motor causes. Membranous or mucous enteritis and colitis are practically the only secretory neuroses. The main features of these affections are severe abdominal pain, constipation, and the passage of large amounts of mucus, unformed and jelly-like, in opaque, firm flakes, or in large tubular casts. Bile-stained mucus is said to come from the small intestine; the paler variety from the large.

Several motor neuroses may occur in association. Physical examination usually yields negative results. In typical cases the abdomen may be scaphoid. Tenderness is common, particularly along the course of the colon. It is often marked over the cæcum. In mucous colitis, redness of the mucosa and excess of mucus can be seen on examination with the speculum.

Diagnosis.—DIRECT.—Persistent intestinal distress in a neurotic individual, the absence of the local signs of obstruction or neoplasm, the absence of anæmia and cachexia, a [illegible] condition of the fæces as to their constitutent fats, carbohydrates, and proteids, abnormal amounts of mucus, and the absence of parasites and their ova justify the suspicion of a neurotic basis for the intestinal disorder.

DIFFERENTIAL.—*Intestinal Obstruction.*—A neurosis, even when associated with severe pain, constipation, and distention, rarely shows general symptoms, such as rapid pulse and collapse. Fever is absent, vomiting is less usual; local signs of obstruction are absent. The bowels are usually easily opened by the proper means. The detection of mucus in the stools in large amounts or in tubular form is important evidence in favor of a neurosis. *Intestinal New Growths.*—Tuberculosis of the intestine, anæmia, and cachexia are unusual in neuroses; local signs are absent. Tests for blood in the stools are negative. The general neurotic condition of the patient is a most important feature. In *gall-stone colic* subsequent jaundice, bile-stained urine, and detection of gall-stones in the fæces are important diagnostic points. Rapid pulse, fever, and persistent tenderness over the liver are usual in gall-stone attacks. In *renal colic* hæmaturia and occurrence of fever may be the only distinguishing features; in *lead colic* anæmia, "blue line," persistent constipation, and a history of exposure are important. With *tabetic crises* other features of tabes are present, for example, absent knee-jerks, Argyll-Robertson pupil, ataxia. *Appendicitis.*—Cases of intestinal neurosis are sometimes operated upon as appendicitis. The differentiation in some of the chronic cases may be extremely difficult. In the neuroses leucocytosis and muscular rigidity do not occur. Since mucous colitis is the condition most likely to simulate appendiceal disease mucous stools should be carefully looked for.

Prognosis.—The intestinal neuroses are refractory to treatment and often require years of careful management. Confirmed cases are often unrelieved by any form of general or local treatment.

x. Intestinal Neoplasms.

New growths or tumors of the intestines, principally malignant. Benign tumors, lipomata, adenomata, myomata, polypoid growths also occur.

1. **Malignant Growths.**—**Carcinomata.**—Cancer of the intestines may involve any part of the bowel from the duodenum to the rectum. The large bowel is more frequently affected and especially the rectum. The growths are usually primary and tend to assume the annular form and to ulcerate, producing obstruction and hemorrhage.

SYMPTOMS.—The general symptoms of malignant disease of the intestines are loss of appetite and strength, persistent and rapid loss of weight, early and severe anæmia, and rapidly developing cachexia. In many cases local symptoms are not recognized for a considerable time, a fact which lends importance to the general early derangements of health. The earlier local symptoms are those of partial obstruction and ulceration, colic, flatulent distention, and vomiting if the growth is in the duodenum, tenderness when it is situated in the large bowel. Malignant disease in the duodenum soon produces obstructive dilatation of the stomach with its characteristic symptoms. Persistent biliary vomiting indicates obstruction below the papilla of Vater. Duodenal cancer is often associated with occlusion of the common bile and pancreatic ducts. Pain in the neighborhood of the growth itself, apart from the recurring colic of the obstruction-hypertrophy, is only marked in large-bowel cancers, and is of a dull aching or intense gnawing character. Hæmatemesis may occur in duodenal cancers. Black, tarry, or bright hemorrhagic stools are seen in the cases in which the growth is situated lower down. Diarrhœa, constipation, or dysenteric symptoms may be present in cancer of the lower bowel. Later local symptoms are those of complete obstruction, perforation, and metastatic deposits in the glands of the abdomen, in the liver, and elsewhere.

PHYSICAL SIGNS. Careful and repeated inspection and palpation are necessary. Important signs are distention, particularly marked in low-lying tumors, intestinal patterns, visible peristalsis, and a visible or palpable tumor. Transmitted pulsation from the aorta may occur. Palpation with the flat of the hand, superficial at first, then deep, slowly covering the whole abdomen, is important in obscure cases. Small tumors may escape observation. Tumors of the duodenum may be central and do not move with the diaphragm; tumors of the jejunum and ileum are also usually central, but may appear in other regions, and are often freely movable. Tumors of the cæcum and transverse colon are movable but not so freely as those of the sigmoid flexure. Contraction of hypertrophied musculature, causing tension and hardening of the tumor mass, may be frequently observed. Movement of the tumor mass with respiration and with postural changes [illegible] of the intestine [illegible] situated in the duodenum and at the [illegible] flexures and rectum, and is readily appreciated by the examining hand. [illegible] suggestive of an aneurism may be [illegible] large [illegible]

[illegible]

Diagnosis.—*Direct.*—Loss of appetite, persistent wasting, progressive anæmia are suggestive. Signs of obstruction, pain, colic, vomiting, distention, visible peristalsis, visible and palpable contractile tumor are indicative of a new growth in a muscular organ, as the intestine. Occult blood in the fæces or vomitus is an important sign of ulceration.

Differential.—*Non-malignant Partial Obstructions.*—The history of old inflammatory abdominal conditions may be suggestive of non-malignant obstruction. Absence of progressive anæmia and cachexia is an important point. The absence of hemorrhage, either occult or gross, particularly if the condition has persisted for some time, is in favor of a non-malignant condition. A tumor is rarely visible or palpable in non-malignant obstruction save just at the point of obstruction and at the time of contraction of the hypertrophied musculature. *Impacted Fæces, Scybala, etc.*—There is absence of anæmia and cachexia, wasting is unusual, signs of obstruction are commonly wanting. The evidences of hypertrophied intestinal musculature do not appear. Visible peristalsis is only present when obstruction is complete. Occult blood is not present nor do hemorrhages take place unless laceration of the mucosa of the rectum or anus has occurred. The scybalous tumor masses may be multiple and follow the course of the large bowel. They are not contractile but doughy and can at times be broken *in situ.* They can usually be removed by proper measures and are often readily distinguished by sigmoidoscopic examination.

Prognosis.—The outlook in intestinal carcinomata is hopeless unless the condition is early recognized and the case submitted to operation. In disease of the lower bowel, an artificial anus—colostomy—may prolong life for months; acute secondary accidents, as rupture of the bowel and complete obstruction, occur.

Sarcomata.—Sarcomata of the intestine are rare. They attain larger size, are less circumscribed and of more rapid growth than carcinomata. The general symptoms of anæmia and cachexia progress with alarming rapidity. Stenosis of the bowel does not occur, since the growth diffuses through large areas of the submucosa, hence visible peristalsis and contractile tumor masses are rarely observed. Hemorrhage is less common. The small intestine and the rectum are the favorite locations of intestinal sarcomata.

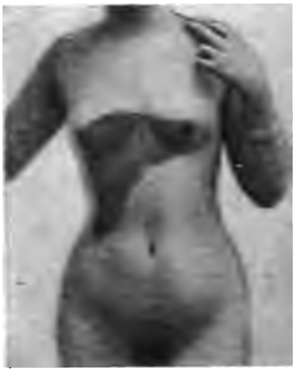
2. **Benign Tumors.**—The benign tumors, principally polypi, occur most frequently in the rectum and large bowel. Hemorrhage and tenesmus are the most marked features. The diagnosis of these and other benign tumors that lie out of reach of the sigmoidoscope must be attended with difficulty. Only when they reach large size do they produce signs of obstruction.

III. DISEASES OF THE LIVER.

i. Anatomical Anomalies of the Liver.

The contour of the liver is modified by the shape of the thorax and the pressure of adjacent organs. Abscess and tumors within the substance of the liver, as echinococcus, gummata, and malignant growths, cause departures from the normal form of the organ. In transposition of the viscera the shape of the liver is the reverse of normal. The convex anterior surface is frequently marked by parallel depressions corresponding to the ribs.

Remarkable changes in shape are produced by the permanent pressure of clothing—corset liver; lacing liver. That portion of the right lobe which is below the groove of compression may reach to the level of the crest of the ilium. It is separated from the main portion of the organ by a deep depression, the result of pressure atrophy, which produces in many cases great thinning, and in extreme cases an entire disappearance of the liver tissue, so that the corset lobe is connected with the liver by a flat band of connective tissue containing only bile-ducts and blood-vessels. The portion thus separated not only occupies an abnormal position, but it is also more or less freely movable according to the degree of atrophy of the compressed part. It, therefore, frequently simulates floating kidney, intestinal tumor, or other movable tumors occasionally found in this situation, especially when a loop of intestine has found its way into the groove. From these conditions it is to be differentiated by its descent and ascent with the respiratory movements of the diaphragm, by its continuity with the liver as demonstrable in many cases by percussion, [illegible] the continuous border of the anterior inferior surface of the liver and the corset lobe as recognized upon repeated careful palpation, and finally, by the presence of the notch or angle in the border at the point where the groove terminates anteriorly. Another deformity produced by the habitual pressure of clothing consists in an elongation of the entire right lobe downward. This change may be mistaken for enlargement of the organ. These changes are common in women but comparatively infrequent in men—belt liver.



[illegible]

Various changes in the position of the liver are encountered. These are usually the result of [illegible] pressure. Rotation upon the transverse axis may [illegible] backward from the pressure [illegible] frequently displaced downward [illegible] abscess and upward [illegible] other abdominal tumors.

ii. Movable Liver.

[illegible]

Definition.—[illegible] abnormal mobility.

Etiology.—[illegible] in enteroptosis and [illegible] floating liver is extremely [illegible] of the abdominal walls [illegible] large hernias in which the [illegible] that the abdominal [illegible] by means of the liga[illegible] disease such as severe [illegible] tight lacing, and rapid emaciation have been regarded as etiological factors in floating liver. The frequency of these events as compared with the extreme infrequency of floating liver renders it in a high degree improbable that they play the part assigned to them in the causation of the latter condition. Therefore it is likely that floating liver can only occur in cases of congenital tendency to relaxation and elongation of the ligaments of the organ, or in which an actual mesohepar is present.

Symptoms.—There is, as a rule, no tenderness upon pressure. Pain is a common symptom. It is referred to the right hypochondrium and the epigastrium, and extends to the right shoulder and lumbar region. It is dull and dragging, and intensified by sudden movements. Spontaneous paroxysmal pain, bearing-down sensations, attacks of colic, with belching, meteorism, and constipation, and anomalous abdominal sensations are common. The pain is usually relieved by firm pressure upon the tumor or by lying down. Respiratory disturbances and palpitation occur. Hemorrhage from the stomach and bowels, ascites, hemorrhoids, and œdema of the legs and feet have been observed. Jaundice is rare, but the skin usually has a subicteroid hue.

Physical Signs.—The abdominal tumor occupies the right side and may extend as low as the pubic arch. The convex surface is directed forward and the entire organ is rotated to the right. The contour may be made out upon palpation. In the dorsal decubitus the liver may be replaced by gentle pressure unless fixed by adhesions—an extremely rare condition. Upon percussion when the liver is dislocated the pulmonary resonance passes directly into the tympany due to the intestines which have found their way into the space between the liver and the diaphragm.

Diagnosis. — A DIRECT DIAGNOSIS rests upon the position of the tumor, its contour, its large size, and the possibility of replacing the dislocated liver in its normal position. The diagnosis is greatly obscured by the presence of ascites and by the diminished mobility resulting from adhesions in the abnormal position.

The DIFFERENTIAL DIAGNOSIS involves the consideration of a greatly thickened mesentery and tumor of the kidney. Neither of these tumor masses is associated with tympanitic resonance in the normal area of liver dulness, nor can either of them be made by manual pressure to pass into the normal position of the liver. Floating liver occurs chiefly in women.

iii. Jaundice—Icterus.

This symptom-complex has been described in a previous section and the mechanism and significance of obstructive and toxæmic jaundice discussed.

The following special forms are of clinical interest:

1. ICTERUS PSYCHICUS (*Icterus ex Emotione*).—Sudden jaundice has been attributed to anger, fright, terror, and a gross insult. Associated symptoms are anxiety, epigastric distress, and diarrhœa. The jaundice passes away in a short time. The cases are not well authenticated and no wholly satisfactory explanation has been adduced for emotional jaundice.

2. HEREDITARY ICTERUS.—The cases are rare and may be regarded linical curiosities. Icterus neonatorum has been observed in every

member of large families in two generations. Another group includes cases of jaundice in a mother and three of her children, appearing in childhood and persisting for years without other evidences of ill health. The jaundice was of mild type. There was no enlargement of the liver or spleen.

3. ICTERUS GRAVIDARUM.—Women in adanced pregnancy occasionally suffer from a form of catarrhal jaundice due to the pressure exerted upon the under surface of the liver by the enlarged uterus. Fecal accumulation acts as an additional cause of bile stasis, and the deformities of the liver resulting from lacing increase the tendency. The death of the fœtus or miscarriage may occur. The jaundice disappears after parturition.

4. ICTERUS MENSTRUALIS.—Mild icterus has frequently been observed just prior to and during menstruation. When the discharge becomes free the jaundice abates. Enlargement of the liver and decolored fæces have been noted.

5. BILIOUSNESS.—Many persons, whose health is otherwise good, occasionally suffer from gastro-intestinal derangements, with headache, furred tongue, sensations of depression and malaise, and a subicteroid discoloration of the conjunctivæ or skin. Relief of these symptoms follows abstinence from food and mild purgation.

6. STARVATION JAUNDICE.—Subicteroid discoloration of the conjunctivæ and skin is frequently present in persons who for any reason are unable to take food for several days. This is witnessed in insane persons who refuse food, and in stricture of the œsophagus, whether spasmodic or organic.

7. SYPHILITIC JAUNDICE.—Icterus appears in certain cases of severe syphilis coincidently with the secondary eruption. Its symptomatic character is shown by its prompt disappearance under antisyphilitic treatment. This form of jaundice is to be differentiated from accidental jaundice, such as occurs in tertiary syphilis as a consequence of diffuse hepatitis or gumma of the liver, and from a coincident catarrhal jaundice.

8. ICTERUS FOLLOWING THE EXTRAVASATION OF BLOOD.—Yellowness of the conjunctivæ and skin may be observed after large hemorrhages into the skin or cavities of the body in scurvy, and after injuries or in lesions of the genital tract in women. The jaundice appears after several days, is not intense, and gradually fades in the course of some days or weeks. Bile pigments are present in the urine.

9. ICTERUS FOLLOWING HÆMOGLOBINÆMIA.—This variety of jaundice is very marked after the attacks of hæmoglobinuria which follow exposure to cold or overexertion in persons suffering from malaria or syphilis. Fever, splenic enlargement, hæmoglobinuria, and jaundice constitute the symptom-complex. The urine contains bile pigments.

10. TOXIC ICTERUS.—A large number of poisons are followed by icterus. Among them the following are important: arseniuretted hydrogen, certain mushrooms, toluylendiamin, glycerin, the bile acids, the chlorates, aniline and its derivative acetanilide, and the nitrites. Filix mas and santonin may cause a yellow discoloration of the skin. Icterus follows poisoning by phosphorus and lead. A very rapid icterus develops after snake bite.

11. INFECTIOUS ICTERUS. — Yellow fever and relapsing fever are characterized by marked icterus. Septic conditions and pneumonia are frequently, enteric fever occasionally, attended by jaundice.

12. EPIDEMIC ICTERUS.—When a number of persons living under the same conditions develop jaundice, as occasionally occurs in boarding schools, camps, barracks, or prisons, the term epidemic jaundice is warranted. Many large and small epidemics have been described in the literature. These epidemics are usually of short duration; in a few instances they have lasted several months. The disease commonly assumes the guise of ordinary catarrhal jaundice and runs a benign course; in some instances it is severe and many deaths occur. In pregnant and parturient women the prognosis is grave. Epidemic icterus has been attributed to atmospheric or climatic influences, dietetic faults, and infectious causes. A combination of these agents may be operative.

13. POSTVACCINAL JAUNDICE. — In rare instances jaundice has appeared in groups of cases among revaccinated persons. The jaundice has occurred at intervals of a few days to several months. It has been attributed to wound infection. This form of epidemic jaundice is rare and its association with vaccination is probably accidental. It is much more likely due to other local influences affecting groups of persons who happen to have been vaccinated.

IV. Icterus Neonatorum—Physiological Icterus.

Definition.—A variety of icterus common in the new-born occurring independently of any particular disease or lesion and pursuing a favorable course.

Etiology.—This affection occurs in about one-half of all babies. It is more common in foundling hospitals than in private practice, in premature infants than in those born at term, in boys than girls, and in cases where parturition has occurred under chloroform. The pathogenesis of the condition is not clear. It has been attributed to rapid destruction of erythrocytes after birth, to stasis in the smaller bile-ducts, to resorption of bile from the intestine, and to œdema of the periportal connective tissue.

Symptoms.—The jaundice appears upon the second or third day after birth and first upon the face and chest, rapidly extending to the rest of the body. It fades more or less rapidly in the course of ten or twelve days. The general condition of the child is otherwise normal. The urine is normal and does not contain bile pigment in solution. The stools, after the discharge of meconium, have their usual golden-yellow color. The pulse-frequency is not lowered.

Diagnosis. — DIRECT. — The comparative mildness of the jaundice and the complete absence of serious symptoms suffice to establish the nature of the affection. Its gradual disappearance within two or exceptionally as late as three or four weeks is not followed by recurrence.

DIFFERENTIAL.—In the following forms of jaundice in the new-born the discoloration is more intense and associated with serious symptoms: (a) congenital absence of the common or hepatic duct; (b) congenital syphilitic hepatitis, in which the characteristic external lesions of syphilis are also manifest, and (c) septic infection by way of the umbilical vein, a fatal form of sepsis associated with phlebitis and, in some instances, with umbilical hemorrhage. Icterus may occur in the new-born as a result of obstruction of the bile-ducts, acute fatty degeneration of the liver, and epidemic hæmoglobinuria.

Prognosis.—The physiological icterus of the new-born is never fatal.

v. Acute Yellow Atrophy.

Malignant Jaundice; Icterus Gravis.

Definition.—An acute disease characterized anatomically by diffuse necrosis of the liver-cells with great diminution in the size of the organ, and clinically by intense jaundice and cerebral symptoms.

Etiology. — PREDISPOSING INFLUENCES. — Acute yellow atrophy is rare. It is more common in women than in men in the proportion of about 8 to 5. This difference is in part explained by the fact that pregnant women are frequently affected after the fourth month or at the time of parturition. The greater number of cases occur between the twentieth and fortieth years. Acute yellow atrophy is comparatively rare among children. It has been observed to follow osteomyelitis, erysipelas, sepsis, enteric and relapsing fever, and syphilis. It has been attributed to ptomaine and mushroom poisoning, to alcohol and to chloroform, to fright, and to profound depressing emotions. The symptoms caused by phosphorus poisoning resemble those of acute yellow atrophy, but the conditions are neither etiologically nor pathologically identical. In rare instances acute yellow atrophy has occurred as an intercurrent disease in hypertrophic cirrhosis, bile stasis, and fatty degeneration of the liver.

THE EXCITING CAUSE.—The actual pathogenic principle is unknown. Various micro-organisms, especially streptococcus and Bacillus coli, have been found in the liver, but in many of the cases examined no bacteria have been present.

Morbid Anatomy.—The liver is greatly reduced in size, flaccid, and folded upon itself. Its capsule is wrinkled and of a dirty yellowish-green color. Upon section the surface is mottled and the outlines of the lobules are indistinct. These are yellowish masses surrounded by a dark reddish tissue, the latter representing a more advanced stage. Microscopically the hepatic cells are indistinct, bile stained, and in all stages of granular and fatty degeneration and necrosis. The capillary vessels and bile-ducts are destroyed, with resulting minute hemorrhages and extravasation of bile.

Symptoms.—The attack usually begins as an acute gastric catarrh, which is shortly followed by more or less intense jaundice with clay-colored stools—the initial stage. In the course of some days or, less frequently, two or three weeks, during which the symptoms have remained comparatively mild, the second stage sets in suddenly with vomiting, restlessness, stupor, delirium, convulsions, and coma. Hemorrhages into the skin and from mucous surfaces are common. The jaundice becomes more intense. Pregnant women usually abort. There may be pain in the region of the liver. The temperature is nor[illegible] subnormal, rising toward the end. Exceptionally there is marked fever throughout the attack. The tongue is coated and dry. The action of the heart, normal or slow in the initial stage, becomes rapid and feeble in the second stage, with enfeeblement of the first sound and not rarely a soft, blowing, systolic murmur. The liver, which is enlarged in the first stage, undergoes, with the development of the cerebral symptoms, a rapid diminution in volume. There may be complete absence of hepatic dulness, as the flaccid organ folds upon itself and falls away from the abdominal wall, coils of intestines taking its place. The splenic dulness is increased. The abdomen is very sensitive, particularly in the epigastric zone, and there is spontaneous pain. The urine is slightly decreased, contains bile pigments, generally small quantities of albumin and tube-casts. Products of disordered metabolism, such as leucin, tyrosin, and sarcolactic acid, are also present. Urea is much diminished and the percentage of nitrogen present as ammonia correspondingly increased. Leucin and tyrosin are sometimes absent from the urine. Albumoses are sometimes present in small amounts.

Diagnosis.—DIRECT.—Intense jaundice, vomiting, diminished area of liver dulness, hemorrhages, enlarged spleen, grave cerebral symptoms, together with leucin and tyrosin in the urine, constitute a characteristic symptom-complex. The initial stage cannot be distinguished from ordinary gastroduodenal catarrh with jaundice.

DIFFERENTIAL. — *Hypertrophic Cirrhosis.* — In rare cases there are intense cerebral symptoms, but enlargement of the liver, fever, the absence of leucin and tyrosin, and the long course of the disease prior to acute cerebral symptoms are distinctive.

Acute Phosphorus Poisoning.—The symptoms may be almost identical, particularly in respect of hemorrhages, jaundice, and decrease in liver dulness, but the gastric symptoms are usually more intense and set in directly after the ingestion of the poison without a prodromal or initial stage, the icterus rather abruptly on the third day, and leucin and tyrosin are said to be absent from the urine. A dilated transverse colon may so displace the liver upward as to simulate atrophy, but the gradual reduction in the area of liver dulness from day to day is most significant.

Prognosis.—The disease is almost always fatal. The outlook is more unfavorable when cerebral symptoms occur early. The duration of the illness varies from a few days to two or three months. About half the cases die between the fifth and fourteenth days, about one-third within five weeks. The duration of the characteristic stage varies from one or two days to a week. In the cases that run a favorable course the cerebral symptoms are less violent and the duration of the disease is prolonged.

vi. Diseases of the Bile Passages and Gall-bladder.

CATARRHAL JAUNDICE.

Icterus Gastroduodenalis; Icterus Simplex.

Definition.—Jaundice due to swelling and mucus in the intestinal portion of the common duct, the result of the extension of gastrointestinal catarrh.

Etiology.—PREDISPOSING INFLUENCES.—Catarrhal jaundice is a common affection and is probably always associated with catarrhal inflammation of the duodenal mucosa. All conditions which predispose to the latter affection therefore favor its occurrence. Chief among these are chronic alcoholism, chronic gastric catarrh, conditions favoring portal obstruction, chronic valvular disease, and chronic nephritis. Malaria is a well-recognized cause. The affection also frequently occurs in connection with the acute infections, particularly enteric fever and pneumonia.

Age exerts an important predisposing influence. Catarrhal jaundice is a disease of young persons and is rare in middle and advanced age. It is common in children after the second year. In adult life it is more common in males than females.

THE EXCITING CAUSE.—The immediate cause is usually an acute indigestion, sudden cold or exposure, or unusual physical strain with irregular meals. In many cases no causal factor can be discovered. When a number of persons are exposed to the same local influences, as in a school, a circumscribed epidemic of catarrhal jaundice may occur.

Morbid Anatomy.—The mucous membrane of the terminal portion of the common duct is swollen and the ampulla of Vater may be obstructed by a plug of tenacious mucus. It is possible that the catarrhal process may invade the smaller ducts, but of this we have no definite knowledge.

Eppinger recently found in the case of a girl who was instantly killed by an accident on the ninth day of an attack of catarrhal jaundice that the portion of the common duct which lies within the wall of the intestine was impermeable and that the occlusion was due to hyperplasia of the lymphoid tissue of the mucosa of the duct. It is probable that certain cases of catarrhal jaundice are due to inflammatory swelling; others to the presence of a plug of tenacious mucus, and yet others to a hyperplastic condition of the lymphoid tissue which surrounds the mucous glands of the appendix in varying proportion in different individuals.

Symptoms.—In many cases the jaundice is preceded by epigastric distress, loss of appetite, a coated tongue, and nausea and vomiting. Not rarely there are also present headache, vertigo, mental depression. Fever is not common, but the temperature may reach 101°-102° F. (38.3°-38.9° C.). The bowels are constipated and the stools light in color but rarely entirely free from bile pigment. The urine contains bile pigments and is scanty and sedimentary but not often albuminous. Hyaline casts are common. The skin and conjunctivæ are more or less deeply jaundiced, but the olive green of chronic jaundice does not occur. The nervous symptoms of jaundice are present in varying degree, especially pruritus and drowsiness. Slowing of the pulse and respiration is less frequent. The liver is usually slightly enlarged. The gall-bladder is rarely palpable. Slight enlargement of the spleen is not uncommon. There are cases in which the above symptoms do not occur and the patient's knowledge of his being jaundiced is obtained from the looking-glass or the inquiries of his friends. The duration of the affection is from two to six or eight weeks, the jaundice gradually fading, the bile pigment first reappearing in the stools, next disappearing from the [illegible] finally from the sclera. There are cases of catarrhal jau[illegible] months with remissions and exacerbations, but the diagnosis in such cases must be guarded, especially in elderly persons.

Diagnosis.—DIRECT.—The diagnosis may usually be made without reserve from the youth of the patient, his previous fair health, the symptoms of gastric catarrh, the moderate intensity of the jaundice, and the short duration of the affection.

DIFFERENTIAL.—The diagnosis from cirrhosis, cholelithiasis, carcinoma, and Weil's disease is considered under those respective headings, to which the reader is referred. It is important to remember that catarrhal jaundice is rare in old persons and that a jaundice persisting beyond six or eight weeks can only be regarded as catarrhal after a rigid process of exclusion in regard to all other possible causes.

Prognosis.—The outlook in simple uncomplicated catarrhal jaundice is highly favorable. It is a benign affection.

CHRONIC ANGIOCHOLITIS.

Definition. — Chronic inflammation of the bile-ducts. It may be catarrhal or suppurative.

1. **Chronic catarrhal cholangitis** may occur as a sequel of acute catarrh. It is always combined with obstruction of the common duct, and is therefore an associated condition in cholelithiasis, parasites, cancer, stricture, and compression of the common duct from without. The obstruction may be complete or incomplete.

(a) *Complete Obstruction.*—The bile passages, the gall-bladder, and the intrahepatic ducts are dilated and contain clear mucus, which is usually sterile. The patients are persistently and deeply jaundiced. Fever is commonly absent.

(b) *Incomplete Obstruction.*—There are one or more calculi in the common duct and, as a rule, in the gall-bladder, or there is pressure from the outside. The bile may escape in small amounts continuously, or the obstruction may be intermittent. The bile passages and gall-bladder are not usually greatly dilated. They contain a thin, bile-stained mucus. The jaundice may vary in intensity and the stools show the presence of bile pigments. Febrile attacks,—hepatic fever,—characterized by chills, rapid rise of temperature, and profuse sweating, are common in this form of obstruction and are doubtless caused by infection.

2. **Suppurative cholangitis** affects the large and small ducts. In the majority of the cases the gall-bladder is also involved. There is dilatation of the bile passages and particularly of the common ducts. The walls are thickened. The intrahepatic ducts are much dilated, and minute collections of pus mixed with bile are formed by the suppurating ducts and disintegrating hepatic tissue. There is usually distention of the gall-bladder, which is filled with pus, occlusion of the cystic duct, and adhesive inflammation of the gall-bladder to adjacent parts. Suppurative cholangitis constitutes one of the most serious complications of cholelithiasis. It occurs also in consequence of the presence of foreign bodies, as fish bones, intestinal parasites, as ascarides, which find their way into the ducts [illegible] the intestine, and in connection with cancer of the ducts. It is a rare sequel of enteric fever, pyæmia, and dysentery.

The onset is insidious. The symptoms vary in intensity and are not always characteristic. As a rule they are severe, consisting of jaundice, enlargement of the liver, pain and tenderness in the region of the gall-bladder, which is often distended, fever of septic type, and a marked leucocytosis. There is commonly a history of biliary colic. Pylephlebitis, endocarditis, purulent meningitis, and peritonitis are occasional complications.

The diagnosis of suppurative cholangitis rests upon a history of gall-stones, jaundice, intermittent fever of hectic type, tenderness and pain in the region of the gall-bladder. The fever is to be distinguished from malaria by the leucocytosis, the variable periodicity, and above all by the absence of blood parasites. A tender point in the region of the twelfth dorsal vertebra, 2.5 to 3 cm. from the middle line, may be present in inflammation of the bile-ducts (Boas). In cases occurring in association with the acute infections jaundice may be absent or slight. The differential diagnosis between certain cases of suppurative cholangitis and abscess of the liver may be attended with difficulty. In favor of the latter condition are absence of jaundice, slight fever or even subnormal temperature, and the absence of tenderness and pain in the region of the gall-bladder.

The outlook is highly unfavorable. The reëstablishment of biliary drainage by the escape of the stone or its removal by operation may be followed by recovery. In a case recently under my observation operation failed and there were many small calculi pocketed in abscess cavities throughout the liver.

VARIOUS LESIONS OF THE BILE PASSAGES.

It is convenient to consider ulceration, perforation, stricture, and fistulæ of the bile-ducts in connection with cholelithiasis, which is their usual cause.

The lumen of the common duct may be partially or completely occluded by the seeds of fruit and by certain parasites, among which lumbricoid worms are common and echinococci and distomata rare causes of obstruction in man.

Obstruction by pressure from without is more common. Carcinoma or fibroid thickening of the head of the pancreas, or, in rare instances, cancer of the pylorus, enlarged lymph-glands, abdominal tumors, and aneurism of the cœliac axis may compress the common duct.

The symptoms have already been described in connection with complete and incomplete obstruction under the heading Chronic Angiocholitis. Complete permanent occlusion of the common duct terminates in death. The conditions which cause occlusion by pressure from without are usually fatal. The diagnosis of the cause of the obstruction may be difficult. Colic, with variable jaundice and intermittent fever, suggests cholelithiasis. Cancerous disease in the rectum or genito-urinary tract, or the stomach or intestines, points to secondary glandular infiltration as the cause of biliary stasis and jaundice. Accessible groups of lymph-nodes and, in particular, the left clavicular lymphatic glands may also be enlarged. The gall-bladder is frequently distended and may be distinctly palpable.

INFLAMMATION OF THE GALL-BLADDER; CHOLECYSTITIS.

The condition may be acute or chronic.

1. **Acute Cholecystitis.**—The inflammation may be catarrhal, suppurative, or phlegmonous. These forms sometimes represent different degrees of intensity.

Etiology. — Acute cholecystitis is commonly due to gall-stones. It may, however, result from bacterial invasion in the absence of cholelithiasis. It is common in the infectious fevers and a subacute form is very often met with in enteric fever. The usual pathogenic organisms are the colon bacillus, the bacillus of Eberth, the pneumococcus, streptococcus, and staphylococcus. The condition is frequently associated with cholangitis and dilatation of the bile passages. The gall-bladder is usually distended. In subacute cases of long duration distention may be prevented by fibrous thickening of the walls. Adhesions with the adjacent parts of the liver or the omentum or colon may take place. The cystic duct is frequently occluded even in the absence of an impacted calculus. The enlargement sometimes takes place upward and inward and there is no palpable tumor. The contents may be a thin, dark-greenish mucus, or mucopurulent, purulent, or hemorrhagic. Perforation may take place, with abscess formation limited by the adhesions or into the peritoneal cavity.

Symptoms.—In the milder forms there may be simply some tumefaction with dulness, circumscribed tenderness, and a rise of temperature. This form is common in enteric fever. The severe forms are ushered in with intense paroxysmal pain in the region of the gall-bladder, the epigastrium, or in the right upper quadrant of the abdomen. With this are associated nausea and vomiting, arrest of peristalsis, rigidity of the abdominal muscles and especially of the rectus upon the right side, and fever. The enlarged gall-bladder may be sometimes recognized upon palpation and percussion, but as a rule the extreme tenderness interferes with physical examination. In the absence of gall-stones jaundice does not commonly occur.

Diagnosis. — DIRECT. — The milder forms are readily recognized; in the more severe cases the condition is often very obscure. The anamnesis is important. The above symptom-complex occurring in a patient who has had attacks of hepatic colic or cholecystitis, or who is convalescent from enteric fever or pneumonia, is significant. The recurrent forms are readily diagnosticated. It is important to remember that cholecystitis may occur without gall-stone disease.

DIFFERENTIAL.—The condition may simulate acute obstruction of the bowels or appendicitis. While these conditions may be differentiated from acute cholecystitis by characteristic symptoms in a large proportion of the cases, there are instances in which the actual organ affected has been revealed only upon operation.

Prognosis.—The outlook depends upon the intensity of the inflammatory process. The purulent and phlegmonous forms are usually fatal Timely surgical intervention may be the means of saving life. The danger of perforation is to be constantly borne in mind.

2. **Chronic Cholecystitis.**—The common cause of chronic cholecystitis is cholelithiasis. The disease may arise in consequence of extension of the inflammation in cholangitis. The muscular and connective-tissue elements of the wall are involved. When the contents undergo resorption, or escape through the cystic duct or by way of a fistulous opening, the thickened wall contracts and the gall-bladder becomes permanently reduced in size. Its walls under these circumstances are sometimes the seat of calcareous changes. Pericholecystitis may develop with diffuse swelling around the organ, and later fluctuation. The cystic duct may be completely occluded by a calculus or by cicatrices. The bile is then absorbed, the mucous membrane, however, continues to secrete an abnormal mucus, and the gall-bladder undergoes gradual distention with thickening of its walls and sometimes more or less extensive peritoneal adhesions. The contained liquid may be light in color and bile-free,—dropsy of the gall-bladder,—or it may be pus—empyema. Gall-stones are frequently present.

Symptoms.—When the dilatation is slight the gall-bladder extends below the border of the liver but cannot be palpated unless the abdominal walls are thin. As the enlargement proceeds it constitutes a palpable pear-shaped tumor, which is movable from side to side and may be displaced backward by moderate pressure, but which resumes its position when the pressure is withdrawn. The enlarged gall-bladder moves upward and downward with the respiratory play of the diaphragm and partakes of the movements of the liver. It may be greatly enlarged and elongated, and instances have been noted in which the contents have amounted to a litre. When the fundus presents toward the abdominal wall and a loop of intestine has found its way into the space between the fundus and the liver, the condition may simulate an echinococcus or ovarian cyst or hydronephrosis. If the walls of the abdomen are thin and relaxed, the tumor formed by a dilated gall-bladder may be visible. Urgent as the symptoms attending the disease which has caused the dilatation may have been, the condition itself usually causes no important subjective symptoms. When pain and tenderness are present they are commonly due to local adhesions and peritonitis.

Diagnosis.—DIRECT.—The diagnosis may be difficult. The anamnesis, the palpable and visible tumor connected with the liver and partaking of its movements, its cystic nature, its elasticity, its gourd-shaped outline, its mobility and tendency to at once resume the position from which it has been manipulated constitute adequate data for a positive diagnosis. The nature of the contents can only be ascertained by their removal. For this purpose an exploratory cœliotomy can be performed,—never an exploratory puncture, which is attended with the danger of the escape of a portion of the fluid into the peritoneal sac.

DIFFERENTIAL.—The diagnosis from an echinococcus cyst may be attended with great difficulty. The hemispherical form, more restricted movements, and hydatid thrill are significant. In hydronephrosis the deeper origin, relatively slighter mobility, except in floating kidney, and the outline are of diagnostic importance, and the occasional disappearance of the tumor with great diuresis seen in intermittent hydronephrosis would be distinctive. Ovarian cysts spring from the pelvis and can be shown by vaginal and bimanual examination not to be connected with the liver but with the uterus.

Prognosis.—In many cases it is favorable after the tumor has ceased to enlarge and is of moderate size. The inconvenience resulting from its presence and pressure upon adjacent organs, and the danger of adhesions and fistula formation, and, in particular, the danger of rupture justify drainage or excision. which is often followed by complete restoration to health.

CANCER OF THE BILE-DUCTS AND GALL-BLADDER.

Primary malignant disease of the gall-bladder is commonly associated with gall-stones—70 to 100 per cent. according to various statistics. The symptoms of the condition often gradually supervene upon those caused by biliary calculus. The fundus is often the starting-point of the growth, which early becomes manifest as a dense tumor in the region of the gall-bladder, developing downward and toward the median line, and not movable by reason of firm adhesions and implication of the surrounding tissue. The mass may attain large dimensions. The gall-bladder is sometimes greatly distended. The condition is much more common in males than females and commonly appears after the fortieth year of life. Pain is a prominent symptom. It is often paroxysmal but continues throughout the intervals. There is also more or less tenderness. Jaundice is present in a majority of the cases. It may be due to implication of the ducts or to pressure upon their walls.

Primary cancer of the bile passages is comparatively rare. The common duct is usually the starting-point of the growth, which may involve the walls of the ampulla of Vater and invade the hepatic and cystic ducts. There is rarely a palpable tumor. Jaundice occurs early and is persistent. If the carcinomatous infiltration involves the portal vein ascites results. There is often profound anæmia, but early cachexia may not be present. Cholæmia is a common terminal condition. Extension to the liver gives rise to symptoms characteristic of carcinoma of that organ.

CHOLELITHIASIS.

Gall-stone Disease.

Definition.—A condition characterized by the formation and presence of biliary calculi in the gall-bladder or bile passages. The great majority of gall-stones are formed in the gall-bladder.

Etiology. — PREDISPOSING INFLUENCES. — All conditions which give rise to the stasis of bile in the gall-bladder predispose to cholelithiasis. The outflow of bile may be impeded by partial or complete occlusion of the bile-ducts by catarrhal swelling of their mucous membrane, the presence of calculi or parasites, adhesions in the region of the porta, or compression by enlarged lymph-glands, the head of the pancreas, or the duodenum. Atrophy of the musculature of the gall-bladder from distention or age may lead to stagnation of the bile. Lacing plays an important part, (a) by restricting the movements of the diaphragm, (b) by causing elongation of the liver, displacement of the gall-bladder, and bending or twisting of the cystic duct, (c) by inducing changes in the anatomical relations which expose the cystic duct to compression, especially when there is, as is frequently the case, displacement of the right kidney, and (d) by causing gastroduodenal catarrh which may be followed by cholangitis and cholecystitis. Relaxation of the abdominal walls and enteroptosis favor the stagnation of bile in the gall-bladder. Lack of exercise, prolonged rest in bed in convalescence from acute or in chronic disease, and sedentary occupations constitute predisposing factors of importance, especially when combined with overfeeding and constipation. Cardiac affections, and in particular mitral stenosis, predispose to gall-stone disease by the passive visceral congestion and catarrhal processes to which they give rise and by the sedentary life which they enforce. There are great differences in the prevalence of gall-stone disease in different localities and different countries, as determined by post-mortem statistics,—a fact ascribed to local differences in mode of life, occupation, and the influence of endemic diseases, which by causing gastro-intestinal catarrh may become indirect factors in the production of calculi. The rare cases reported in the newborn and in infancy are simply clinical curiosities. The disease is rare under thirty. The liability increases progressively after forty. Women suffer more frequently than men in the proportion of 3 to 2. The pressure of the pregnant uterus upon the bile-ducts and its interference with the movements of the diaphragm, and the relaxation of the abdominal wall after frequent pregnancies are to be considered. The more sedentary life of women constitutes a predisposing influence of importance.

THE ORIGIN OF GALL-STONES.—The theory of Naunyn is generally accepted. A catarrhal condition of the mucosa of the gall-bladder, leading to an increased formation of cholesterin and lime salts, is the primary cause of the formation of gall-stones. This lithogenous catarrh may be produced by various causes, but the most important factor in its production is the presence of various bacteria. Among those which have been isolated are the colon bacilli, streptococci, staphylococci, pneumococci, and typhoid bacilli. They may gain áccess to the gall-bladder by way of the blood, or from the intestine by way of the common and cystic ducts. They have been demonstrated in the centre of a gall-stone. Cholesterin, lime salts, and bilirubin deposited around collections of epithelial débris and bacteria constitute the beginnings of biliary calculi. The masses thus formed grow in size by the gradual accretion of similar substances. Gallstones have been experimentally produced by the injection of cultures of bacteria into the gall-bladder of animals. The above facts account for the frequent occurrence of cholelithiasis after the acute infectious fevers, especially enteric fever.

THE CHEMICAL AND PHYSICAL CHARACTERS OF GALL-STONES.—Gallstones are composed chiefly of cholesterin, bilirubin in combination with calcium, and calcium carbonate. These constituents are present in varying proportions. Ordinary calculi consist of 70 to 90 per cent. of amorphous or crystalline cholesterin. The small, dark stones found in the ducts are principally composed of pigment in combination with calcium and calcium carbonate. Free bile pigment is not usually present. Traces of iron, manganese, and copper, and bile acids and fatty acids are also present. The color of the stones is not uniform, but irregular, and depends upon the quantity, character, and mode of deposit of the pigment which they contain. White or pale fawn-colored calculi consist of nearly pure cholesterin. Other stones may be yellow, greenish, or brown. An excess of pigment may give them a dark reddish-brown or black color. The cortex, main body, and nucleus are usually colored differently. The consistency also varies. Cholesterin stones are often so soft that they may be crushed between the fingers. Recently formed concretions are commonly soft; older ones may be harder with a soft central nucleus. The outer layer may be hard and enclose an unformed mass of cholesterin. The larger the proportion of lime salts the harder the calculus. Section usually shows the cholesterin to be deposited in concentric layers with radiating crystalline striæ, the result of recrystallization.

Gall-stones vary in number from one to hundreds or even a thousand or more. Single stones are usually ovoid and may be of large size—3 or 4 cm. or, in one reported instance, 7.5 cm. in long diameter. The solitary stone is usually closely embraced by the gall-bladder. Multiple calculi are commonly polygonal, with smooth, faceted surfaces, and owe this form to the pressure exerted among themselves while soft. Traces of faceting may be seen in the small calculi numbered by hundreds occasionally met with. It sometimes happens that a small number of ovoid, unfaceted calculi are found. They are of the dense variety and consist largely of the bilirubin-calcium combination. When this form is present in great numbers, the individual stones are not larger than small shot and are spoken of as gall-sand. Gall-stones impacted in the ducts sometimes undergo enlargement by further accretions of cholesterin and lime salts. Stones are found in this situation of such a size that they could not have passed through the cystic duct. Small, ovoid, greenish-black calculi are sometimes found in the intrahepatic bile-ducts, especially in cirrhosis of the liver. In a majority of the instances they are all of the same variety and composed of bilirubin-calcium. In fact, in cases in which numerous gall-stones are present, they are almost always all of the same variety.

Symptoms.—The subject of cholelithiasis may be clinically considered under the following headings: gall-stones quiescent in the gall-bladder; the symptoms which attend the passage of a stone through the ducts; the symptoms produced by the permanent obstruction of the ducts; ulcerative lesions caused by gall-stones; and gall-stones in the intestines.

1. **Gall-stones Quiescent in the Gall-bladder.**—In a great majority of cases biliary calculi, so long as they remain in the gall-bladder in which they are formed, produce no symptoms. Their presence in this viscus is frequently discovered at the autopsy in cases in which they have caused no manifestations whatever during life. According to Kehr symptoms occur in only about 5 per cent. of all cases. Persons who suffer from repeated attacks of biliary colic frequently have no trouble from them during the intervals. In a small proportion of the cases there are symptoms which suggest their presence, even though they are not sufficiently characteristic to justify a positive diagnosis. When, however, such symptoms occur during the intervals between attacks of colic, especially when such attacks have been followed by the passage of faceted calculi, their signif-

icance is clear. These symptoms consist of subjective sensations of weight in the right hypochondrium, aggravated some hours after taking food, frequent dull pain in the region of the gall-bladder radiating toward the right shoulder and the lumbar region, and nervous and mental derangements such as are common in neurasthenia—depression, irritability, precordial and epigastric distress, and headache, coryza, and flying neuralgic pains. Upon physical examination the gall-bladder may sometimes be found to be enlarged and palpable, and in very rare instances a tremitus caused by the movement of multiple calculi among themselves may be detected.

Gall-stones in the intrahepatic ducts rarely give rise to symptoms. If numerous or large they may occasion pain, enlargement of the liver, or jaundice, but these symptoms are not of diagnostic value. When infection takes place they cause diffuse intrahepatic cholangitis.

2. **The Symptoms Attending the Passage of a Gall-stone Through the Ducts.**—Gall-stones occasionally become arrested in the cystic or the common duct without causing pain. Small stones may traverse these passages without giving rise to colic. This has been observed in cases in which the repeated passage of larger stones is inferred to have caused a gradual dilatation of the ducts. When stones of a larger size are passed by the bowel in the absence of a history of colic, it is probable that they have reached the intestine by way of a fistulous communication with the gall-bladder or ducts.

Biliary Colic.—Commonly the passage of a gall-stone is attended by the symptoms of gall-stone colic. The attack usually begins with violent pain in the right hypochondrium with its focus of intensity in the region of the gall-bladder. In some cases the pain is referred to the epigastrium or the lower thoracic region, or on both sides, or to the right mammary region. It may radiate toward the abdomen or back, and occasionally to the right shoulder. It is usually agonizing and the patient groans and rolls about in uncontrollable distress, or he may twist his body to the right, or sit with his thighs and knees strongly flexed and his body bent forward so as to relax the abdominal muscles. There may be temporary remissions of pain which are followed by exacerbations of greater violence. The gall-bladder is often palpable and tender, and the liver may be somewhat enlarged, with tenderness over the hepatic area. Vomiting, chills or chilliness, a rise of temperature sometimes to 103°–104° F. (39.5°–40° C.), profuse sweating, and great general relaxation occur. In cases marked by high fever there may be enlargement of the spleen and febrile albuminuria. It is probable that there are under these circumstances bacterial invasion and acute cholecystitis. The fact that the symptoms of gall-stone colic are sometimes present in acute cholecystitis without gall-stones is not to be overlooked. Jaundice is a common symptom. It does not occur so long as the stone is engaged in the cystic duct, but follows the lodgement of the stone in the common duct. When the stone is of small size and passes rapidly through the common duct into the intestine, jaundice may not occur. In any case jaundice does not occur until several hours, often twenty-four, have elapsed from the beginning of the attack. It is usually transient, but may persist for several days or weeks. The jaundice is the very type of obstructive jaundice and is associated with clay-colored stools, the presence of bile pigments in the urine, itching of the skin, and other characteristic symptoms.

The duration of the attack varies from a few hours to several days. When the stone escapes into the intestine the pain ceases, often as abruptly as it began, leaving some degree of local tenderness, which rapidly subsides, and lassitude, from which the patient gradually recovers. Not rarely the stone lodges in the ampulla of Vater and acts as a ball-valve, causing recurrent attacks of pain and jaundice. The pain is a true colic caused by the spasmodic contraction of the musculature of the bile-ducts and the violent pressure of the stone upon the mucous membrane. The swelling and tenderness of the gall-bladder and liver are due to bile stasis and consequent distention of these organs. In bacillary invasion there is the superadded pain of inflammation. Rare accidents are fatal syncope and the rupture of the gall-bladder into the peritoneal sac. Palpitation and precordial distress may occur, while general convulsions and hysterical seizures are occasionally observed in neurotic subjects.

Direct Diagnosis of Biliary Colic. — The diagnosis rests upon the location of the focus of pain, its radiation, local tenderness, the abrupt onset of the attack, vomiting, chill or chilliness, with fever and the symptoms of obstructive jaundice. The history of previous attacks is suggestive; the presence of gall-stones in the stools is conclusive. Their absence is, however, only of negative importance in diagnosis. It may be due to a faulty method of examination, to the return into the gall-bladder of a stone which has engaged in the cystic duct, to cessation of muscular spasm in the walls of the ducts, to the passage of the stone from the narrow cystic duct into the wide common duct and its retention there, and, finally, to the disintegration of the stone in the intestine.

The stools must be thoroughly stirred with a large quantity of water and poured through a fine-meshed sieve. The coarser particles are retained and can be examined. A double bag of netting may be arranged upon a stout wire ring like a landing net and placed in the bowl of the water-closet. The fæces may be washed by repeated flushing and the retained particles examined for calculi. If they are not at first found every stool should be examined for several days, as they may be retained in the intestine for some time. Force should not be used in the examination, since recently formed biliary calculi are soft and may readily be disintegrated in handling them. The seeds of various fruits, particles of bone, and small fecal concretions are sometimes brought to the physician as gall-stones, and the rounded saponaceous masses voided after the ingestion of large quantities of olive oil are frequently mistaken for them, but these substances never contain cholesterin or bile pigment in quantity, nor do they present the internal structure of gall-stones.

Differential Diagnosis.—In *right-sided renal colic* the pain begins in the lumbar region and radiates toward the groin. There is retraction of the testicle and pain in the glans penis. Jaundice, tenderness in the region of the gall-bladder, and fever are not usually present. A calculus may be voided by way of the urethra. *Peptic ulcer* may suggest biliary colic. The pain, however, usually follows the ingestion of food and is burning in character, passing to the back. The vomiting is less urgent and the vomitus may contain blood. There is localized epigastric tenderness and anæmia. *Nervous hepatic colic*—the pseudobiliary colic of nervous women—may lead to an erroneous diagnosis. The pain is referred to the right side and may radiate to the back or shoulder. It is dull and dragging rather than colicky. The attack follows emotional excitement or fatigue. There may be tenderness upon pressure, but jaundice does not occur. *Intestinal colic* is relieved by belching, the passage of flatus, or defecation. It is more generalized and less intense than biliary colic and not followed by jaundice. *Lead colic* may simulate gall-stone colic, but the occupation of the patient is suggestive, while stubborn constipation, the gingival line, wrist-drop, hard arteries, and albuminuria constitute a characteristic symptom-complex. Jaundice is absent.

3. **The Symptoms Caused by Permanent Obstruction of the Ducts by Gall-stones.**—The obstruction may involve the cystic duct, the common duct, or the hepatic ducts.

I. OBSTRUCTION OF THE CYSTIC DUCT.—Occlusion of the cystic duct by a calculus or by the contraction of a cicatrix following ulceration does not always cause serious symptoms. It is liable to be followed by, (a) *dropsy of the gall-bladder—hydrops vesicæ felleæ.* The tumor is cystic and gourd-shaped or pear-shaped, its narrow extremity being at its connection with the liver. The contents in recent cases are bile mixed with mucus or mucopus,—in older cases a clear, thin mucus containing albumin and of variable reaction to litmus paper. The tumor projects downward and may attain large dimensions. It is freely movable from side to side, unless fixed by adhesions, and when pushed backward turns to its original position as soon as the pressure is withdrawn. When the belly wall is thin and relaxed the outline of the distended gall-bladder may be visible, fluctuation may be elicited upon light bimanual percussion and palpation, and when there are many calculi present gall-stone crepitus may be felt. (b) *Atrophy of the Gall-bladder.*—This condition frequently follows dropsy of the gall-bladder. The contents undergo gradual resorption and the bladder contracts around any stones that it may contain, or, in the absence of a stone, into a small fibrous mass, or there may be diverticula in which calculi are embedded. In old cases of this kind lime salts are sometimes deposited upon the mucosa or in the bladder wall. (c) *Acute cholecystitis*, usually simple *but in rare cases phlegmonous.* (d) *Suppurative cholecystitis*—empyema of the gall-bladder. The gall-bladder may be greatly enlarged and contain as much as a litre of pus. Perforation may take place into the peritoneal cavity; more commonly adhesions take place with abscess formation.

The occurrence of these conditions may constitute the first direct evidence of cholelithiasis. Under no circumstances should exploratory puncture be performed. Aspiration has been followed by fatal results.

II. OBSTRUCTION OF THE COMMON DUCT.—The duct may be occluded by a single stone in the ampulla of Vater or in any part of its course or by a number of stones which may also extend into the cystic and hepatic ducts. The obstruction may be, (a) *complete.* The calculus is tightly impacted in the common duct, or a large stone in the cystic duct compresses the common duct at its upper part or the hepatic duct. There is complete bile stasis with deep and persistent jaundice and without septic phenomena. The common duct behind the obstruction, and the cystic and hepatic ducts may be enormously dilated and simulate the gall-bladder, for which they have been mistaken. The condition cannot always be differentiated from compression of the duct by new growths, though pain, a history of biliary colic, and absence of dilatation of the gall-bladder are in favor of a diagnosis of complete obstruction by gall-stones. Or, (b) *incomplete.* In this form there is cholangitis, which may be simple or suppurative. (*a*) Incomplete obstruction with non-suppurative cholangitis. There may be a single movable calculus in the diverticulum of Vater or in the duct above it,—ball-valve mechanism,—or a small faceted stone partially impacted, or a series of stones. The ducts above the obstruction are dilated, but the gall-bladder is often contracted. There are variations in the degree of jaundice and in the amount of bile pigment in the fæces. The liver is only slightly enlarged and the gall-bladder, as a rule, not at all distended. Finally there are irregular attacks of fever accompanied with demonstrable enlargement of the spleen. In well-marked cases of ball-valve calculus the paroxysms of fever are irregularly recurrent and resemble attacks of ague. They are characterized by remarkable rises of temperature,—103°–106° F. (39.5°–41.1° C.),—intense chills, profuse sweating, gastric disturbances and hepatic tenderness and pain. The jaundice is variable and often intense. The resemblance to malaria is superficial, the periodicity not being regular, the blood parasite not present, and quinine useless. This fever is known as hepatic fever or the hepatic intermittent fever of Charcot. The attacks in many instances recur after irregular intervals, during which the temperature is normal, for many months.

Courvoisier's Law.—In the great majority of cases of obstruction of the common duct by gall-stone the gall-bladder is contracted; in the majority of cases of obstruction from other causes the gall-bladder is dilated.

(*β*) Incomplete obstruction with suppurative cholangitis. The ducts are invaded by pyogenic organisms. The suppurative inflammation may extend to the intrahepatic ducts—diffuse intrahepatic cholangitis—and to the gall-bladder—empyema. Abscess of the liver and perforation of the gall-bladder with abscess formation may occur. There are septic phenomena. The liver is enlarged and tender; jaundice is of moderate intensity and persistent and there is fever of intermittent or remittent type. The course of the disease is comparatively short and the termination fatal. This is by no means rare as a terminal condition in old cases of cholelithiasis.

In cholelithiasis of the common duct there is frequently an associated catarrhal or interstitial pancreatitis and Cammidge's test may show characteristic crystals.

4. **Ulcerative Lesions Caused by Gall-stones.**—Biliary fistulæ are far from uncommon. Ulceration of the bile passages may occur without symptoms. As a rule, however, they tend to grave derangements of health. By the erosion of arterial branches in the course of the formation of fistulous tracts in various directions, they may cause hemorrhages which may be latent or manifest in the stools or vomit. In rare instances gall-stones

have perforated into the portal vein. Much more common are fistulous communications with the intestinal tract. The stomach is involved comparatively rarely, the duodenum frequently, the small intestine much less commonly, while fistulous communications with the colon have been occasionally encountered. There are instances of fistulæ involving the ureters, with the passage of stones into the bladder and of the direct passage of biliary calculi into the urinary bladder. Perforation into the pleura and into the lung may also occur. Cutaneous fistulæ of spontaneous origin are very uncommon, though they are by no means rare after operation. They usually open in the region of the fundus of the gall-bladder, but may appear near the umbilicus or above the pubes. The formation of these ulcerative tracts is always preceded by adhesions between the viscera directly involved and followed by the discharge of biliary calculi into the distant organ. The tracts themselves are often long and tortuous and sometimes there are diverticula containing gall-stones. Abscess formation is common.

5. **Gall-stones in the Intestines.**—When by way of the common duct, as sometimes may occur, or by a fistulous tract, a large gall-stone finds its way into the gut, it may cause intestinal obstruction, either directly or by ulceration followed by cicatricial contraction. The obstruction may occur at the pylorus and cause symptoms suggestive of carcinoma. More commonly it is in the lower part of the ileum. In the region of the cæcum the condition may simulate appendicitis. A small stone may enter the appendix or a diverticulum. A stone in the colon may sometimes be recognized upon rectal examination.

Diagnosis.—Cholelithiasis is recognized during life in a small proportion of the cases only. The direct diagnosis while the gall-stones remain quiescent in the gall-bladder may, in rare instances, be made by the discovery of a tumor in the region of the gall-bladder in which gall-stones may be felt. The walls of the bladder may be thickened by chronic inflammation or the seat of nodular carcinomatous growths. The differential diagnosis between an enlarged gall-bladder and a tumor of the kidney depends upon the fact that the former may be pushed backward into the abdomen, but directly returns to its former position, while the latter will remain in the position into which it is replaced.

Cholelithiasis cannot, as a rule, be demonstrated by the X-rays, since the stones are mostly composed of cholesterin and organic matter which do not cast a shadow, and such shadows as are cast by those more densely constituted are obscured by the shadow of the liver.

The attack of colic is commonly the first positive sign of gall-stone disease. The symptoms of biliary colic and the differential diagnosis between that affection and others which resemble it have already been discussed. The discovery of gall-stones in the stools constitutes a positive diagnostic sign.

Exploratory puncture of the gall-bladder is attended with the danger of the escape of some of the contained fluid into the peritoneum and a general peritonitis. It is therefore to be emphatically condemned. An exploratory operation by incision is, on the other hand, comparatively safe and wholly justifiable in the presence of obscure and dangerous symptoms.

Prognosis.—The fact that in a large majority of the cases no symptoms occur, and that the diagnosis is so often simply a post-mortem finding, justifies the assertion that cholelithiasis is a benign disease. Although it is so often latent it is not, however, free from danger. On the contrary, the migration of the stones by way of the natural passages and, to a greater extent, by artificial channels caused by ulceration and adhesions may be attended by great suffering and serious risk of life. Even under these circumstances the outlook is not wholly unfavorable. When infection of the bile passages and gall-bladder has taken place with septic fever, especially when empyema of the gall-bladder, suppurative cholangitis, or abscess of the liver has occurred as a complication, the prognosis is highly unfavorable. The general recognition of the fact that cholelithiasis is a surgical disease has rendered the prognosis far more favorable than it was at a period when the sole dependence was upon drugs and mineral waters.

vii. Affections of the Blood-vessels of the Liver.

1. **Anæmia.**—Nothing is known of anæmia of the liver as a clinical condition.

2. **Hyperæmia.**—Two forms are to be considered, active and passive.

(a) **Active Hyperæmia; Congestion of the Liver.**—The liver is one of the most vascular organs of the body. The rapid influx of blood by way of the portal vessels during digestion brings about a physiological hyperæmia which is transient, but which in the cases of persistent overindulgence in the pleasures of the table may become the cause of functional or even of organic changes. Excessive quantities of food, strong spices, coffee, and especially alcohol are credited with causing hyperæmia of the liver, a condition to which sedentary habits also contribute. The condition thus caused constitutes a conspicuous feature in general plethora and obesity, and is frequently associated with gout, gravel, and glycosuria. Toxic agencies such as are present in the acute fevers and malaria, and, indirectly, the mode of life common among Europeans in tropical climates are causal factors.

SYMPTOMS.—The manifestations of active hyperæmia of the liver are indefinite and rarely present alone. They consist of sensations of pressure and fulness in the right hypochondrium and epigastrium, which may amount to actual pain and are sometimes intensified by movement, deep respiration, and pressure. They are associated with the evidences of enlargement of the organ and certain symptoms of deranged digestion, such as epigastric weight, heart-burn, flatulence, and fulness in the head. In some instances a periodical recurrence of this group of morbid phenomena accompanies the return of menstruation or the suppression of the flow, or the arrest of an habitual hemorrhoidal flux. A slight icterus frequently accompanies the attack of hyperæmia of the liver. The tendency to the development of organic disease as a result of permanent hyperæmia is to be considered.

(b) **Passive Congestion.**—This form of hyperæmia is much more common. All conditions which favor the accumulation of blood in the venous system and the transference of blood-pressure from the arterial to the venous side of the circulation lead to passive hyperæmia of the liver, which constitutes, in fact, an important part of the general visceral congestion characteristic of such states. Cardiac affections, both valvular and myocardial, especially when the power of the right ventricle is diminished, are important etiological factors. Pulmonary diseases which cause mechanical interference with the pulmonary circulation, such as acute and chronic bronchitis, emphysema, consolidation of the lung from exudate or sclerosis, tumors, and extensive pleural effusion or adhesions, also give rise to passive hepatic congestion, partly by reducing the calibre of the pulmonary vessels and partly by restricting the respiratory excursus. Deformities of the spinal column may act in the same way. Local hyperæmia is seen in the corset liver in the part separated from the main organ. The liver is enlarged, dense, and of a deep brownish-red color—the cyanotic or cardiac liver. Later it may be contracted, owing to the overgrowth of connective tissue.

Symptoms.—In the early stages symptoms are absent or subordinated to those of the general condition. Later epigastric fulness, especially after eating, dyspeptic disturbances and hemorrhoids occur. Hæmatemesis may occur. The portal obstruction may cause ascites. A mild jaundice is common. The stools may be clay-colored, and bile pigments may be present in the urine. Physical examination shows the liver to be enlarged. It is usually tender, especially below the arch of the ribs or upon pressure with the flat hand. Liver pulsation is often present.

Diagnosis.—The passive hyperæmic liver varies in size, whereas the enlargement of the liver from other lesions is persistent. This fact, considered in connection with the various etiological factors above enumerated, has great diagnostic significance.

3. **Diseases of the Portal Vein.** — (a) Occlusion or narrowing of the portal vein may be caused by, (1) acute or chronic inflammation of the vessel wall or by its invasion by a neoplasm; (2) compression from without by tumors, enlarged lymph-glands, gall-stones, or the cicatricial contraction of the adjacent parts, especially such as result from syphilis and tuberculosis; (3) mechanical obstruction caused by Distoma hæmatobium; and finally (4) from other causes not clearly understood. (b) Thrombosis.—The foregoing conditions cause slowing of the blood stream in the portal vein and thus favor thrombus formation, which may occur in cirrhosis, syphilis of the liver, malignant growths involving the wall of the vein, hyperplasia of the lymph-glands in the porta, compression or perforation of the walls of the vessel by hepatic calculi, parasite invasion, and arteriosclerosis. The coagulation forms a wall-thrombus, which may partially or wholly occlude the vein. (c) Adhesive Pylephlebitis.—In rare instances a collateral circulation is established, the thrombus becomes thoroughly organized, and the vein is converted into a fibrous cord.

Symptoms.—The condition manifests itself by acute symptoms which may supervene in the course of hepatic cirrhosis, chronic peritonitis, or abdominal tumor, or occur suddenly in persons apparently well. These symptoms consist of sudden intense epigastric pain with hæmatemesis, melæna, followed, in the course of a few days, by ascites and enlargement of the spleen. Icterus is sometimes present.

(d) Suppurative Pylephlebitis.—This condition is due to infection by pyogenic bacteria. It is secondary to intestinal ulceration such as occurs in dysentery, enteric fever, or tuberculosis, to suppurative foci, as appendicitis, pelvic abscesses, ischiorectal abscess, inflamed hemorrhoids, or fistula. Multiple abscess formation occurs within the branches of the portal vein. Septic phenomena are rapidly developed. They consist of irregular chills, fever of rapid and extreme oscillation of temperature, colliquative sweating, and profound asthenia. Local symptoms are not constant. There may be tenderness and pain over the liver and spleen, the latter being enlarged. Icterus is not usually marked.

4. **Diseases of the Hepatic Artery.**—(a) Dilatation occurs in cirrhosis of the liver. (b) Sclerotic changes in the wall are common. These conditions are of pathological rather than of clinical interest. (c) Aneurism is infrequent. A number of cases have been studied clinically. The diagnosis is obscure. Important symptoms are pain, hemorrhage from the bowel and uterus. A pulsating tumor has never been found. The cases closely simulate gall-stone disease or duodenal ulcer. The combination of the symptoms of these two conditions is of diagnostic significance. Death may occur suddenly from hemorrhage into the gastro-intestinal tract or into the peritoneum.

5. **Diseases of the Hepatic Veins.**—(a) Dilatation occurs when the right heart is permanently dilated and hypertrophied. (b) Stenosis is far less common. It may be due to, (1) compression by tumors, especially gummata and enlarged lymph-nodes; (2) disease of the vessel walls; (3) thrombosis; and (4) embolism. The symptoms are obscure and the clinical diagnosis is uncertain. The spleen is palpable, the liver enlarged, and ascites occurs. (c) Thrombosis may occur as a result of stenosis. (d) Emboli may pass into the hepatic veins from the right auricle when the blood current is reversed, as may occur in tricuspid insufficiency. The subsequent course of embolism and thrombosis of the hepatic veins varies according to the nature of the cause of the occlusion, namely, whether it be simple, infected, or the result of malignant disease. Infarcts are occasionally encountered.

viii. Abscess of the Liver—Suppurative Hepatitis.

Definition.—Suppuration within the liver, either in the parenchyma or in connection with the blood-vessels or bile passages.

The following forms occur: (1) solitary abscess, (2) embolic or pyæmic abscesses, (3) suppurative pylephlebitis, (4) suppurative cholangitis, (5) local abscess formation caused by foreign bodies or parasites.

The distinction sometimes made between primary and secondary abscesses of the liver cannot always be observed. Those abscesses due to traumatism or the extension of suppurative processes from the gall-bladder or bile-ducts are primary; those in which infection has taken place by way of the blood stream are secondary.

Etiology.—Predisposing Influences.—Residence in the tropics, especially when associated with excesses at table and overindulgence in alcohol, amœbic dysentery, cholelithiasis, appendicitis, traumatism in the region of the liver and blows upon the head, ulceration of the intestines and suppurative processes in the pelvis, general sepsis with metastatic abscesses, and echinococcus cysts in the liver are all predisposing factors of importance.

THE EXCITING CAUSE. Infection of the liver substance, the blood-vessels, the bile-ducts, or the gall-bladder by pyogenic organisms is the direct cause of hepatic abscess. The avenues of infection are the portal vein, the hepatic vessels, the common duct, and penetrating wounds or fistulous tracts. The organisms found in hepatic abscess comprise *Amœbæ dysenteriæ*, streptococci, colon bacilli, pneumococci, typhoid bacilli, *Bacillus pyogenes*, and actinomyces.

Morbid Anatomy. Tropical abscess is usually solitary, though two or more abscesses are occasionally encountered. When single, the abscess is commonly situated in the right lobe, extending to the upper surface, less frequently toward the concave surface of the organ. Recent abscesses, as seen at the surface of the liver, are of a grayish-yellow color with a well-defined outline. In a more advanced stage the walls are shreddy and necrotic and contain a greenish- or reddish-brown viscid pus commingled with blood, in which there are fragments of liver tissue. This fluid shows fatty and granular detritus, cellular elements, occasionally Charcot-Leyden crystals, and amœbæ, which are also present in the tissue forming the wall. Cultures are commonly sterile. The abscess wall consists of an inner necrotic layer, a middle layer in which there is proliferation of connective tissue, and an outer layer of intense hyperæmic tissue. The chronic abscesses frequently have an extremely dense wall. Rupture may occur into the lower lobe of the right lung, into the pleura (causing an empyema), into the vena cava, the portal or hepatic veins, or into the stomach, intestine, peritoneum, pericardium, or externally. Perforation into the right kidney is a rare event. Traumatic abscess is usually single and has no distinctive anatomical characters. Pyæmic abscesses are almost always multiple and may be very numerous. When large they are due to the coalescence of several small cavities. The infected emboli find their way to the liver through the hepatic artery or form infected thrombi in the portal vein. Echinococcus cysts undergo suppurative changes as the result of infection following trauma or inflammation of the bile passages. The abscesses may be of great size, and contracted hydatids, dead scolices, shreds of the cyst wall, fat, and bilirubin may be found in the pus.

Symptoms. 1. SOLITARY ABSCESS.—This condition is common among Europeans in the tropics and chiefly affects males. It occurs also in the temperate zones. It is almost always associated with amœbic dysentery, though cases occur in which no history of dysentery can be obtained. The abscess may present no definite symptoms and finally reveal its presence by rupture which may prove fatal. The principal symptoms are fever, pain, tenderness, and septic phenomena. Enlargement of the liver may be [illegible] The temperature is irregular. There is paroxysmal [illegible] regular, sometimes of irregular, [illegible] ing to subnormal ranges [illegible] F. (39.5°–40.5° C.), [illegible] frequently simulates a [illegible] cases fever may be absent. [illegible] the epigastrium or the [illegible] often associated with sen- [illegible] much increased when the patient turns upon his left side, and with tenderness upon pressure at the costal margin in the mammillary line. The shoulder pain is due to irritation of the terminal filaments of the right phrenic nerve which are distributed to the capsule of the liver. The stimulus is transmitted to the fourth cervical, which anastomoses with the phrenic and sends sensory branches to the shoulder. The facies of the patient is sallow, pallid, muddy, and faintly icteroid. Irregular diarrhœa, digestive disturbances, and great mental depression are common. Leucocytosis is sometimes present, often absent, and therefore not usually of diagnostic value. Enlargement of the liver is most marked in the right lobe and upward. This condition is in contrast with many diseases of the liver in which the enlargement is in a downward direction, as fat infiltration, hypertrophic cirrhosis, and carcinoma. The upper margin of liver dulness is usually higher in the back near the spine than anteriorly. In extensive abscess formation the lower margin of the liver may extend a hand's breadth below the edge of the ribs. The entire right hypochondrium may bulge, the ribs protrude and be widely separated, and fluctuation may be detected. Palpation may be painful. The margin of the liver is felt to be rounded and blunt, the superficial venules over the liver may be dilated, and there may be circumscribed cyanosis with œdema. Upon deep respiration a friction fremitus may sometimes be detected. Perforation into the lung may occur or amœbic infection through the diaphragm without rupture. The base of the right lower lobe shows signs of consolidation, there is intense paroxysmal cough, with characteristic expectoration resembling anchovy sauce and containing Amœbæ coli in varying numbers and actively motile. The color of the expectoration is due to altered blood. The sputum may be blood-tinged or bright red and very abundant. It may contain pus and shreds of liver tissue. Recovery may take place in the course of several weeks. Perforation into the pleura causes empyema with characteristic symptoms and physical signs; into the pericardium is followed by fatal collapse; into the stomach gives rise to vomiting of blood-tinged fetid pus; into the intestine to the presence of pus in the stools. Coincident with the escape of pus in these various directions there is sudden subsidence of the liver tumor, together with sensations of collapse. Rupture into the vena cava is followed by rapid death with symptoms of asphyxia. In the rare cases of perforation of a liver abscess into the pelvis of the right kidney, the urine contains pus of a brownish-red color which may show the presence of liver-cells or blood-corpuscles.

2. Embolic or Pyæmic Abscesses.—The multiple small abscesses in the liver, which occur in some cases of general septicopyæmia, may cause pain and tenderness in the hepatic region and a slight subicteroid discoloration of the skin. When these symptoms are superadded to the rigors, high temperature, sweating, and prostration of the septic condition, and especially when a suppurative focus can be discovered, the diagnosis of metastatic abscesses in the liver may be made.

3. Suppurative Pylephlebitis.—The clinical manifestations are the same as in pyæmic abscess. The liver is enlarged and tender, there is fever of septic type, and a muddy, icteroid skin.

4. SUPPURATIVE CHOLANGITIS.—The history of attacks of gall-stone colic or of the recovery of gall-stones from the stools, and the symptoms of cholangitis or the presence of a distended gall-bladder are important for the diagnosis.

5. FOREIGN BODIES AND PARASITES.—Needles have penetrated the wall of the œsophagus or stomach and entered the liver substance, causing abscess. A needle or a fish-bone has been known to perforate a branch of the portal vein and give rise to pylephlebitis. The part played by echinococcus cysts in producing liver abscess has been described. The penetration of round worms into the common duct and, less commonly, the presence of flukes have caused suppurative processes. These parasites probably act merely as carriers of pyogenic germs.

Diagnosis.—DIRECT.—Abscess of the liver is often latent and wholly overlooked, notwithstanding the fact that the patients almost always present the appearance of serious illness. This is due to the fact that local symptoms may be altogether absent or subordinated to those of the primary disease. When pain, tenderness, and enlargement of the liver are present, and a source of infection can be found in the structures tributary to the portal vein; or a focus of purulent inflammation elsewhere; or when there is a history of traumatism involving the liver, or of bone injury, especially injury to the bones of the skull, or of cholelithiasis, a positive diagnosis may be made. Nevertheless the symptoms are frequently obscure, and the diagnosis, even when abscess is suspected, cannot in many cases be positively determined. This statement is especially true of abscesses of moderate size centrally situated in the substance of the liver, and of the multiple small abscesses which occur in general septic conditions and in acute cholangitis and pylephlebitis. Of positive diagnostic import are pain referred to the region of the liver and the right shoulder, enlargement of the liver in an upward direction, bulging, particularly when circumscribed, and fluctuation. Examination by the X-rays yields, as a rule, unsatisfactory results, owing to the density of the liver, but in exceptional cases may be of service.

DIFFERENTIAL.—The following conditions are to be considered: (a) *Malarial Fever.*—The regularly intermittent fever often closely simulates malarial fever, but the absence of marked splenic enlargement and of the malarial blood parasite, and the failure of quinine to control the fever are of diagnostic significance. (b) *Right-sided Empyema.*—When the abscess ruptures into the pleura an empyema is produced, but perforation of the lung commonly follows, and the true nature of the condition is revealed upon the expectoration of pus resembling anchovy sauce and containing amœbæ. (c) *Subphrenic Abscess.*—The downward displacement of the liver, the fact that the lower border of the lung descends upon deep inspiration, and a history of gastric ulcer may be of service in the differentiation from hepatic abscess. When gas is also present,—pyopneumothorax subphrenicus,—the diagnosis is less difficult. (d) *Abscess of the Abdominal Wall.*—Perforation through the abdominal wall presents little difficulty. A mural abscess may closely simulate liver abscess. Such lesions, usually associated with tuberculosis of the ribs, are superficially situated, and an exploratory needle does not follow the movements of respiration. The liver is not enlarged. (e) *Empyema of the Gall-bladder.*—The situation of the tumor, its gourd-like outline, and some degree of lateral movement upon pressure are important points in diagnosis. (f) *Echinococcus Cysts.*—Slowness of growth, absence of fever and signs of inflammation, very obscure fluctuation, and the hydatid thrill are characteristic of these cysts. When they become infected the differential diagnosis from hepatic abscess is attended with great difficulty. The presence of hooklets or shreds of cyst walls in the pus is of positive diagnostic significance. (g) *Abscess of the Left Lobe.*—An abscess in this position is uncommon. It may, in the absence of inflammatory symptoms, simulate carcinoma ventriculi, from which it may be distinguished by the age of the patient, the history of the case, an examination of the gastric contents, and the presence of foci of infection in the intestines or elsewhere. (h) *Aneurism of the Aorta.*—Hepatic abscess to which the movements of the aorta are transmitted may simulate an aneurism in the region of the cœliac axis. In abscess the pulsation is to and fro, not expansile, diastolic shock is absent, a bruit is not heard, and the pain is less severe and paroxysmal than in aneurism and has a different focus of maximum intensity. (i) *Hepatic Fever.*—The intense fever with chills and sweating which occurs in certain cases of gall-stone disease is frequently regarded as due to abscess of the liver. It is now known that this symptom may occur in the absence of suppuration. The fever occurs in paroxysms, which may have a regular periodicity or recur at irregular, often prolonged, intervals, during which there is complete apyrexia and the nutrition may be fairly well maintained. The varying jaundice, which is intensified during the febrile attacks, is suggestive, and the long duration of the condition is of diagnostic importance.

Exploratory puncture may be made in a doubtful case. The patient must be etherized and the skin cleansed as in any surgical operation. The aspirator needle, which should be of large calibre, should be introduced at the suspected point in the infra-axillary region or over the area of hepatic dulness behind. Repeated puncture may be necessary.

Prognosis.—The outlook in pyæmic abscesses is ominous. In diffuse suppurative cholangitis and pylephlebitis and in traumatic abscess it is highly unfavorable. In solitary, tropical abscess it is much more hopeful. Recovery may take place after aspiration or incision. In the more chronic cases of solitary abscess, perforation into the lung or the intestine or through the skin may be followed by recovery. In individual cases marked septic phenomena, persistence of dysenteric symptoms or of suppuration in pelvic abscess or bone disease, the supervention of amyloid disease, and the development of cachexia are highly unfavorable. The danger of rupture into the vena cava, the pericardium, or the peritoneum renders the prognosis uncertain.

ix. Fatty Liver.

Definition.—This term is used to designate all conditions characterized by an abnormal increase in the fat of the liver.

Under physiological conditions the fat in the liver-cells varies and is dependent upon the amount and character of the food. The ingestion of large quantities of fat is followed by an increase of fat-globules in the peripheral cells of the acini. This increase is transient, disappearing in the course of several hours.

Pathologically two different forms of fatty liver occur—fatty infiltration and fatty degeneration. These forms sometimes coexist. Fatty infiltration is the result of an increased deposit of fat in the parenchyma of the liver in the absence of fatty change in the protoplasm of the cells.

Fatty degeneration consists of a destruction of the protoplasm of the cells with fat accumulation.

Etiology.—Fatty infiltration occurs in general obesity and in persons who habitually consume inordinate amounts of fats and carbohydrates; at the middle periods of life; and in many women after the menopause. It is common in conditions in which there is deficient oxidation, marked anæmia, advanced phthisis, or the cachexias. Chronic alcoholism leads to fatty degeneration of the liver by interfering with the oxygenation of fats and carbohydrates, the oxygen being largely required for the combustion of the alcohol consumed. Fatty degeneration is caused by certain poisons, as phosphorus, the toxin of acute yellow atrophy, and arsenic, mercury, and antimony.

Morbid Anatomy.—The ordinary fatty liver—fatty infiltration—is uniformly enlarged and may reach double the normal weight. It is smooth, of a pale yellow or drab color, and greasy. In fatty cirrhosis the surface shows irregular granular prominences. On section the light yellow color and empty blood-vessels are noticeable and the knife is smeared with grayish-white fat. Microscopically the cells are distended with small and large fat droplets.

Symptoms.—Clinically fatty infiltration is to be distinguished from fatty degeneration. In the former the parenchyma is not degenerated but simply contains an excess of fat. The hepatic functions are therefore maintained, and general symptoms do not occur. In the latter the liver-cells are affected by a degenerative process, their functions are no longer performed normally, and grave symptoms are present, as in acute yellow atrophy, q.v., and phosphorus poisoning. The symptoms in fatty infiltration are not well defined. The appearance of the patient and the symptoms referable to other organs are dependent upon the primary disease. Jaundice does not occur. The stools may be light-colored or even whitish-gray, and putty-like in consistency. The urine does not contain bile pigments in excess. Hemorrhoids may occur, but the signs of portal obstruction are not common. In obese persons the physical signs are often obscure, but in phthisis and cachectic conditions the greatly enlarged liver may be sometimes visible through the belly wall and almost always recognized upon palpation. It is smooth, with a rounded lower border, projecting far below the margin of the ribs, and is painless.

Diagnosis.—Slight grades of fatty liver cannot be recognized during life. When the condition is well developed it is, except in the case of great obesity, easy of recognition. The great enlargement, smooth surface, and nearly normal contour are characteristic. The underlying disease, as anæmia, phthisis, the various cachexias, is of diagnostic importance. The soft consistence enables us to exclude amyloid liver, leukæmic tumors, and hyperæmia.

Prognosis.—Fatty liver is a secondary pathological condition, and the prognosis is that of the primary disease.

x. Chronic Interstitial Hepatitis.

Cirrhosis of the Liver.

Definition.—A chronic disease of the liver due to various toxic or infectious causes, mechanical irritation, or stasis of blood or bile, and characterized by an overgrowth of the interstitial connective tissue of the organ.

It is of interest to investigate the etymological significance of this unfortunate word "cirrhosis," originally selected by Laennec to describe an anatomical peculiarity of the contracted liver far from constant, and subsequently applied to changes in other viscera attended by an overgrowth of connective tissue, so that there have been those who have spoken of cirrhosis of the kidney, cirrhosis of the lungs, and the like. The word is derived from the Greek *κιρρος*, tawny or orange-yellow, the color of the liver substance in some of the cases. It has nothing whatever to do, save by a remote and misleading association, with the sclerotic changes due to hyperplasia of connective-tissue stroma.

The difficulties in the diagnosis of hepatic cirrhosis are not, as in most other diseases, accidental. They do not arise from the peculiarities of individual cases. They are essential and due to the fact that no definition can be framed that at once pathologically and clinically includes all the cases. This arises from Laennec's choice of a term descriptive of an inconstant phenomenon and without pathological significance. "Cirrhosis" is conspicuous among the nosological terms that hamper medicine and obstruct the progress of knowledge. If it could be erased from the list of diseases our conception of the various conditions to which it is applied would be greatly simplified. The one underlying lesion common to all of them is an overgrowth of the fibrous tissue of the liver. The term chronic interstitial hepatitis is at once descriptive of the anatomicopathological condition and sufficiently comprehensive to include all the cases. Its more general use is to be desired.

Etiology.—The etiological relations of individual cases may be indicated by qualifying adjectives:

(a) In general toxic, and in particular alcoholic, plumbic, gouty, diabetic, rachitic, and the like. We must include here forms of interstitial hepatitis due to chronic phosphorus poisoning and the abuse of condiments. The assumption that intestinal autointoxication gives rise to interstitial hepatitis requires confirmation.

(b) In general infectious; in particular the specific febrile infections are occasionally followed by chronic interstitial hepatitis. Malaria produces similar changes, and syphilis gives rise to three well-characterized forms, namely, diffuse syphilitic hepatitis, commonly congenital, gummata which undergo fibroid transformation, and an extensive perihepatitis with increase in connective tissue of the portal canals.

(c) Mechanical irritation. The long-continued exposure to an atmosphere laden with dust particles such as produce pneumonoconiosis, whether these be mineral or metallic, may also give rise to an inflammatory process in the connective tissue of the liver.

(d) Congestive; the chronic hyperæmia of the blood-vessels occurring in heart disease gives rise to an interstitial hepatitis—the cardiac liver.

(e) Obstructive, the result of chronic obstruction in the bile-ducts.

Pathology and Classification.—Vaughan, in a recent study of the subject, concludes that so-called atrophic and hypertrophic cirrhosis are not different forms of the same disease but wholly different diseases. He states that the former is known as atrophic cirrhosis because from its earliest possible recognition the liver is less than normal in size; while the hypertrophic is known as such because at every stage of the disease the liver is larger than normal; that the atrophic is known as venous cirrhosis because of the early and constant involvement of the intrahepatic branches of the portal vein, the hypertrophic as biliary cirrhosis because of the early appearance and constancy of icterus; that in atrophic cirrhosis the primary destructive changes are in the hepatic cells, while in hypertrophic cirrhosis the epithelium of the gall-ducts is the site of the primary involvement; that the former might be known as toxic, the latter as infective cirrhosis.

This clear-cut classification, while in some respects convenient and while it provides categories for well-marked cases of widely different morbid conditions, by no means meets the requirements of all the cases and does not appear to be wholly justified upon etiological, anatomical, or clinical grounds. There are cases of atrophic cirrhosis in the causation of which alcohol plays no part, and cases of hypertrophic cirrhosis in hard drinkers. My clinical experience leads me to believe that alcohol is a very common cause of the latter form of cirrhosis.

Cases of cirrhosis of the liver without jaundice and with the evidences of a high degree of portal obstruction, in which the liver is normal in size or slightly enlarged, are by no means rare. In addition to these there is the fatty cirrhosis common in beer-drinkers. There is a large group of cases in which the symptoms are neither those of the atrophic nor the hypertrophic form. These are designated mixed forms, and are thought to be due to the coexistence of the two forms, the toxic and the infective, in the same individual. On the other hand, Hawkins of St. Thomas's Hospital, in the article on cirrhosis of the liver in Allbutt's System, expresses the opinion that the terms "atrophic" and "hypertrophic" are scarcely worth retaining. "The former," he observes, "has lost much of its fitness now that statistics show that the hob-nailed liver, to which it was originally applied, is not necessarily small, but is often increased both in size and weight," while "the phrase hypertrophic cirrhosis has become so complicated by the postulate of a biliary cirrhosis that its significance is vague and uncertain." Hawkins does not recognize a vascular or toxic form corresponding to the atrophic form of writers—Laennec's cirrhosis—and a biliary or infectious form—Hanot's cirrhosis,— but states that "two forms of cirrhosis of the liver are induced by the excessive use of alcohol." These two forms are separated both in their morbid anatomy and in their clinical features. In the first, which is by far the more common, the newly developed fibrous tissue is "multilobular," that is, it tends to surround large groups of hepatic lobules. This form is commonly associated with ascites but seldom with jaundice. In the second and less common form of *alcoholic cirrhosis* the new tissue is mostly developed around single lobules and the condition is properly described as "unilobular." There is little tendency to ascites, while jaundice is common.

In this connection may be mentioned Flexner's researches in regard to the new tissue in cirrhosis of the liver. He found both the white fibrous tissue and the elastic tissue increased and the chief distinction between the histology of the atrophic and hypertrophic cirrhosis to depend upon the degree of extralobular growth and the freedom with which the lobules are invaded, and that "in hypertrophic cirrhosis there would appear to be less interlobular growth and an earlier and finer intralobular growth."

Symptoms.—Clinically not all forms of interstitial hepatitis can be recognized. The symptoms are often vague and referable to other organs, especially those of the gastro-intestinal tract. In many of the cases the condition during life can only be suspected, the morbid phenomena being those of the primary disorder. Direct physical signs are available for diagnosis only when there are definite changes in the size or contour of the liver. Hence the cases of chronic interstitial hepatitis, whatever their course, must be arranged in three groups according as the liver is found upon physical exploration to be, (a) of about the normal size, (b) atrophic, and (c) hypertrophic.

(a) Since there are no characteristic symptoms in many cases of chronic interstitial hepatitis, and since in many of the cases the liver remains of normal size throughout and is always of normal size until the disease has made some progress, it follows that the diagnosis is impossible in a considerable proportion of the cases and in the earlier stages of all cases. The most that can be done is to assume that when certain etiological factors, as alcoholism, malaria, valvular disease of the heart with failing compensation, are operative, and more or less well-pronounced gastrohèpatic symptoms present, the patient may have an interstitial hepatitis. The diagnosis under such circumstances must be purely an anatomical one, and this is frequently the case in the fatty form of interstitial hepatitis in which symptoms directly referable to the liver are often absent, and not rarely in the multilobular form either with or without atrophy. In the latter instance the true nature of the malady may reveal itself in sudden copious hæmatemesis or in the peculiar toxæmia caused by the entrance of portal blood, which has not traversed the liver, into the general circulation.

When, however, there are decided changes in the size of the liver the state of affairs is wholly different. The interstitial hepatitis reveals itself not only in physical signs, but also in symptoms of significance.

(b) The liver is diminished in size—*Laennec's cirrhosis.* A high degree of atrophy may occur, provided the collateral circulation has been established, without the development of ascites or other symptoms which attract the attention of the patient to the liver. As a general rule, however, the malnutrition, vomiting, the enlargement of the spleen, hemorrhoids, the distended superficial abdominal veins, and the increased girth caused by the ascites are diagnostic. It is of cardinal importance also to remember that all these phenomena can occur in an interstitial hepatitis in which the liver is not only not atrophic but even somewhat enlarged. Under these circumstances jaundice is not usually present.

(c) The liver is increased in size. The hypertrophic form of chronic interstitial hepatitis,—*Hanot's disease,*—like the forms already considered, may present extreme difficulties in diagnosis in its early stages. The liver may be as yet of normal size. The early icterus may resemble that of catarrhal jaundice—a resemblance heightened by its fluctuation or temporary disappearance, and by the occurrence of fever. The recurrence or persistence of the jaundice, its intensity, and the presence of bile in the stools are of diagnostic importance. When the disease is established the uniform enlargement of the liver, the splenic tumor, the deep and persistent jaundice, and the occasional attacks of fever are diagnostic. The fever is of subcontinuous or remittent type and extends over periods of days or weeks. Chills and sweating are not common, as in the fever of impacted gall-stone —Charcot's or true hepatic fever.

Diagnosis.—Direct.—The diagnosis of well-defined cirrhosis of the liver, the terminal condition, is usually a simple matter. Whether it be the atrophic form of Laennec or the hypertrophic form of Hanot, the symptom-complex in most of the cases is characteristic. In the former the pinched face with its distended venules and muddy or subicteroid hue,—*facies hepatica,*—the spare chest and thin arms, the distended belly with its conspicuous superficial veins, and the diminished area of liver dulness leave no doubt of the nature of the malady. In the latter the jaundice, the fairly well-preserved nutrition, the big liver without ascites, and occasional irregular fever render the diagnosis equally clear. The two conditions are clinically distinct because they are the manifestations of essentially different pathological lesions. The one thing that these lesions have in common is an overgrowth of the interstitial connective tissue of the liver. It might be said that they have nothing in common clinically save that they are diseases of the liver. Yet they are described respectively as the atrophic form and the hypertrophic form of the same affection, hepatic cirrhosis.

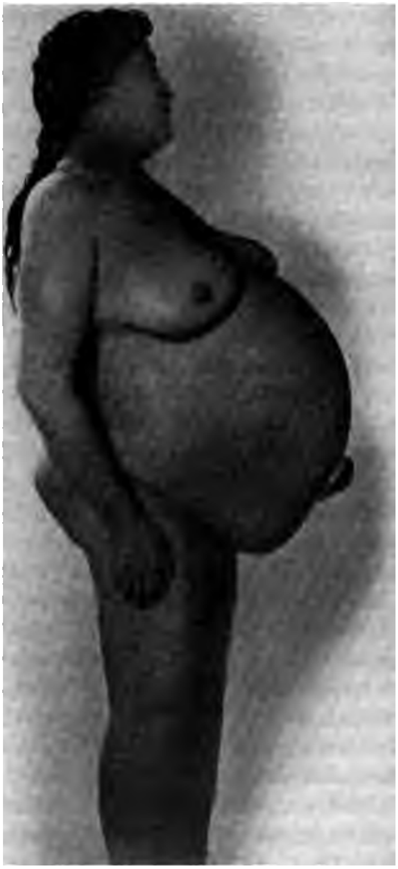


Fig. 314.—Ascites due to atrophic cirrhosis of the liver.—Jefferson Hospital.

The diagnosis of hepatic capsulitis—the *capsular cirrhosis* of authors—is usually attended by insuperable difficulties. The symptoms are those of the atrophic form of chronic interstitial nephritis. Jaundice is not usually present. The kidneys are granular. This condition may be associated with perisplenitis and proliferative peritonitis.

DIFFERENTIAL.—Adhesive pylephlebitis closely resembles the atrophic form of interstitial hepatitis. The etiological factors and the rapidity with which the peritoneal effusion develops and reforms after tapping, as was shown in a case recently in my service in the Pennsylvania Hospital, are important in the differential diagnosis. It is said that thrombosis of the portal vein is followed by an atrophy of the liver, which renders the diagnosis difficult. In the cases that have come under my observation death has occurred in the course of a few weeks, and the liver was of normal size.

Prognosis.—In the atrophic form the outlook is highly unfavorable. From the time when the diagnosis can be made the duration of the disease is often not more than a few months, rarely more than two or three years. Atrophic cirrhosis in advanced stages has been found post mortem in cases in which no characteristic symptoms were present during life. Life has been much prolonged in some of the successful cases of omentopexy.

In the hypertrophic form the disease, after it has reached a stage in which a positive diagnosis can be made, runs an unfavorable course. The progress is often slow and the disease for a long period may not continuously prevent the patient from conducting his business or taking part in the ordinary affairs of life.

xi. New Growths in the Liver.

Neoplasms of the liver are benign and malignant. The benign are fibroma and angioma; the malignant are carcinoma, sarcoma, and malignant adenoma. Carcinoma and sarcoma may be primary or secondary.

1. **Benign New Growths.**—These are of no great clinical importance.

(a) **Fibromata.** — These tumors consist of dense connective tissue and are frequently found post mortem. They are usually small and cause no symptoms during life. In very rare instances they are of larger size, and when so situated as to compress the bile passages and prevent the discharge of bile into the intestine they may cause cholangitis and death from cholæmia.

DIAGNOSIS.—In a suspected case an exploratory laparotomy should be performed in the hope that the tumor may be found and removed.

(b) **Angiomata.**—These vascular tumors are also described under the terms cavernomata and telangiectasis. They are commonly multiple and of small size, causing no symptoms. They may be found at all ages and have been observed in the fœtus. In very rare instances they are solitary and of large size,—an orange,—the pregnant uterus. They then give rise to pressure symptoms.

DIAGNOSIS.—Small angiomata cannot be recognized by clinical methods. In some instances the surface of the liver is nodular. A large tumor giving rise to distressing pressure symptoms may justify a diagnosis by exclusion. Its true nature can only be positively determined by an exploratory incision.

PROGNOSIS.—The outlook is favorable as regards life. Large solitary angiomata have been successfully resected.

2. **Malignant Tumors of the Liver.**—Cancer of the gall-bladder and bile-ducts and its relation to cancer of the liver has already been considered.

Malignant tumors of the liver are of great clinical importance.

(a) **Carcinoma Hepatis.**—Carcinoma invades the liver less frequently than the uterus, the stomach, or the breast. Cancer of the liver may occur at any age and has been observed in the new-born. A number of cases of primary carcinoma of the liver have been observed in children. The part played by heredity in cancer of the liver is uncertain. About 17 per cent. of the cases show hereditary predisposition. Half the cases are first observed after the fortieth year. Primary cancer of the liver is more common in men and is frequently associated with cirrhosis. Secondary cancer is much more frequent in women, a fact attributed to the remarkable tendency to cancer of the uterus, ovaries, and breasts. Primary cancer is common in the gall-bladder, rare in the parenchyma of the organ. The frequent association of gall-stone disease with cancer of the gall-bladder and bile-ducts is attributed to the chronic irritation caused by the presence of calculi.

PRIMARY CARCINOMA presents three principal types: (i) *Massive Carcinoma.*—The liver is greatly enlarged, its surface usually smooth. On section the growth is sharply contrasted with the normal tissue which surrounds it. It is grayish-white and not usually softened. It is of very great size and at first solitary, though later surrounded by smaller metastatic nodules. (ii) *Nodular Carcinoma.*—Round, grayish-white, or yellow nodules of cancerous tissue of varying size are irregularly scattered throughout the liver. The occasional occurrence of one large mass surrounded by numerous smaller and less dense nodules makes it probable that the former is the original seat of the disease. The liver is not usually greatly enlarged. Sclerotic changes are common and the organ may be reduced in size. Transitional forms between massive and nodular cancer occur. (iii) *Adenocarcinoma with Cirrhosis.*—The liver is usually contracted but may be enlarged. The surface is dark green, with irregularly distributed yellowish nodules of varying size beneath the capsule. The adenomata appear throughout the liver substance as small round masses varying in size from a millet-seed to a pea. Sometimes only a single tumor is present.

SECONDARY CARCINOMA.—The liver may be enormously enlarged. Nodules are present upon its surface, which may often be felt and seen through the abdominal walls. They may be dense or soft, and frequently, in consequence of retraction of the connective tissue, are distinctly umbilicated—*Farre's tubercles.* The tumors are irregularly scattered throughout the substance of the organ. They are usually light in color, grayish-white or greenish, and sharply defined, both on the surface and in the interior of the liver, from the surrounding liver substance, which is often hyperæmic. The nodules themselves are often hemorrhagic. They may be present in such numbers as almost completely to replace the liver parenchyma.

Histologically the primary cancers are epitheliomata; the secondary cancers are of the same structure as the respective primary growths—as a rule, alveolar or cylindrical carcinomata.

(b) **Sarcoma.**—Sarcoma of the liver is much less common than carcinoma. Primary sarcoma is very rare. Most of the cases are secondary, though the primary growth may be so small as to be easily overlooked. Melanosarcoma is the must common and most important variety. It appears in rare cases as a primary tumor. As a rule, it is secondary to pigmented sarcoma of the eye or skin. The primary growth is of diagnostic importance. The secondary sarcomata of the liver sometimes form enormous tumors. They occur either as large nodular masses throughout the liver or as diffuse, infiltrating growths. In the latter form the enlargement of the liver is less marked. On section the surface is black or marbled. They constitute part of a widely extended metastatic process in which many viscera are often involved.

Symptoms of Malignant Disease of the Liver.—The condition may be latent, especially when the growth develops centrally or in the diaphragmatic surface of the liver, or the symptoms may be overshadowed by those of the primary tumor. When a primary cancerous growth in any organ, as the breast, stomach, or rectum, is present or has been removed, the secondary nature of a neoplasm in the liver is obvious. When no such growth is discoverable, it is not always possible to determine whether cancer of the liver is primary or secondary. Loss of appetite and nausea are early symptoms. Vomiting is less common. Emaciation and loss of strength are marked and progressive. The skin loses its elasticity and becomes dry, wrinkled, and of a muddy color. Sensations of fulness and weight in the epigastrium and right hypochondrium are followed by pain which extends to the chest and right shoulder. The liver is commonly enlarged, tender upon pressure, and nodular. In some cases of primary cancer, and in those cases in which cirrhosis is marked, the liver may be of normal size or even contracted. Persistent jaundice is present in more than half the cases. It is intense when there is compression of the common duct. Ascites is much less frequent. It occurs as the result of compression of the portal vein, or its invasion by the growth, advancing cirrhosis, or extension of the growth to the peritoneum. The anæmia is progressive. Œdema of the feet or general œdema occurs; there are signs of metastasis to the pleuræ and peritoneum. Fever of continuous or remittent type occurs and death results from asthenia. The blood shows the changes of secondary anæmia which may be of high grade. Poikilocytosis may be present. Profuse diarrhœa may cause concentration of the blood.

Physical Signs.—INSPECTION.—The abdomen is distended, especially in the epigastric zone and upon the right side. In advanced cases the nodular surface and even the umbilication of the nodules may be visible through the emaciated abdominal wall. The superficial veins are usually enlarged. PALPATION.—The enlargement may be recognized and the border felt some distance below the margin of the ribs. When the left lobe is affected, a distinct tumor may be felt in the epigastrium. The surface is commonly irregularly nodular, with an uneven margin and distinct central depressions in many of the nodules. In cases of diffuse infiltration the surface is usually smooth. It is also hard. Enlargement of the superficial lymph-nodes, especially the inguinal, supraclavicular, and cervical nodules, is common. When slight it is not due to metastasis and not of diagnostic importance. Actual metastasis with decided enlargement of the left supraclavicular gland occasionally occurs in malignant disease of the abdominal organs. PERCUSSION.—The upper and lower limits of dulness may be determined and the progress of the growth estimated, the presence and increase of ascites observed, and pleural and peritoneal new growths recognized. The spleen is not usually enlarged.

Diagnosis. — DIRECT. — Enlargement of the liver, which may be smooth or nodular, and in particular when the nodules are umbilicated; cachexia; compression symptoms in the territory of the portal vein or vena cava—ascites, œdema; in the territory of the bile passages—jaundice; signs of metastasis in the pleuræ, lungs, or peritoneum, constitute a characteristic symptom-complex. In the presence of a primary growth or the history of one removed, the direct diagnosis may be made. In the absence of such a primary growth the diagnosis remains uncertain in proportion as one or more of the above groups of clinical phenomena are ill defined or absent. The Röntgen rays may be of great service in the diagnosis of doubtful cases in which localized dense neoplasms exist in regions inaccessible to the ordinary means of clinical examination.

Sarcomata of the liver are mostly secondary growths. In melanosarcoma the primary growths are commonly in the choroid or the skin. The enlargement is rapid and reaches a high grade. Multiple tumors are often present in the skin, and metastases are widely extended. Melanuria is an inconstant but important symptom. The liver tumor may not develop until some months after the removal of an eyeball, as in a case of mine in the Philadelphia Hospital.

DIFFERENTIAL.—*Fatty Liver.*—The uniform enlargement and smooth surface are suggestive of cancerous infiltration, especially in the fatty liver of emaciation and cachexia. But the soft consistency of the enlarged liver, its slow growth, and the absence of jaundice are important. *Amyloid Liver.*—A history of suppuration or syphilis, enlargement of the spleen, urinary phenomena, in particular albumin and casts which take the iodine test, and a less rapid and marked cachexia are diagnostic. Gummatous nodules may greatly embarrass the diagnosis. *Echinococcus of the Liver.*—Ordinary echinococcus cysts are little likely to be confounded with malignant disease. The enlargement with hard nodules upon the surface, characteristic of multilocular echinococcus, may give rise to uncertainty, especially as jaundice and ascites are frequent in both conditions. The slow progress of multilocular echinococcus, the enlargement of the spleen, and the slight tendency to cachexia are important in the differentiation. Exploratory puncture may bring away softened material containing cholesterin and hæmatoidin crystals. *Chronic Interstitial Hepatitis—Hypertrophic Cirrhosis.*—The absence of emaciation and cachexia, of pain and tenderness, and of a primary focus are of diagnostic importance. The enlarged liver is less dense than in infiltrated cancer and the jaundice more variable in intensity. *Atrophic Cirrhosis.*—The muddy complexion or jaundice, wasting, ascites, and other signs of portal obstruction common to the two affections may cause great uncertainty. Differential points are an alcoholic history, tardy course, enlargement of the spleen, and the absence of primary malignant disease in other organs in cirrhosis. That form of hepatic cancer with cirrhosis in which the liver is reduced in size cannot, in the absence of a primary focus or metastasis, be recognized during life.

Prognosis.—The outlook is in the highest degree unfavorable. The duration of the disease rarely exceeds eighteen months. Secondary cancers run a much more rapid course than the forms associated with cirrhosis. Resection of malignant growths in the liver has been performed.

IV. DISEASES OF THE PANCREAS.

i. Hemorrhage into the Pancreas.

The hemorrhage usually occurs as a manifestation of the necrosis incident to acute pancreatitis. There are instances, however, in which large hemorrhage into the organ and adjacent structures has occurred in the absence of inflammation.

The etiology is that of acute hemorrhagic pancreatitis. The condition occurs in middle life.

Symptoms.—The onset is sudden. The patients are usually in their ordinary health. In some instances there have been digestive symptoms or previous attacks of biliary colic. Pain is intense and located in the upper part of the abdomen. It is sharp, sometimes colicky, and increases in severity. It is accompanied by nausea and vomiting, which are frequent and intractable but not followed by relief. The patient becomes restless, anxious, and depressed. The surface is cold and covered with a clammy sweat. The pulse is feeble, rapid, and thready. The appearance of the patient is that common in internal hemorrhage. There is epigastric tenderness followed by tympany which is usually moderate. The temperature is normal or subnormal, the patient falls into syncope which terminates fatally in the course of twenty-four or forty-eight hours.

ii. Acute Pancreatitis.

(a) ACUTE HEMORRHAGIC PANCREATITIS.

Definition.—The term acute hemorrhagic pancreatitis is employed to designate a rapidly developing destructive process,—necrosis,—accompanied by hemorrhage into the substance of the organ and adjacent parts, and in nearly all cases by disseminated areas of fat necrosis. In some instances there is no evidence of inflammation; in others there are inflammatory changes.

Etiology.—PREDISPOSING INFLUENCES.—It is in the highest degree probable that cholelithiasis is the chief predisposing influence to acute hemorrhagic pancreatitis. This hypothesis is supported by the following facts: It has been experimentally shown that similar lesions are produced by the injection of bile into the gland by way of the duct of Wirsung; the condition is of common occurrence in individuals suffering from cholelithiasis; biliary calculi are present in the bile-ducts or in the duodenum in a large proportion of the cases; and an impacted gall-stone has been found, as in a case reported by Halstead and quoted by Opie, at the duodenal opening of the ampulla of Vater not of sufficient size to occlude either the common duct or the pancreatic duct, and thus converting them into a continuous closed channel.

THE EXCITING CAUSE.—The statement of Opie that, "While at present it cannot be denied that other causes may produce the condition, only one etiological factor has been demonstrated, namely, the impaction of a gall-stone in the diverticulum of Vater, diverting bile into the pancreatic duct" is unquestionably true. The immediate cause then is a mechanical one. The bile and the pancreatic secretion are present at low pressure, but the bile is forced into the pancreatic duct by the contractions of the gall-bladder, and in some of the cases the walls of the pancreatic duct have been stained with bile.

In the cases in which no evidence of gall-stone disease is found upon post-mortem examination two conditions may occur: first, a neoplasm interfering with the discharge of the bile into the duodenum; and second, occlusion of a narrow canal between the ampulla of Vater and the duodenum by catarrhal swelling or a plug of tenacious mucus.

Symptoms.—No sharp line of demarcation can be drawn between acute, gangrenous, and suppurative pancreatitis, which are in fact consecutive processes in cases in which death does not take place rapidly. The attack may have been preceded by attacks of biliary colic, or it may supervene upon such an occurrence. Again, since anatomical studies of the relative diameter of the common duct and the canal leading from the diverticulum of Vater into the duodenum have shown that a small calculus readily passing through the common duct may fully occlude the duodenal opening, the onset of the symptoms of acute pancreatitis may constitute the first clinical phenomena of gall-stone disease.

There is little to add to the terse and graphic description of Fitz: "It (the attack) begins with intense pain, especially in the upper abdomen, soon followed by vomiting, which is likely to be more or less obstinate, and not infrequently by slight epigastric swelling and tenderness with obstinate constipation. A normal or subnormal temperature may be present, and symptoms of collapse precede by a few hours death, which is most likely to occur between the second and fourth days." Nausea is marked and continues between the attacks of vomiting. The vomitus is not characteristic. It does not at first contain bile. Collapse symptoms occur early and, considered in connection with the above symptoms and the rapidly fatal result, suggest acute poisoning.

Diagnosis.—DIRECT.—The diagnosis rests upon the sudden occurrence of the foregoing symptoms in an adult who has suffered from chronic gastroduodenal catarrh or from attacks of biliary colic; the location of the pain and tenderness in the upper abdomen; the absence of the distinct, board-like rigidity characteristic of early peritonitis and an early high leucocytosis. Da Costa, in seven counts in four cases at the German Hospital in Philadelphia, found a leucocytosis ranging from 11,000 to 30,000. If the patient survives, circumscribed epigastric fulness, which may be tense and tympanitic or dull upon percussion, may develop.

Opie has suggested that the fat-splitting ferment, which, free in the tissues, causes the fat necrosis, may be excreted by the kidneys, and, using the ethyl butyrate method of Castle and Loevenhardt, which depends upon the power of a fat-splitting ferment to decompose that substance with the liberation of butyric acid, was able in one instance to demonstrate the presence of a marked acid reaction, while a control specimen remained unchanged.[1]

DIFFERENTIAL.—*Acute Poisoning.*—The anamnesis is important. In poisoning by meat or fish a number of persons are usually simultaneously affected. There is a period of prodromes consisting of languor, nausea, and griping pain in the belly. The attack begins suddenly with chilliness, faintness, and headache. Collapse symptoms supervene with vomiting and diarrhœa, which is often uncontrollable. In poisoning by corrosive chemicals the surrounding circumstances, certain marks upon the lips and garments, and the behavior of the patient are important. Collapse is preceded by intense pain in the stomach, followed by colic and in many instances by diarrhœa. *Strangulated Hernia.*—In a doubtful case the sites of hernial tumors are to be carefully examined; the history is important; constipation and fecal vomiting are significant. *Intestinal Obstruction.*—In acute obstruction we find constipation, abdominal pain, and vomiting. The pain is at first colicky, later continuous and severe. Vomiting is an early symptom. Nausea is less marked than retching. The vomitus consists at first of the stomach contents, then of bile-stained mucus, and finally of a darkish liquid with a fecal odor. In many cases neither fæces nor flatus are passed by the bowel; in some the contents of the bowel below the constriction are voided. Abdominal tenderness and tympany come on later. If the obstruction be seated in the small bowel the distention may be slight, but it is not confined to the epigastrium. Pain and tenderness are later symptoms and are not circumscribed. Collapse symptoms are not usually at first present. There is, as a rule, a very high leucocytosis, 60,000 or more. Acute hemorrhagic pancreatitis is very often mistaken for intestinal obstruction. *Embolism of the Larger Mesenteric Vessels—Infarction of the Bowel.*—This accident gives rise to sudden colic, nausea, vomiting, and bloody diarrhœa. The condition resembles acute obstruction, marked tympanites develops, and death occurs in collapse. *Perforative Peritonitis.*—The differentiation becomes apparent when the symptoms are enumerated. In perforation of an ulcer of the stomach, bowels, or gall-bladder, necrosis of the appendix, rupture of an abscess of the liver, spleen, kidney, or Fallopian tube, chilliness or rigor, intense abdominal pain, and exquisite tenderness are early symptoms. The pain and tenderness are general but more intense as a rule in the region of the perforating lesion. There is early spastic contraction of the abdominal muscles upon one or both sides—a very significant sign. The patient assumes and maintains an attitude by which the tension of the abdominal muscles is diminished, and lies with his head and shoulders elevated and his thighs and legs strongly flexed. Later the tension relaxes, the abdomen becomes tympanitic, and both pain and tenderness abate. When there is a history of gall-stone disease the differential diagnosis becomes as important as it is obscure. Absence of muscular tension, circumscribed pain and tenderness in the epigastrium, and early profound collapse are suggestive of pancreatitis.

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[1] Diseases of the Pancreas, Opie, 1903, p. 322.

Cammidge found that in cases of pancreatic disease a peculiar substance, probably pentose, is present in the urine and can be detected by its forming long, yellow, flexible crystals arranged in sheaves in the presence of phenylhydrazine. The test is very elaborate and can only be carried out in a laboratory.

(b) GANGRENOUS PANCREATITIS.

Etiology.—Necrosis of the whole or a portion of the gland may follow hemorrhage or hemorrhagic pancreatitis in the cases in which death does not occur in the first three or four days. The tissue of the pancreas is dry and friable, and the necrotic organ lies nearly free in the omental cavity. Death usually occurs in the course of two or three weeks. There are recorded cases in which the necrotic pancreas has been discharged by way of the rectum, with recovery.

Symptoms.—The clinical symptoms are those of acute hemorrhagic pancreatitis, but the illness is of longer duration. As the gangrenous pancreas occupies a position in the posterior wall of the lesser peritoneal cavity, peritonitis ensues, and this cavity becomes filled with pus and necrotic material. These changes are followed by fever, delirium, and the general symptoms of septic infection.

Diagnosis.—The condition cannot be positively determined during life. A probable diagnosis rests upon the occurrence of the symptoms of acute hemorrhagic pancreatitis with a prolongation of life, and the occurrence of septic phenomena.

(c) ACUTE SUPPURATIVE PANCREATITIS—PANCREATIC ABSCESS.

Suppurative inflammation of the pancreas presents nothing characteristic, with the exception that it most commonly occurs as a later stage of acute hemorrhagic and gangrenous pancreatitis. There may be a single large abscess, multiple small abscesses, or diffuse purulent infiltration. The lesser peritoneal cavity may be distended with pus.

Etiology.—Cholelithiasis constitutes a predisposing influence. The actual cause of the disease is to be found in an antecedent hemorrhagic pancreatitis. In some cases the condition has followed traumatism.

Symptoms.—The clinical manifestations follow those of acute hemorrhagic pancreatitis and are the result of the invasion of the necrotic pancreatic and peripancreatic tissues by pus-producing organisms. There may be epigastric prominence, or a deep-seated mass may be felt in the median line. Irregular chills and fever, with profuse sweating and progressive loss of flesh and strength, occur. In some instances the disease runs a protracted course with irregular fever, epigastric pain, and vomiting. Slight icterus, fatty diarrhœa, and glycosuria occur in some cases. Perforation into the stomach, duodenum, or peritoneum may occur. Portal thrombosis has been noted.

Diagnosis.—The recognition of the condition is extremely difficult. Circumscribed epigastric prominence or a resistant deep-seated mass in connection with the above symptoms is suggestive. The gravity of the condition justifies surgical diagnosis by an exploratory operation.

Prognosis.—The prognosis in the acute cases is uniformly fatal, death occurring in the course of twenty-four or thirty-six hours. In a limited number of subacute cases, in which life has been prolonged, spontaneous recovery has occurred with the discharge of a portion of the necrosed gland by the bowel, or surgical operation has been followed by cure.

iii. Chronic Pancreatitis.

The gland undergoes sclerotic changes as the result of chronic inflammation. It is sometimes diminished in size; in other cases it is larger than normal, and may form a palpable epigastric tumor. Two types of interstitial inflammation have been distinguished—an interlobar and an interacinous.

Etiology.—PREDISPOSING INFLUENCES.—Age is important. Chronic pancreatitis is much more common between the fortieth and sixtieth years of life than at any other period. The disease is frequently secondary to disease of the intestine and bile passages, and of the liver.

EXCITING CAUSE.—Inflammatory irritants may reach the organ by way of the duct of Wirsung or Santorini. Obstruction to the outflow of the pancreatic secretion may be followed by chronic pancreatitis. A form of interstitial pancreatitis of the new-born occurs in syphilis. A history of tuberculosis, syphilis, and the abuse of alcohol may frequently be obtained. The association of cirrhosis of the liver with chronic pancreatitis has been noted.

Symptoms.—The symptoms are obscure, and the condition is rarely recognized during life. At operations for surgical diseases involving the gall-bladder and bile passages the head of the pancreas is not infrequently found enlarged and is so hard as to suggest malignant neoplasm. In such cases it has frequently happened that the patient has recovered and remained well for years. Usually there is a history of epigastric pain, nausea, and persistent vomiting. The signs of arteriosclerosis are frequently present. The enlarged head of the pancreas may press upon the common bile-duct and produce jaundice. Fatty diarrhœa, glycosuria, and diabetes may occur. The islands of Langerhans are the source of a glycolytic ferment necessary to the metabolism of the carbohydrates. Functional or organic disease of these bodies may be followed by an accumulation of glucose in the blood and by glycosuria — *pancreatic diabetes.* Minor functional derangements may give rise to alimentary glycosuria, that is, glycosuria following the ingestion at once, while fasting, of amounts of glucose less than about 200 grammes—the quantity which can be taken in health without causing glycosuria.

Hyaline Degeneration of the Pancreas.—Opie has especially drawn attention to this condition. It affects chiefly the islands of Langerhans and is associated with diabetes mellitus. It may occur as an independent condition, or may be associated with a moderate degree of increase in the interstitial tissue, or arteriosclerosis. It is equally common in the two sexes, and has been chiefly observed after middle life. Chronic interstitial pancreatitis of the interacinous type has been present in the majority of the cases.

iv. Pancreatic Calculi.

Pancreatic lithiasis is a rare condition. It may cause chronic interstitial pancreatitis, dilatation of the duct, a large retention cyst, acute suppurative inflammation, or finally, as in the case of cholelithiasis, carcinoma.

Symptoms. — In pancreatic colic the pain is sudden, intense, and paroxysmal. It has its maximum intensity at the costal margin to the left of the middle line, and passes through to the back. Fatty diarrhœa and glycosuria occur, but are not constant. Vomiting may occur. Pancreatic calculi are in some instances associated with gall-stone disease.

Diagnosis. — DIRECT. — The occurrence of the above symptoms in paroxysms, usually at intervals of months or years, without jaundice, is suggestive. The presence in the stools, after the attack, of round, smooth or rough, opaque, white calculi, which are composed chiefly of calcium carbonate, renders the diagnosis positive.

DIFFERENTIAL.—The symptoms when small stones are passed may suggest gastralgia or gastric ulcer; in other cases cholecystitis or a biliary calculus in the cystic duct. The focus of pain upon the left side and the character of the stones, if passed, are diagnostic.

v. Pancreatic Cysts.

The term pancreatic cyst has been used to designate any cystic tumor in, or associated with, the pancreas, although such tumors differ among themselves, etiologically, in situation and in clinical features.

VARIETIES.—Congenital cystic disease; retention cysts; proliferative cysts; hemorrhagic cysts; hydatid cysts; and pseudocysts.

Etiology.—Sex is without influence, the condition having been observed in about the same number of men and women. Age is important, the largest proportion of cases reported being between twenty and forty years. Cases have been noted in the new-born, and between the sixtieth and seventieth years. The greater number of cysts are caused by traumatism, inflammation, or impacted calculi. They may occupy any part of the gland. Congenital cystic disease may occur in the pancreas as in the kidney and liver. The causes of retention cysts are the impaction of calculi, cicatricial stenosis, pressure upon the duct, and dislocation of a part of the organ. Proliferative cysts are of two kinds: simple, or cyst adenoma, and malignant, or cystic epithelioma. There is a marked tendency to hemorrhage into pancreatic cysts. Collections of blood in the substance of the organ are characteristic of the acute forms of necrosis and inflammation. Hydatid cysts are exceedingly rare. Pseudocysts are circumscribed collections of fluid found in the proximity of the pancreas, but not having their origin in the substance of the gland. They commonly occupy the lesser peritoneum.

The fluid is usually thick and viscid, alkaline, of variable color, clear, milky, yellow, green, or brownish-black. There is usually, in the colored fluids, an admixture of blood. The specific gravity varies from 1.010 to 1.020 or higher. Serum albumin, paraglobulin, mucin, and urea are present. The presence of fat-splitting, proteolytic, or diastasic ferments cannot usually be demonstrated, although the last may sometimes be found. The secretion of the chemically inflamed pancreas may contain only traces of these ferments, and cannot find its way into the cyst unless there is free communication with the glandular parenchyma. Moreover, similar ferments have been demonstrated in the contents of mesenteric and ovarian cysts. The discharge from a fistula resulting from operation may contain the pancreatic ferments.

Symptoms and Signs.—In small cysts symptoms are absent or indefinite. In larger cysts there are pressure symptoms, especially epigastric discomfort, weight, and fulness. Pain, especially after food, confined to the upper part of the abdomen, radiating to the back and toward the left, vomiting, and constipation occur. Jaundice, usually slight, may result from the pressure of a cyst in the head of the pancreas upon the common duct. Light-colored stools containing free fat and much undigested muscle fibre are significant but by no means constant. Alimentary glycosuria and diabetes occur only in cases in which there is extensive destruction of the gland.

The cystic tumor lies behind the posterior layer of peritoneum, which forms the lesser sac. In the great majority of cases the enlarging tumor displaces the stomach upward and to the right, and the transverse colon downward, and approaches the surface below the greater curvature of the stomach; occasionally it presents above the upper border of the stomach; and finally it may push itself between the layers of the transverse mesocolon and force the transverse colon before it, or displace both the transverse colon and the stomach upward.

The tumor occupies the epigastrium, at first usually to the left of the middle line between the costal border and the umbilicus. It may lie in the middle line, or more rarely to the right. It is usually smooth, spherical or oval, elastic, and tense. It may attain enormous dimensions and reach to the symphysis pubis, suggesting an ovarian or parovarian cyst. It is commonly immovable, or but slightly movable, either upon pressure or with the respiration, but in rare cases may be feebly so in consequence of its attachment to the tail or body of the pancreas by a narrow pedicle. When small it may resemble a solid mass, and transmitted aortic pulsation may suggest an aneurism. In large pancreatic cysts fluctuation can almost always be elicited. There is flatness upon percussion.

Diagnosis. — DIRECT. — The presence of a cystic tumor located—or at first located—in the upper abdomen and having the above features is of diagnostic significance. Inflation of the stomach and colon may be important, especially in small cysts. The contents may be removed for examination by aspiration—an unsafe procedure, to be employed only in extreme cases. A history of recent traumatism, as a kick or blow upon the epigastrium, is important.

DIFFERENTIAL.—Various cystic tumors present points of resemblance to pancreatic cysts. Among the more important are the following: *Ovarian Cysts.*—Only enormous cysts of the pancreas can give rise to difficulties. Examination of the pelvic organs, preferably in the Trendelenburg position, the distention of the colon with air, and the history of the case will remove every doubt even in those cases in which the appearance of the abdomen closely resembles that caused by tumor of the ovary. *Cysts of the liver* are usually hydatid. When in the left lobe, they can scarcely be differentiated from pancreatic cyst except by aspiration of the fluid or an exploratory operation. An *enormously distended gall-bladder* may simulate pancreatic cyst. *Cyst of the suprarenal capsule,* especially upon the left side, may be readily mistaken for cyst of the pancreas. The differential diagnosis by physical signs alone is not possible. *Cysts of the Kidney, Hydronephrosis, Pyonephrosis.*—The tumor moves to some degree with respiration; it is distinctly unilateral, and occupies the lumbar region rather than the epigastrium. The relation of the colon to it may be determined by percussion after inflation. Renal symptoms, such as aching pain, frequent micturition, and the previous disappearance of the tumor with the passage of a great quantity of water, or hemorrhage from the genito-urinary tract. point to the kidney. *Cysts of the Mesentery.*—These tumors are characterized by their location near the umbilicus, movability in a lateral or rotary direction, and tympany around the circumference of the mass, and in a band across it. If a pancreatic cyst develops between the layers of the transverse mesocolon the band of resonance will be due to the colon. *Omental Cysts.*—A cyst developing in the omentum directly below the stomach would push the stomach up and the transverse colon down. By the physical signs the diagnosis would not be possible. The anamnesis and the general symptoms might justify a provisional diagnosis. *Cysts of the posterior wall of the stomach, cysts of the spleen, and certain retroperitoneal cysts* are of very rare occurrence and present unusual diagnostic difficulties. In many of the cases the differential diagnosis between these tumors and cysts of the pancreas is impossible.

Prognosis.—The condition, if early diagnosticated and relieved by operation, usually terminates in recovery. To this statement a reservation must be made in regard to hemorrhagic cysts.

vi. Tumors of the Pancreas.

Neoplasms are rare. Sarcoma, adenoma, and lymphoma are extremely infrequent. Carcinoma mostly involves the head of the organ, is often primary, and commonly occurs after middle life. Miliary tubercle is an accompaniment of acute miliary tuberculosis. Syphilis occurs in the form of chronic interstitial inflammation or gummata. The frequency with which the head of the organ is involved, especially in carcinoma, accounts for certain symptoms. The tumor exerts pressure upon the common bile-duct, the pancreatic duct, the duodenum, and the pylorus. The stomach, colon, aorta, vena cava, portal vein, superior mesenteric vein, and the splenic artery and vein may also be compressed. The breaking down of the growth may cause perforation of any of these organs.

Etiology. — CARCINOMA. — Men are more frequently affected than women. The condition has been found with greatest frequency between forty and sixty years.

Symptoms.—In some cases the symptoms are obscure. Generally epigastric pain radiating to the back, often intense and aggravated at night, is a prominent symptom. Jaundice, a gall-bladder tumor, and enlargement of the liver are common. A tumor in the pyloric region, and fixed, may often be made out. There is rapid wasting. Constipation is usual; the stools are large and contain free fat and undigested muscle fibre in unusual amount. Sugar may be present in the urine, and the symptoms of diabetes may be noted. A subnormal temperature also occurs. A dense shadow may be noted upon X-ray examination.

Diagnosis.—DIRECT.—The occurrence of the above symptoms with cachexia renders the diagnosis of carcinoma of the pancreas positive.

DIFFERENTIAL. — In default of a definite symptom-complex the possibility of interstitial pancreatitis, or malignant disease of the common duct, the liver, or the pylorus is to be considered. *Chronic Pancreatitis.*—A long history, recurrent painful attacks, and epigastric tenderness are in favor of benign disease; loss of flesh is less marked than in carcinoma, and vascular disturbances and dropsy are far less common. Anæmia is less pronounced, and cachexia is absent. *Cancer of the Bile-ducts.*—There is almost always a history of gall-stone disease. If the duct of Wirsung be involved the differential diagnosis cannot be made; if not, the destructive signs of pancreatic disease, especially the rapid wasting, will be absent. *Cancer of the Liver.*—Enlargement of the liver with nodules upon its surface and borders, and moderate jaundice, or its absence, are diagnostic. *Cancer of the Pylorus.*—Marked gastric symptoms, retention vomiting, dilatation of the stomach, absence of free hydrochloric acid, and the presence of altered blood in the vomited matters are against pancreatic carcinoma alone, but the conditions are frequently associated.

V. DISEASES OF THE PERITONEUM.

i. Ascites.

Abdominal Dropsy—Hydroperitoneum.

Definition.—An accumulation of serous fluid in the peritoneal cavity.

Etiology.—GENERAL CAUSES.—The accumulation may be part of a general dropsy caused by derangement of the mechanism of the circulation, as in disease of the heart. In some cases of heart disease the dropsical effusion may be limited to the peritoneum. Ascites occurs also in various forms of hydræmia and in advanced renal dropsy. LOCAL CAUSES.—Chronic inflammation of the peritoneum; portal obstruction; abdominal tumors.

Character of the Ascitic Fluid.—The fluid in ascites from stasis is clear, yellowish or greenish-yellow, alkaline in reaction, of a specific gravity of 1.010–1.015, and contains in solution the soluble substances of the blood. Red blood-corpuscles are also present, usually in small numbers.

In *inflammatory ascites* the fluid contains flakes of fibrin, other masses of coagulated material, large numbers of pus-corpuscles when it is purulent, and many blood-corpuscles when it is hemorrhagic. In cancerous ascites molecular débris and cancer-cells may be present. Various bacteria, as streptococci, staphylococci, colon bacilli, gonococci, B. typhosi, pneumococci, and tubercle bacilli may also be present. The presence of tubercle bacilli may be determined by inoscopy. The differential diagnosis between transudation and exudation cannot always be made from the characters of the fluid alone. For this purpose neither the albumin percentage nor the specific gravity avails. Cytodiagnosis is far less useful in the determination of the nature of peritoneal than that of pleural effusions. The effusion in cirrhosis is sometimes darker in color than in other conditions, and that in cancer and tuberculosis is usually hemorrhagic. In the cells of carcinomatous ascites mitosis is more common than in other forms, and the cells in melanotic sarcoma may contain pigment granules, though the fluid is clear.

Chylous and Chyliform Ascites.—The fluid is milky in appearance and resembles chyle. Quincke recognized two essentially different forms, one a transudate, — *ascites chylosus*, which owes its characters to the actual presence of chyle, — the other *ascites adiposus*, — chyliform or pseudochylous, — the appearance of which is due to the admixture of fat derived from the metamorphosis of the disintegrating cells of an inflammatory exudate or the endothelium. Fat is present in both forms in minute dust-like particles. In chylous ascites there are very few cells in a state of fatty degeneration. In chyliform ascites, on the other hand, there are many cells containing fat granules. The presence of sugar does not justify the conclusion that a milky ascites is chylous, since it has been definitely established that sugar may be present in any form of transudate or exudate in the serous sacs. Both of these fluids, subjected to agitation with ether after the addition of potassium hydroxide, clear up to a greater or less extent, and both respond to the osmic acid and other tests for fat. They are bacteria free and do not undergo decomposition for indefinite periods. Upon standing they separate into a thick sedimentary layer, and a fatty, cream-like layer at the top.

Milky, Non-fatty Ascites.—The fluid resembles the fatty forms, but neither microscopically nor clinically reveals the presence of fat. The milk-like appearance has been ascribed to various proteid and mucoid substances, and to lecithin, but the subject is still under investigation.

Hæmoperitoneum may result from traumatism and the rupture of viscera, as the liver, spleen, or mesentery. It occurs also in extra-uterine fetation and the rupture of aneurism. Recurrent hemorrhage into the peritoneum has been observed in the absence of assignable cause. Blood-stained fluid may be present in acute pancreatitis, volvulus, twisting of the pedicle of an ovarian cyst, and other similar conditions.

Symptoms.—These vary according to the amount of fluid. Small amounts occasion no discomfort, and the gradual accumulations of a considerable ascites may not be realized by the patient. It is very common in the ascites associated with cirrhosis of the liver for the patient to first become aware of his condition by his inability to make his waistband meet. Large accumulations of fluid give rise to much distress by pressure and tension of the abdominal walls, sensations of fulness and weight, and interference with the play of the diaphragm, causing dyspnœa and cyanosis. Œdema of the lower extremities and pudenda, occurring subsequently to large ascites, is due to interference with the return of the blood by the pressure of the fluid upon the ascending vena cava, and interference with the action of the heart by upward pressure against the diaphragm. The superficial venous trunks—mammary, epigastric—are frequently widened and tortuous, and reveal, upon stripping, the upward current of the contained blood.

Physical Signs.—(See Methods of Physical Diagnosis, Part II.)

Diagnosis.—Small quantities of fluid gravitate into the pelvis and fail to manifest themselves by the signs elicited upon external examination. If necessary, the patient may be placed in the knee-elbow posture when a small collection of ascitic fluid gravitating into the most dependent region may be recognized upon percussion, or a finger may be lightly pressed into the inguinal ring, the patient being in the erect posture. Gentle tapping will cause a wave which is felt by the finger. Again the finger, introduced into the rectum or vagina, the patient being in such a position that the fluid gravitates into the cul-de-sac of Douglas, may perceive the fluctuation produced by tapping on the lower part of the belly wall. Less than 1500–2000 c.c. cannot be detected by physical examination.

Paracentesis Abdominis.—The patient should sit on a chair or upon the side of a low bed, with his knees separated and a large jar or small tub between his feet. The puncture should be made in the median line midway between the symphysis and navel, under strict antiseptic precautions, with a straight trocar, one-eighth of an inch in diameter; it should be determined by previous percussion that an intestinal coil does not lie beneath the point at which the patient is to be tapped. Pressure should be applied by a many-tailed bandage to the abdominal wall as the fluid escapes. If the canula becomes obstructed by floating intestine, a change in direction or the insertion of a probe will clear it. Fluids may be allowed to escape until the flow ceases. A strip of adhesive plaster may be placed over the opening, some oozing from which is likely to occur.

DIFFERENTIAL DIAGNOSIS.—*Large cysts* may simulate ascites. The error is most common in *cysts of the ovary;* much more rare are pancreatic cysts of such size. There is tympanitic resonance in the flanks, and the circular or oval area of tympany is not present in the umbilical region. Examination per vaginam may yield important information. *Enormous lipomata* have been mistaken for ascites. There is an obscure sense of fluctuation, but the shifting areas of tympany characteristic of ascites do not occur. The condition is exceedingly rare; females are chiefly affected; the tumor develops in middle life, and is of slow growth. An *overdistended bladder* may reach to the umbilicus, or above it, and has been mistaken for ascites. The dribbling of urine,—incontinence of retention,—and the outline of the swelling should put the practitioner upon his guard. The catheter will at once settle the matter. *Encysted inflammatory exudates,* when large, may simulate ascites, especially when pain and fever, or other constitutional symptoms, are no longer present. In most cases the careful employment of the methods of physical examination is adequate for the diagnosis.

ii. Acute General Peritonitis.

Definition.—Acute diffuse inflammation of the peritoneum.

Etiology.—The peritoneum is peculiarly exposed to local and general infection by traumatism, extension from the viscera which it invests, the perforation of hollow viscera, rupture of the capsules of organs or the walls of abscesses, and by way of the lymph channels and the blood. Peritonitis may be primary or secondary.

1. PRIMARY PERITONITIS.—Infection takes place by the blood or by the lymph stream, and not as the result of any lesion of the viscera with which the peritoneum is in relation, or any wound or surgical operation. Acute general peritonitis is sometimes attributed to exposure to cold or damp, and has then been described as rheumatic. The form which occurs as a terminal event in renal disease, gout, and arteriosclerosis is more common. Whether or not these forms of peritonitis—idiopathic peritonitis—are in the strict sense primary remains unsettled.

2. SECONDARY PERITONITIS.—From the point of view of the source of infection, three groups of cases may be recognized. (a) Those in which the infection of the peritoneum takes place from without by way of traumatism or surgical operation. (b) Those in which one of the abdominal organs, or an abscess, ruptures and its contents are discharged into the peritoneal cavity. Intestinal perforation is the most common accident of this kind. (c) The cases in which bacteria find their way through the wall of the intestine in the absence of a large or small solution of continuity, or enter the peritoneal cavity by way of the lymph channels, as in certain forms of puerperal peritonitis, or peritonitis consecutive to infection of the pleura.

BACTERIOLOGY OF ACUTE PERITONITIS. — One or several varieties of bacteria may be found in the exudate—single or mixed infection. Those which are the most important are the Streptococcus pyogenes, the Diplococcus pneumoniæ, and the Bacillus coli communis. These are frequently present alone — monoinfection. In rare instances the Staphylococcus pyogenes aureus has been found in pure culture in the peritoneal exudate. The gonococcus may also cause peritonitis, whether in simple or mixed infection has not yet been positively determined. In simple infection the following organisms have also been found: Micrococcus lanceolatus, B. pyocyaneus, and the B. influenzæ. In so-called primary—idiopathic—peritonitis, and in postoperative peritionitis, simple infection is much more common than mixed infection; in the secondary forms mixed infection is more common, but monoinfection may occur.

Clinical Etiology.—The vast majority of cases which arise in consequence of disease of the abdominal or pelvic organs invested by peritoneum, or in near topical relation to the peritoneal cavity, are demonstrably "secondary." The organs most commonly involved in the primary affection are the intestines and the reproductive organs in the female.

Intestines.—In the order of frequency and importance, lesions of the vermiform appendix stand first. Intestinal ulceration, especially the ulcers of enteric fever and peptic ulcer in the duodenum, come next. Other forms of intestinal ulcer, as tuberculous, dysenteric, diphtheritic, are less apt to cause perforation, and when this accident occurs it is usually into a region of the peritoneum shut off by adhesions and often already the seat of a circumscribed abscess. Certain forms of intestinal ulcer, both acute, as after extensive burns of the surface, or peptic ulcer, and chronic, as those which occur occasionally in scurvy and leukæmia, show no tendency to perforation. Carcinoma causes chronic adhesive peritonitis and circumscribed abscess formation, but scarcely ever acute diffuse inflammation of the peritoneum. *Acute Occlusion of the Bowel.*—Volvulus and strangulation and less frequently intussusception give rise to peritonitis. Chronic stenosis is rarely the cause of this condition. Rupture of the bowel, like perforation, is at once followed by general peritonitis.

The Stomach.—Peptic ulcer and carcinoma may prove the point of departure for acute peritonitis. Sudden perforation, before adhesions have formed, may occur; more commonly, adhesions with adjacent viscera take place as the result of a local peritonitis, with abscess formation or perforation into the colon.

Liver, Gall-bladder and Bile Passages.—Perihepatitis and local adhesive inflammation are common in diseases of these organs, but acute diffuse peritonitis is infrequent. It may result from rupture of an abscess or hydatid cyst, or from strangulation or acute intestinal obstruction caused by pericholecystitic adhesions. An abscess about an infected gall-bladder has, in rare instances, ruptured into the general cavity of the peritoneum.

The Spleen.—Acute diffuse peritonitis arising from disease of this organ is rare. Rupture of the capsule from traumatism, and the rupture of an abscess before adhesions to neighboring viscera have formed, are the two principal events.

The Pancreas.—Acute hemorrhagic pancreatitis gives rise to inflammation of the lesser peritoneum, and when the patient survives for a period general peritonitis may occur by extension. The rupture of a pancreatic abscess into the general peritoneum is an extremely rare event.

Kidneys and Bladder.—Acute diffuse purulent peritonitis occasionally results from the rupture of an abscess of the kidney. Rupture of a hydronephrosis may not be followed by peritonitis, if the urine does not contain pathogenic bacteria. Perforation of the bladder as the result of ulceration, or the more serious forms of diphtheritic ulceration, may result in a localized abscess or in acute peritonitis.

The Genital Organs.—Acute gonococcus peritonitis, having its starting-point from the vas deferens or seminal vesicles, is exceedingly rare. In the female, however, the sexual organs constitute the most frequent starting-point of acute peritonitis. Puerperal peritonitis is the most common form. Gonococcus infection is an extremely common cause of pelvic peritonitis, and occasionally, in young girls, of acute general peritonitis, which may also occur in children suffering from vulvovaginitis by extension to the tubes.

LESIONS OF THE ABDOMINAL PARIETES, MESENTERIC AND RETROPERITONEAL GLANDS, AND INFLAMMATORY DISEASE OF THE OTHER SEROUS CAVITIES.—Peritonitis sometimes arises in consequence of inflammation or suppuration in the abdominal walls, the burrowing of psoas or other abscesses, rectal disease, or caries of the vertebra, ribs, or bones of the pelvis. It may also be secondary to disease of the mesenteric glands, especially in tuberculous disease, and enteric fever—pseudo-abscess. Pleurisy, pulmonary abscess, gangrene of the lung, and purulent pericarditis may be followed by acute general peritonitis in consequence of infection through the diaphragm.

ACUTE INFECTIOUS DISEASES.—Acute peritonitis sometimes occurs in the course of rheumatic fever. It is common in septic conditions and especially in puerperal sepsis. It is extremely rare in the continued and eruptive fevers, and when it occurs in the course of these infections is secondary to some local lesion, as perforation in enteric fever. It sometimes occurs in erysipelas, especially when the abdominal walls are involved, in influenza of the gastro-intestinal form, and in pneumonia.

PERITONITIS IN THE FŒTUS AND NEW-BORN.—A septic peritonitis of the fœtus arises in consequence of infection from the mother by way of the placental circulation. Not alone the peritoneum, but also the other serous cavities, the pleuræ, pericardium, and meninges are affected. In the new-born, infection by way of the umbilical wound, usually through the lymph channels, is a frequent cause of general peritonitis.

The blood-vessels of the peritoneum in recent cases are more or less deeply injected, and the coils of intestine distended and bound together by lymph. The exudate may be fibrinous, serofibrinous, purulent, gangrenous, or hemorrhagic. The fluid exudate varies in amount from a few small collections of clear serum among the adherent loops of bowel to many litres.

Symptoms.—The symptoms of peritonitis in general may be arranged in the following groups: (a) Symptoms immediately due to the peritoneal inflammation,—pain and the phenomena denoting the presence and amount of the exudate. (b) Symptoms caused by derangements of organs and structures implicated in the process, as the stomach, intestine, bladder, abdominal muscles, and diaphragm,—vomiting, constipation, meteorism, frequent and painful micturition, early rigidity, and late paresis of the abdominal walls, hiccough. (c) Constitutional or toxæmic symptoms: fever with its attendant phenomena, circulatory disturbances, anæmia, modifications of the urine, nutritional disorders, and manifestations of sepsis. These symptoms show great variation in their intensity and association in the different forms of peritonitis and in different cases.

(a) **Peritoneal Symptoms.**—Pain and tenderness in the abdomen characterize the onset. The pain is severe and continuous, the tenderness exquisite. In non-perforative cases the pain gradually reaches its maximum; in perforative cases it is almost always extremely severe from the onset. In enteric fever and other stuporous states the occasional absence of pain is due to the mental condition. The pain is continuous, and not only is it increased by pressure, but also by movement. The patient lies motionless in the [illegible] with the legs and thighs flexed. Respiration is shallow, [illegible]. Cough is suppressed, and sneezing is attended with agonizing pain. To this persistent, characteristic pain is superadded colic due to intestinal peristalsis, and recurring in paroxysms. The pain and tenderness may be present uniformly over the whole abdomen. It is very often most intense below the umbilicus. Frequently, but not invariably, these symptoms are most intense in the area corresponding to the starting-point of the inflammation, as the ileocæcal region, the pelvis, or the epigastrium.

A fibrinous exudate sometimes manifests itself by a friction sound, usually best heard in the upper part of the abdomen, over the liver or spleen. A fluid exudate gradually collects in a majority of the cases. Its presence may be first recognized by dulness in the flanks. As it increases, it gives rise to the characteristic physical signs described under the heading "Physical Diagnosis" (see Part II).

(b) **Visceral Symptoms.**—Vomiting is one of the earliest symptoms in acute peritonitis, and greatly increases the pain. It usually continues several days, not ceasing until the fatal termination, an improvement in the patient's condition, or the outpouring of a large exudate. In the last instance, the cessation of vomiting may be an unfavorable sign. The vomitus consists, at first, of the gastric contents; later, of a bile-stained greenish fluid, and in some instances of blackish material with a fecal odor. Vomiting may be absent in large perforation of the stomach. Constipation is the rule; in some cases the bowels move spontaneously every day or two; in puerperal septic peritonitis diarrhœa is common. Hiccough is a common and distressing symptom.

Muscular rigidity, the result of reflex irritation, is an early and extremely valuable sign of acute peritonitis. It may cause retraction, even a scaphoid abdomen, and by restraining peristalsis may diminish the pain. It is especially marked in peritonitis due to perforation. In rapidly fatal cases the abdomen may be flat and rigid throughout the entire course of the attack.

Painful micturition is due to traction exerted upon the inflamed peritoneum by the contractions of the bladder. Retention of urine is common, especially in men. More often there is great vesical irritability, with frequent micturition. The urine is diminished, high colored, and often contains albumin. It is characteristic of acute diffuse peritonitis that the urine contains large quantities of indican. The micro-organisms which cause the disease have been present. As the rigidity passes away, meteorism takes its place. It is due to paresis of the intestine, and may appear early in the disease, especially in the perforative cases. In extreme cases the bowel is completely paralyzed, and no auscultatory signs of peristalsis can be heard, the belly is enormously distended, especially in its upper and middle segments, and the skin is tense, smooth, and glistening. In puerperal peritonitis the distention, owing to the relaxation of the stretched walls, is greater than in other forms. The splenic dulness may be obliterated; the liver dulness is greatly diminished and may wholly disappear in the midclavicular line; the diaphragm is pushed up so that the apex beat of the heart may be felt in the fourth intercostal space. The obliteration of liver dulness in the front of the body may be due to tympany alone, and is not, therefore, a positive sign of pneumoperitoneum or of the perforation of an air-containing viscus. If, when the patient is turned upon his left side, dulness disappears in the axillary line, there is free air in the cavity of the peritoneum.

(2) **General Symptoms.** The attack begins, in a majority of the cases, with chilliness or a rigor. Fever follows, but is not constant and does not conform to type. The temperature may rise suddenly to a considerable height, but does not often exceed 104° F. (40° C.), or it may gradually rise for several days. In either case it becomes irregular or drops to normal as the attack progresses. As death approaches, the temperature may show rapid oscillations. In perforative peritonitis, the temperature very often drops to subnormal ranges, temperature of collapse,—and remains there until death. The pulse is rapid, small, and wiry. Its frequency is 120–160 per minute and bears no constant relation to the temperature.

Leucocytosis of the polynuclear neutrophilic type, 18,000–40,000, is usual, except in the gravest cases, in which there may be leucopenia.

The clinical picture of acute diffuse peritonitis, from the time that the disease is fully established, is very characteristic. The facies indicates great suffering and anxiety and presents the signs of collapse. The nose is pinched and pointed, the eyes are sunken, the temples flattened; there is cyanosis, and the brow is wet with drops of sweat—*facies Hippocratica.* The patient lies motionless, the respirations are shallow and rapid, the pulse is thready, the knees are drawn up, the hands and feet cold and shrunken.

This form of peritonitis usually terminates fatally. The perforative cases often run their course within forty-eight hours; the non-perforative cases in four or five, or sometimes in eight or ten, days. Exceptionally death occurs very suddenly, with signs of cardiac paralysis.

Diagnosis. DIRECT. The diagnosis rests upon the sudden onset of severe abdominal pain, tenderness, fever, vomiting, rigidity of the abdominal muscles, and collapse symptoms. When the attack is fully developed the facies and attitude are very suggestive. Inquiry into the previous health will often reveal the primary cause of the attack. It is to be borne in mind that intestinal perforation and disease of the pelvic organs in females are the most common primary conditions. A history of attacks of pain in the iliac region suggests perforating appendicitis; pain after eating, epigastric tenderness, hæmatemesis, or dark blood in the stools, peptic ulcer; of recent headache, nose-bleeding, prostration, and diarrhœa, the ambulant form of enteric fever. In females recent abortion or confinement, acute suppurative disease of the pelvic viscera, and salpingitis are common antecedent conditions. In enteric fever the signs of perforation may be masked by the patient's mental condition. In many of the cases the previous condition cannot be determined.

DIFFERENTIAL. The following conditions are often mistaken for peritonitis: Acute Enteritis.—[illegible] the pain is colicky, and less continuous; the tenderness is less acute [illegible]; diarrhœa is a more prominent symptom, and early [illegible] tympany are not so conspicuous. In the severe cases there may be a very marked degree of [illegible]. *Intestinal obstruction, volvulus, and* [illegible] may not only simulate peritonitis, but [illegible] often [illegible] it. The symptoms in common being pain, tenderness, vomiting, and tympanites. Muscular rigidity is not so marked, local distention of the bowel, violent peristaltic movements, and the more tardy development of tympany and collapse point to occlusion of the bowel rather than inflammation of the peritoneum. *Rupture of a tubal pregnancy or an abdominal aneurism* may give rise to symptoms suggestive of perforative peritonitis. The history is very important. Restlessness and air hunger are much more marked in large internal hemorrhage than in inflammation. Embolism of the superior mesenteric artery may be attended with sudden agonizing pain, frequent vomiting, collapse, and tympany. *Acute Hemorrhagic Pancreatitis.*—A history of gall-stone disease and the localization of pain in the epigastrium are important in the diagnosis. In perforative and rupture cases, in which the peritoneum is suddenly flooded with the contents of the intestine or pus, death frequently takes place from shock in the course of a few hours, before an actual inflammation has time to develop. *Hysteria* may mimic peritonitis.

iii. Acute Circumscribed Peritonitis.

This form of inflammation of the peritoneum is, (1) adhesive, or (2) purulent.

1. ADHESIVE INFLAMMATION is of very frequent occurrence in local disease of the abdominal organs. It is usually narrowly circumscribed, and unattended by other immediate symptoms than pain and tenderness, the manifestations of the primary affection dominating the clinical picture. The anatomical changes consist in vascular injection, fibrin formation, and slight serous exudation. The organs involved are chiefly the liver, gall-bladder and bile passages, spleen, stomach, coils of intestines, the appendix vermiformis, and the sexual organs in the female. Perihepatitis, perisplenitis, either circumscribed or involving the whole organ, belong to this category. Intestinal adhesions following local disease or operation, adhesive inflammation affecting the gall-bladder, bile-ducts, duodenum, and the pyloric end of the stomach, or a similar process involving the Fallopian tubes or ovaries may be the cause of distressing subsequent symptoms. Tuberculous, cancerous, suppurative, or hydatid disease may be the cause of localized peritonitis.

2. PURULENT.—Infection with pus-producing micro-organisms may be primary and cause acute suppurative circumscribed peritonitis, or it may occur later and lead to the formation of localized abscess and small pockets of pus among the adhesions and other lesions resulting from the non-suppurative form, so that a transitional condition may be recognized. There are certain points in which acute suppurative circumscribed peritonitis preferably arises; among these, the region of the appendix, the pelvic organs in the female, and the lesser peritoneum are most important.

(a) *Appendicular Abscess.*—The most common cause of acute circumscribed suppurative peritonitis is appendicitis—a condition fully described under the heading "Diseases of the Intestines" (q. v.).

(b) *Pelvic Peritonitis.* — Suppurative inflammations, septic, tuber[illegible]us, or gonorrhœal, are very common. They result in the formation [illegible]rimetric and parametric abscesses. Salpingitis and abscesses of the broad ligament occur. Suppuration is frequently preceded by extensive adhesive inflammation. General peritonitis may arise by extension of the infection, or by rupture.

(c) *Subphrenic Peritonitis.* — Inflammation may involve the lesser peritoneum alone, and inflammatory exudates may be confined to its cavity. Perforating ulcers of the stomach, duodenum, or colon are sometimes so situated that they communicate directly with it, and into it pancreatic hemorrhages and abscesses may be discharged. Effusions into this space may cause an oval, smooth, tense tumor, extending into the epigastric, umbilical, and left hypochondriac regions, and simulating a pancreatic cyst. The physical signs vary greatly from time to time, according to the condition of the adjacent stomach. If the latter is distended with food, the line of demarcation between it and the tumor cannot be made out either by percussion or palpation, while if it is filled with gas, it may yield tympanitic resonance over the greater part or the whole of the tumor, causing it at times to altogether disappear. A subphrenic abscess has, in rare cases, followed pneumonia or empyema; more frequently it results from an appendicular abscess, a renal or hepatic abscess, or trauma. It may occur in connection with cancer of the stomach.

The diagnosis of simple subphrenic abscess is difficult, because the signs and symptoms are very frequently indefinite. The subjective symptoms attract attention to the upper part of the abdomen. Among these pain is most important, and may be referred to the right or left side, the back, and so forth, according to the seat of the abscess. It may be localized, or radiate into the abdomen or lower thoracic belt. When the pain is local it is usually associated with tenderness. Circumscribed phenomena in the epigastrium or left hypochondrium are suggestive. Fluctuation is rare and present only when the abscess is superficial. In rare cases there is circumscribed œdema of the overlying skin.

Pyopneumothorax Subphrenicus—*Leyden.*—When the subphrenic abscess is due to a perforating peptic ulcer air is also almost always present and the condition simulates pneumothorax.

Symptoms.—The nature of the condition is obscure and in a majority of the cases not recognized *intra vitam.* The symptoms vary according to the cause. The pus collection between the liver and the diaphragm, whether in relation with the right or the left lobe when air is not also present, closely simulates an encysted empyema at the base of the pleural sac. When it occurs in association with pneumonia, or empyema, or an abscess in a neighboring organ, the symptoms of the primary affection are more or less rapidly, often suddenly, reinforced by those of the new affection, namely, severe epigastric pain, urgent and persistent vomiting, and respiratory embarrassment. In rapidly developing cases shock may also occur. Later symptoms are chills, irregular fever of septic type, anæmia, and rapid wasting. Burrowing may occur into the pleura or, in the case of pleural adhesions, into the lung, with paroxysmal cough and copious purulent expectoration. When caused by a perforating ulcer of the stomach or duodenum, the onset is abrupt, with great pain, and the vomited material is bilious or bloody. When the abscess cavity contains gas, the diaphragm may be forced upward upon the right side as far as the third rib, and the liver displaced downward; when upon the left side, the heart is displaced upward.

Diagnosis. — Subphrenic pyopneumothorax is very frequently overlooked. The physical signs are those of pneumothorax or pyopneumothorax upon the right or left side, according as the abscess cavity is situated upon the right or the left side of the suspensory ligament. The antecedent symptoms, in the majority of the cases, point to disease of the abdominal organs and not to disease of the lungs or pleuræ. Upon forced inspiration the lower border of the compressed lung is depressed in subphrenic abscess; the liver is usually depressed to a remarkable degree, and its lower border is distinctly palpable. The heart is displaced upward rather than laterally. In both conditions the intercostal spaces may be either obliterated or bulging. As the greater number of cases are the result of perforating peptic ulcer, the local symptoms appear very suddenly, while the general symptoms are usually more severe than in ordinary cases of pneumothorax. Exploratory puncture may be made for diagnostic purposes. The presence of material from the gastro-intestinal tract at once determines the differential diagnosis. The position of the diaphragm may be positively determined by skiagraphy. In pyopneumothorax it forms the floor, in pyothorax subphrenicus the roof, of the abscess cavity.

iv. Chronic Peritonitis.

(a) **Local Adhesive Peritonitis.**—The inflammation of the peritoneum which follows operations or accompanies local disease of the abdominal viscera is more frequently chronic than acute. When it involves coils of intestines, it gives rise to partial stenosis with constipation and colicky pains, and may ultimately be the cause of acute obstruction of the intestine by strangulation.

(b) **Diffuse Adhesive Peritonitis.**—In tuberculosis and general carcinomatous infiltration of the peritoneum the adhesions are sometimes so extensive as to entirely obliterate the cavity. This form of peritonitis is rare in other conditions, but has been encountered in tumors of the peritoneum, and after trauma. It occurs also in syphilis during intra-uterine life.

Symptoms.—The condition may not be attended with definite symptoms. Pain and tenderness are usually present.

(c) **Chronic Proliferative Peritonitis.**—There is great thickening of the membrane, without extensive adhesions. Moderate serous effusion may be present. The mesentery is shortened, and the omentum may be rolled into a firm transverse tumor. In some instances there is a general chronic inflammation of the serous membranes,—Concato's disease, polyorrhomenitis,—involving with the peritoneum both pleuræ and the pericardium. This form of peritonitis occurs in the subjects of chronic alcoholism, in chronic passive congestion, and in tumors, but is especially associated with cirrhosis of the liver.

Symptoms. — The disease may be latent, the symptoms being subordinated to those of the primary condition. They comprise abdominal uneasiness and distention, colicky pains, constipation, and diarrhœa. Jaundice is sometimes present. Ascites may occur, or the shortening of the mesentery, the consequent drawing together of the intestines into a tumor-like mass, and the retracted and indurated omentum may simulate tumors of various abdominal organs.

Diagnosis. — The DIRECT DIAGNOSIS rests upon the concurrence of alcoholism, cirrhosis of the liver, chronic intestinal disease, chronic nephritis, with symptoms of peritoneal disease, and ill-defined tumor-like masses in the abdomen. It is confirmed if the evidences of bilateral chronic pleurisy and indurative mediastinitis are present.

THE DIFFERENTIAL DIAGNOSIS.—This relates to the recognition of the condition, notwithstanding the resemblance of some of its features to tumors of the stomach, liver, or other abdominal organs. To this the vagueness and irregularity of the symptoms and signs, their variations as time goes on, the primary affection, and the evidence of chronic disease in the other serous sacs all contribute. The anomalous nature of the pseudotumors and their independence of the organs may, when the effusion is not too abundant, be recognized upon careful palpation.

v. Tuberculous Peritonitis.

Tuberculosis of the Peritoneum.

The diagnosis of this condition has been fully considered under the appropriate subcaption of Tuberculosis, in the section on The Infectious Diseases. It has, in recent times, acquired peculiar importance in consequence of the remarkable success attending laparotomy in certain forms of the disease.

vi. New Growths in the Peritoneum.

Neoplasms of the peritoneum are rare. They comprise benign and malignant tumors.

1. **Benign Tumors.** — Cysts of various kinds, lipomata, fibromata, myxomata, angiomata, and other rarer forms are occasionally encountered. They may occupy any region, but are more often found in the omentum and mesentery than elsewhere. They are single or multiple. (a) CYSTS.—Cystic tumors are found in the omentum, more frequently in the mesentery. Cysts of the mesentery may be classified according to their contents into serous, chylous, hemorrhagic, dermoid, and hydatid cysts. Serous cysts are very rare. They may be single or multiple. Chylous cysts contain a milk-like opaque fluid having the characteristics of chyle, and are probably due to the retention of chyle in the lacteals, or receptaculum chyli. They have been regarded as embryonic. They are usually found in the mesentery. Hemorrhagic cysts are commonly the result of trauma, and contain a brownish-red fluid. They may be chylous or of peripancreatic origin. Dermoid cysts containing hair, bone, teeth, and mucilaginous material have been found in the omentum and mesentery. They may be multiple. Hydatid cysts usually occupy the omentum or mesentery. When primary, the cyst is commonly single. Secondary hydatid disease of the peritoneum is much more common. The cysts are usually multiple, and may be present in enormous numbers. Mesenteric and omental cysts

vary greatly in size. They may reach a capacity of several litres. (b) LIPOMATA are met with in the subperitoneal tissues of the anterior abdominal wall. They are usually small, but may attain such size as to simulate ascites. They may also develop in the omentum or in the mesentery, and grow to such a size that they completely fill the abdominal cavity. They may be of retroperitoneal origin. They occur more commonly in women, and after middle life. They are of slow growth and, yielding an obscure sense of fluctuation, suggest ascites. (c) FIBROUS TUMORS of the peritoneum are rare. They may arise from the omentum, mesentery, or the pelvic organs, and reach the size of the closed fist. Other benign tumors are exceedingly rare.

Symptoms.—Recurrent vomiting, constipation, and pain may precede the discovery of the tumor, which may occupy various positions and may be single or multiple. Mesenteric tumors are, as a rule, freely movable and may thus be distinguished from pancreatic cysts, retroperitoneal tumors, and tumors of the uterus and its appendages. An ovarian cyst with a long pedicle may, however, be very movable. Malignant tumors early contract adhesions and are usually fixed. The differential diagnosis between mesenteric and omental tumors is often attended with insurmountable difficulties. It can as a rule only be made upon abdominal section.

2. **Malignant New Growths.**—These are primary and secondary. They are of more common occurrence than the benign forms. (a) Most PRIMARY MALIGNANT GROWTHS of the peritoneum are endotheliomata. Sarcomata may occur in rare instances as primary growths starting in the mesentery and omentum. They may reach an enormous size. (b) Much more common are SECONDARY CARCINOMATA. The peritoneum is involved by metastasis from distant organs, or by direct extension from organs which it invests. The primary growth may involve the mamma, pancreas, stomach, intestines, especially the colon, and the rectum, or the uterus. In many of the cases of diffuse carcinomatous growths in the peritoneum there are the signs of an associated inflammation—*carcinomatous peritonitis.* In this form of peritonitis the exudate is usually encysted. It may consist of a yellowish serum, or a blood-stained fluid; it may be chylous, or chyliform. It is very rarely purulent. Peritoneal carcinoma is more common in middle and advanced life than earlier. It occurs with somewhat greater frequency in women than in men.

Symptoms.—Pain may be absent altogether. When present it is less severe than in other forms of peritonitis. Vomiting, constipation with attacks of diarrhœa, hiccough, and tympanites are common symptoms. Fluid exudate may be absent, scanty, abundant, freely movable, or encysted. After the withdrawal of the fluid, irregular and ill-defined tumor masses may be recognized upon palpation, especially the rolled omentum lying transversely or obliquely across the upper part of the abdomen, as a firm sausage-like growth, as in tuberculous and proliferative peritonitis. The fluid may be hemorrhagic, and contain large multinuclear cells, or groups of cells, and the number of cells showing mitosis is greater than in simple or tuberculous effusions (Dock). The temperature is usually normal or subnormal. Fever is, however, sometimes present. The cachexia may be marked, and emaciation is progressive.

Diagnosis.—DIRECT.—With the evidences of the primary disease, or the history of the removal of a carcinomatous breast or uterus, the diagnosis may be made without difficulty. The age of the patient, the presence of nodular masses about the navel, and enlarged inguinal glands are important. If no primary focus can be found the diagnosis may be obscure.

DIFFERENTIAL.—The clinical resemblance to tuberculous peritonitis, as regards the symptoms, the tumor masses, and the physical signs, may be very close. As a rule, the multiple nodules of cancer are larger than those of tuberculosis. Cancer is an affection of the later periods of life, tuberculosis of the peritoneum of its earlier periods. But to this rule there are many exceptions. Inflammation and sinus formation, with discharge of pus from the navel, sometimes occurs in tuberculosis. In the absence of tuberculous disease elsewhere the diagnosis becomes difficult, since the clinical phenomena of tuberculous peritonitis not only closely resemble those of carcinomatous peritonitis, but both have features in common with the chronic proliferative form and diffuse hydatids of the peritoneum. In the last, the hydatid fremitus, and hooklets in the aspirated fluid, are of positive diagnostic value.

vii. Retroperitoneal Sarcoma.

Retroperitoneal sarcoma (Lobstein's cancer) is a rare affection. Steel finds that it occurs most frequently in the first, fourth, and sixth decades of life. Males are somewhat more commonly affected than females. The tumor may spring from the lumbar region, on the right side somewhat more frequently than the left, from the posterior wall of the abdomen near the attachment of the mesentery, or, less frequently, from the pelvis. The growths may arise from the retroperitoneal lymph-glands, the connective tissue around the vessels, or from the remains of the Wolffian body. They are often lobulated and are very prone to degeneration, with hemorrhage and the formation of pseudocysts.

Symptoms.—Vague digestive derangements and dragging abdominal pain are followed by pressure symptoms, such as neuralgic pains in the lumbar region, abdomen, legs, and genitalia, and then œdema of the lower extremities. There may be partial occlusion of the intestine. In a case of sarcoma of the retroperitoneal lymphatic glands, recently under my observation, none of these symptoms was present. There are the signs of a deep-seated tumor, situated centrally or to the right or left of the median line, sometimes moving slightly with respiration, more commonly fixed, usually solid but sometimes cystic—pseudocysts. When the tumor is situated laterally, it is obliquely crossed by the colon, which it pushes forward as it increases in size. The health is rapidly impaired, and cachexia develops.

Diagnosis.—The diagnosis rests upon the presence of the above phenomena in association with a rapidly growing central or lateral tumor about the level of the umbilicus. The differential diagnosis between retroperitoneal sarcoma and tumors arising from the kidneys and suprarenal capsules cannot always be made.

X.

THE DIAGNOSIS OF DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE.

i. Acute Nasal Catarrh.

Coryza; Acute Rhinitis; Cold, or Cold in the Head.

Definition.—Acute catarrhal inflammation of the mucous membrane of the nasal cavities.

Etiology.—Acute nasal catarrh is very common. It is in most instances an independent affection, but it occurs also in the acute infectious diseases. It often follows exposure to cold or damp, especially when such exposure is partial, as in wetting the feet or sitting upon damp ground. It frequently prevails extensively in cold, damp, and changeable weather. Such local epidemics are to be distinguished from true influenza or grippe, to which they bear a superficial resemblance. House epidemics of coryza occasionally arise under circumstances that point to the contagiousness of the affection. Children are especially prone to it. It occurs in infants in consequence of gastric or intestinal irritation, indigestion, or the presence of intestinal worms, and is occasionally the result of injuries inflicted by foreign bodies—buttons, grains of corn, pebbles, peas, cherry-pits, and similar objects—introduced into the anterior nasal chambers. It results from the action of mechanical or chemical irritants upon the nasal mucous membrane. Among these are dust, smoke, ipecacuanha, and the fumes of ammonia, bromine, and iodine. Annoying coryza often follows the internal administration of iodine.

Coryza as a manifestation of acute constitutional infection is an early and prominent symptom of measles, influenza, and pertussis. It is sometimes associated with the ophthalmia of the new-born as the result of gonorrhœal infection incurred during parturition, and occurs as an early manifestation of congenital syphilis.

Symptoms.—The attack begins suddenly, with chilliness or shivering, a decided feeling of malaise, headache, and repeated sneezing. There is feverishness, with slight quickening of the pulse, a dry skin, and muscular pains. The nose at first feels dry and stuffy and mouth-breathing is necessary. The sense of smell is lost, that of taste greatly impaired; the voice acquires a peculiar nasal twang, and nursing infants, being unable to breathe through the nose, are suckled with difficulty. The catarrhal inflammation tends to involve the contiguous mucous tracts. In the course of a few hours from the beginning of the attack there may be a flow of thin, clear, irritating mucus, which excoriates the edges of the nostrils and the upper lip and renders the use of the handkerchief painful. Herpes labialis is common. About the second or third day the secretion becomes mucopurulent, opaque, thick, tenacious, and abundant, and tends to accumulate in the nasal cavities. The swelling of the mucous membrane subsides, nose-breathing is re-established, and recovery takes place within a week or ten days. Repeated attacks of the acute affection tend to produce the chronic form of the disease. Most of the cases are subacute, with symptoms of moderate intensity, little or no constitutional disturbance, and run their course in two or three days.

Diagnosis.—There is no difficulty in the diagnosis of simple acute nasal catarrh. Healthy new-born infants are not likely to suffer from snuffles. This affection, when associated with ophthalmia, is due to the same specific infection. When due to syphilis it is associated with characteristic lesions. Acute nasal catarrh in children, due to the lodgement of foreign bodies, is prolonged, and the discharge, after a time, is frequently admixed with blood. Furthermore, it is almost always one-sided. In such cases a careful examination of the nasal chambers must be made. The progress of a case of measles or influenza will speedily dissipate any uncertainty as to the nature of the acute catarrh with which each of these diseases begins. The coryza of iodism ceases upon the withdrawal of the drug.

ii. Chronic Nasal Catarrh.

Definition.—Chronic catarrhal inflammation of the mucous membrane of the nasal cavities. The cases may be arranged in three groups—rhinitis simplex, rhinitis hypertrophica, and rhinitis atrophica.

Etiology.—Repeated attacks of acute nasal catarrh may end in the chronic form of the disease. Habitual exposure to cold and draughts, a changeable and humid atmosphere, and the constant inhalation of dust are among the causes of chronic rhinitis. Insufficient food, inadequate clothing, improper ventilation, want of sunlight and fresh air, and other unhygienic conditions are predisposing influences. Chronic nasal catarrh is frequently a manifestation of local syphilitic or tuberculous processes. The nasal catarrh of early life tends to assume the atrophic form—a fact which emphasizes the importance of the prompt and efficient treatment of every case of rhinitis. Congenital asymmetry of the nasal fossæ, with marked deflection of the septum, hypertrophy of the adenoid tissue in the vault of the pharynx, traumatism, foreign bodies, and nasal polypi are local causes of chronic nasal catarrh.

SIMPLE CHRONIC NASAL CATARRH (*Rhinitis Simplex.*)—This term is used to designate the transitional condition between prolonged or neglected acute catarrh and that in which hypertrophic or atrophic lesions are present. The mucous membrane is irritable and there is a constant sensation of discomfort in the nose. Catarrhal symptoms follow trifling exposure. The erectile tissue is relaxed and is readily distended with blood, so that one or both nostrils are frequently occluded. The secretion is increased; it is variable in consistency, being sometimes thin and watery, sometimes thick and tenacious. Upon inspection the mucous lining of the nasal chambers is seen to be red, watery, and irregularly swollen.

CHRONIC HYPERTROPHIC NASAL CATARRH (*Rhinitis Hypertrophica*).—Obstructed nasal respiration, constant, often abundant, discharge of mucus or mucopus, frequent sneezing, nasal cough, hawking, and expectoration of tenacious mucus, dryness of the throat, habitual mouth-breathing, especially at night, and disturbed sleep are symptoms. The voice has a peculiar nasal quality, and the hearing is very frequently impaired. In infants the inability to take nourishment without frequent interruption for respiration leads to malnutrition, and the nasal obstruction may cause attacks of suffocative spasm. In older children habitual mouth-breathing begets a peculiar, dull, facial expression, mental hebetude, and retardation of the development of the thorax, with characteristic deformities. The mucous membrane of the nasal chambers is congested throughout, and its epithelial and subepithelial tissues are hypertrophied. The characteristic lesion consists in permanent enlargement of the turbinate bodies. There is marked increase in the connective tissue with cell infiltration, dilatation of the sinuses of the erectile tissue, and loss of contractility in their walls. In a large proportion of the cases hypertrophy of the adenoid tissue in the nasopharynx and catarrhal or follicular pharyngitis occur—nasopharyngeal catarrh.

CHRONIC ATROPHIC OR DRY NASAL CATARRH (*Rhinitis Atrophica, Rhinitis Fœtidus Atrophicus, Ozæna*).—A chronic affection of the nose, constituting the terminal stage of neglected cases of rhinitis simplex and rhinitis hypertrophica. It is characterized by atrophy of the mucous membrane, with shrinkage of the turbinated bodies and diminution of the nasal secretion, which becomes mucopurulent or purulent and undergoes inspissation, with the formation of adherent and frequently offensive crusts. Upon inspection grayish crusts are seen, the removal of which exposes a smooth, pale, or a slightly excoriated, mucous surface. Actual ulceration is rare. The turbinate bodies are greatly reduced in size, their sinuses obliterated, their connective tissue contracted. The entire lining membrane of the nostrils is atrophied. The mucous membrane of the pharynx is often dry and glazed. The sense of smell is lost. Ozæna is present in a large proportion of the cases, but not in all. Odors having the same intensity and foulness are occasionally encountered in other affections of the nose attended with ulceration, as syphilis, the traumatism produced by foreign bodies, and caries and necrosis due to other causes. Atrophic rhinitis is more common in females than in males. In confirmed cases the outlook as regards cure is hopeless; as regards relief from the formation and retention of crusts and from the odor, much may be accomplished so long as a judicious treatment is persistently followed out.

iii. Autumnal Catarrh.

Hay or Rose Cold; Hay Asthma; Hay Fever; Summer Catarrh; Catarrhus Æstivus; Periodic Coryza.

Definition.—An affection of the upper air-passages, characterized by irritability of the mucous membrane, with catarrhal and asthmatic manifestations, by the abruptness of the onset of the attack, which recurs annually at or near a fixed date in the spring, summer, or early autumn, and by its immediate cessation upon the patient's reaching certain localities or upon the occurrence of frost.

Etiology.—The exciting causes are certain irritants in the atmosphere which act upon a supersensitive nasal mucous membrane in individuals of neurotic temperament. Among the most important of these is the pollen of various plants. This is not, as was for a long time supposed, the sole cause of the attack. Inorganic dust of various kinds, the odors of certain flowers and other substances, emanations from animals, as the horse, and from feathers are capable of intensifying the symptoms during the attack and also of inducing similar symptoms at other seasons of the year. The intense glare of the summer sun, excessive heat, overexertion, and indigestion commonly aggravate the attack. That these agencies are, as has been affirmed, exciting causes of the disease is questionable. Hay fever and bronchial asthma are not only closely associated clinically, but they also resemble each other in respect of the causes by which the attack may be excited. Local nasal trouble plays an important part in the etiology of the disease. The resemblance to asthma in this respect is striking. The causal relation between nasal disease and bronchial asthma is fully recognized. A similar relation between disease of the nasal mucous membrane and hay fever has also been demonstrated. The lesions are those of hypertrophic rhinitis. There is in many cases deflection of the septum. Superadded to these is the presence of areas of hyperæsthesia in the nasal mucosa—hyperæsthesia often so exquisite that the touch of a probe will instantly excite the characteristic train of symptoms. This local sensitiveness is an almost constant factor in the etiology of the disease. Its presence in persons who do not suffer from autumnal catarrh proves, however, that something more is required; that is, the neurotic constitution. Males suffer more frequently than females. The disease may develop at any period in life. More than 33 per cent. of the cases begin before the age of twenty years. Dwellers in cities are especially liable to the disease, but those who live in the country do not enjoy exemption. The affection may show itself wherever the peculiar irritants which excite it and persons of neurotic constitution, with hyperæsthetic nasal mucous membranes, are found together. There are certain localities in which the disease does not prevail. These regions are usually circumscribed and possess in common the attribute of an uncultivated soil. They are mostly mountainous, as certain districts in the White Mountains, the Adirondacks, and the Catskills. But elevation is not the essential factor. Relief may be experienced in any wilderness, at certain sea-shore places, on islands, or at sea.

The prominence of the psychical element in many cases is remarkable. In one case J. N. Mackenzie induced the attack by means of an artificial rose. In a large proportion of the cases recurrence of the attack takes place year after year on a certain day of the month—a fact for which no rational explanation beyond the hypothesis of expectant attention has been found.

Symptoms.—The attack makes its annual return at or about the same date. There is sometimes a period of prodromes which consist of lassitude and nervous irritability. The onset is abrupt. Itching of the palate and throat is a most annoying symptom, both common and characteristic. I have seen cases in which, year after year, this persistent itching constituted the chief local symptom. Frequent uncontrollable sneezing; nasal obstruction; free rhinorrhœa, usually thin and watery, sometimes mucopurulent; great irritation of the eyes with itching of the lids and lachrymation; loss of the sense of smell; impairment of that of taste, and not rarely disturbances of hearing, constitute the usual symptoms. These occur in paroxysms and are aggravated by changes of temperature, by sunlight, and the open air. Constitutional disturbances consist of subjective sensations of heat and cold, great lassitude, complete loss of appetite, and sleeplessness. After a time the catarrh extends to the bronchi and the patient is annoyed by cough; asthmatic symptoms are common and add greatly to the distress of the patient. The symptoms vary in localization and in intensity, and in the same person in succeeding years. The whole duration of the attack, if not cut short by change of climate, is about six weeks. The autumnal cases usually cease abruptly upon the appearance of frost.

Prognosis.—The prognosis is favorable as regards recovery from any given attack and as regards length of life. The prognosis as regards the recurrence of the attack is much less hopeful.

iv. Epistaxis.

Nose-bleed.

Definition.—Bleeding from the nasal passages.

Etiology.—Bleeding from the nose may be due to local or constitutional causes or to a combination of both. In children its occurrence is favored by the great vascularity of the nasal mucous membrane, the frequent presence of "hemorrhagic spots," and erosions of the septum caused by picking the nose. Other local causes are chronic rhinitis, intranasal ulceration, new growths, the presence of foreign bodies, and various kinds of traumatism, especially contusions of the face.

In fractures involving the bones of the face and cranium blood may escape from the accessory sinuses or from the middle ear by way of the nose, or in hemorrhage from the lungs, œsophagus, or stomach, some part of the blood may be discharged from the nose. These blood-losses, *not from*, but merely *by way of*, the nose, do not in a strict sense constitute epistaxis—a term restricted by systematic writers to hemorrhage having its source within the nasal passages.

Among the constitutional causes are exposure to extreme cold or undue heat, or to a rarefied atmosphere, as in the ascent of high mountains and in balloon ascensions. It frequently occurs in both sexes at the age of puberty. It may result from the suppression of the menstrual flow or follow the sudden arrest of a habitual hemorrhoidal discharge. It is of frequent occurrence in anæmia in its various forms, and in persons of plethoric habit. The tendency to nose-bleed is hereditary. In hæmophilia nose-bleed constitutes a common manifestation of the hemorrhagic diathesis. It is also common in scurvy and purpura, and occurs in erysipelas, the malarial and the malignant fevers, and in nasal diphtheria. Slight nose-bleed occurs in the first week of typhoid fever with such frequency as to acquire diagnostic importance. Nose-bleed not infrequently results from the congestion and shock of the violent convulsive cough of pertussis.

It is by no means a rare symptom in advanced disease of the kidneys and in various affections of the liver. In the venous engorgement of cardiac and pulmonary diseases, even with marked cyanosis, nose-bleed is uncommon. Finally, it may result from violent mental emotion.

When epistaxis is due to general causes, the blood escapes by capillary oozing from one, rarely two or three, limited areas of the respiratory portion of the cartilaginous septum, and in most instances it proceeds from one side only. In a very small proportion of the cases it comes from the turbinate bodies or from the floor of the nostril. The mucous membrane is deeply congested, of a violaceous-red color, and shows minute spots of ecchymosis.

Symptoms.—Prodromes sometimes occur. They consist of giddiness, fulness in the head, and a sensation of dryness, tickling, or obstruction in the nostrils, which impels the patient to more or less forcibly blow the nose. More frequently these symptoms are absent, the bleeding occurring suddenly and without warning. The blood may flow in drops or for a time in a continuous stream. Ordinary, slight nose-bleed generally ceases in a short time and is without immediate clinical importance whatever may be its remote significance. The graver bleedings may be protracted for hours or days, and while a fatal case is of rare occurrence, serious consequences are likely to follow profuse hemorrhage. The arrest takes place by clotting at the point of oozing. It is important to examine the pharynx, as the clot in the nostril may lead to the escape of blood by way of the posterior nares and its being swallowed. The vomiting of blood thus swallowed may be mistaken for hæmatemesis; its expulsion by cough, for hæmoptysis, but not if due care be observed in the investigation of the case.

II. DISEASES OF THE LARYNX.

i. Acute Catarrhal Laryngitis.

Definition.—Catarrhal inflammation of the mucous membrane of the larynx.

Etiology.—"Taking cold," exposure to a cold, damp atmosphere, overuse of the voice in speaking, shouting, or singing, especially under unfavorable atmospheric conditions, as in crowded and badly ventilated halls or in the open air, are common causes of acute laryngitis. It may follow the inhalation of air charged with smoke or irritating gases or vapors. Less frequently it is due to the lodgement of foreign bodies, the action of very hot liquids or corrosive poisons, or external violence. It occurs as a local manifestation of measles, influenza, and variola, and as a complication in other acute infectious diseases, as scarlet fever, enteric fever, and erysipelas. Catarrhal laryngitis is frequently associated with catarrh of the nasopharynx and bronchi. The predisposition to largynitis varies greatly in different families and individuals.

Symptoms.—There is a sensation of dryness and tickling in the throat; the inspiration of cold air and talking cause pain. Cough is a prominent symptom. It is tickling and hoarse, or "laryngeal" in character; at first dry, later attended with scanty mucopurulent expectoration, which is

(b) CHRONIC BRONCHITIS.

The affection very rarely arises as the termination of a single attack of acute bronchitis. It sometimes follows the repeated attacks which result from continuous exposure to the cause of catarrhal affections.

Etiology.—Chronic bronchitis is a secondary disease. It constitutes an important manifestation of certain circulatory derangements, as heart disease, thoracic aneurism, arteriosclerosis, some chronic pulmonary affections, as pneumoconiosis, asthma, and emphysema, and constitutional conditions, as gout and chronic uræmia. Important predisposing influences are climate and season. The winter cough of elderly and invalid persons is well known and is often absent when the patients are able to avoid the cold and changeable weather of the north by a temporary stay in a warm, dry, and equable climate. The influence of age is marked. Acute bronchitis is a disease of the young; chronic bronchitis a disease of the old. Chronic bronchitis is more common in men. It sometimes comes on in women at an early age without obvious cause and runs an indefinite course, with mild symptoms and slight secondary changes—bronchiectasis and emphysema.

Pathology.—The lesions are not evenly distributed. They affect different parts of the bronchial mucosa in varying degree and in irregular patches, and consist of loss of epithelium, atrophy of the glands and muscularis, thinning of the mucous membrane, and dilatation of the walls of the tubes. Bronchiectasis and emphysema gradually come to pass.

Symptoms.—There is cough of variable severity, less troublesome in dry warm weather and always worse in the cold and changeable weather of winter and early spring. There are often paroxysms in the morning, with comparative freedom throughout the day. In some cases cough is especially troublesome at night. The sputum has no constant characters. It differs in different cases and at different times in the same case. In the dry catarrh expectoration is absent. As a rule it is abundant, coming up in considerable quantities at a time. Sometimes there is a little tenacious mass of clear mucus at intervals. Very common is a clear, thin fluid. In the advanced cases shortness of breath is common upon exertion. It is due to emphysema and to some extent also to cardiac weakness. Fever is absent. There is no pain. The general health is often good, and the patients may be fully able to attend to their affairs. The disease is, however, progressive, and tends to an ultimate dyscrasia with advanced emphysema, bronchiectasis, and dilatation of the right heart.

Physical Signs.—In the early stages the physical signs do not differ from those of the stage of expectoration in acute bronchitis. Already perhaps the percussion sound has the vesiculotympanitic quality. There is prolongation of the expiratory sound, and various râles are heard, some sonorous, some sibilant, and occasionally moist râles of every size. At the bases posteriorly there are heard subcrepitant and crepitant râles, and when there is much fluid secretion there may be slight impairment of resonance.

Clinical Varieties.—(1) *Dry Bronchitis; Catarrhe Sec.*—This form is characterized by troublesome, paroxysmal cough, with slight expectoration, sometimes none at all. It is not uncommon in old people with emphysema.

Diagnosis.—DIRECT.—Acute laryngitis of the adult rarely presents difficulty in diagnosis. The severer cases suggest œdema of the larynx—acute laryngeal œdema—while those attended by complete loss of voice may be mistaken for hysterical aphonia or paralysis of the vocal cords due to other causes. These questions are at once settled by the laryngoscope.

DIFFERENTIAL.—In children the diagnosis of acute laryngitis is, in certain cases, attended with serious difficulty. The condition is to be distinguished from laryngismus stridulus by the presence of fever, the catarrhal symptoms, the mode of onset, the character of the paroxysms, their nocturnal occurrence, the hoarseness and loss of voice, the absence of the prolonged crowing inspiration which terminates the attack of laryngismus, and the course and duration of the disease.

The diagnosis between spasmodic croup and laryngeal diphtheria—*membranous croup*—may be for a time impossible. The principal points in favor of spasmodic croup are the milder character of the constitutional symptoms which precede the signs of laryngeal obstruction, the paroxysmal nature of the obstruction, and the complete relief between the attacks, the progressive amelioration of the symptoms after the second night, the absence of exudation upon the tonsils and adjacent parts, and the absence of enlargement of the cervical nodes.

Prognosis. The outlook is favorable. The most alarming symptoms, as a rule, promptly subside after the emesis caused by ipecac, or after a warm bath and the proper administration of simple sedative remedies.

iii. Subacute Laryngitis.

By far the larger number of cases of catarrhal laryngitis are of the mildest type. The patients are not ill; the only symptoms are a slight tickling cough, with hoarseness or aphonia.

The condition acquires importance from its great relative frequency; from the fact that, being accompanied by trifling subjective symptoms, it is likely to be neglected; and, finally, because in many cases prolonged habitual exposure to the original cause, or use of the voice when the larynx is slightly congested or inflamed, convert a passing local indisposition into a serious disease. In fact the larger proportion of cases of chronic laryngitis arise in this way.

iv. Chronic Laryngitis.

Etiology. This form may be the sequel of an acute attack; more [illegible] the result of the persistent action of causes which give rise to [illegible] use of the voice and its habitual overuse [illegible] in the open air are very common causes [illegible] associated with chronic [illegible] which is caused by habitual [illegible] with certain cases of nasal [illegible] cases of elongation of the uvula [illegible] males than in females and is especially a disease [illegible]

Symptoms.—There is a tickling sensation in the throat accompanied by a desire to obtain relief by coughing. As a rule pain is not present except after prolonged use of the voice or coughing. Many patients complain of a disagreeable feeling of dryness. The voice is rough and hoarse and at times almost lost. The cough is ringing, loud, deep; expectoration is as a rule scanty and tenacious, but occasionally abundant and sometimes fetid. Upon laryngoscopic examination the mucous membrane is found irregularly thickened and discolored, but the redness is less intense than in the acute form. The vocal cords are of a grayish-red color, and in debilitated and cachectic persons there may be seen minute superficial erosions. The epiglottis is in many cases irregularly thickened. The general health is often impaired.

Diagnosis.—The local sensations, chronic alteration of the voice, and peculiar cough suggest the true nature of the affection, but a positive diagnosis can be made only after careful laryngoscopic examination. In every case of chronic laryngitis the history of the patient in all particulars must be carefully investigated in order to determine whether or not the local affection be primary, or secondary to some other disease, as alcoholism, tuberculosis or syphilis.

v. Œdematous Laryngitis.

Acute Laryngeal Œdema; Œdema of the Glottis.

Etiology.—Œdema of the mucous and submucous tissues of the larynx occasionally occurs as a serious and frequently fatal complication in the course of acute catarrhal laryngitis, whether due to cold or to internal or external traumatism; in chronic disease of the larynx, as tuberculosis and syphilis; in connection with perichondritis of the larynx; as a complication of severe inflammatory affections of neighboring structures, as the tonsils, parotid glands, or the cellular tissue of the neck; in the course of acute infectious diseases, as scarlatina, typhoid fever, variola, and erysipelas; and, finally, as an extension of the general œdema in acute or chronic nephritis.

Symptoms.—Rapidly progressive dyspnœa is the chief symptom. It is at first inspiratory; later also expiratory. Respiration is accompanied by loud stridor. The voice becomes husky and soon fails. Signs of impending suffocation supervene, and unless relief is afforded death takes place in the course of a few hours. If a laryngoscopic examination prove successful, the epiglottic and aryepiglottic folds are seen to be greatly swollen, the latter almost meeting laterally; the false cords are also œdematous. These changes can be felt with the finger, and upon depressing the tongue the swollen rim of the epiglottis may sometimes be brought into view.

The diagnosis is unattended with difficulty and depends upon physical exploration.

vi. Pseudomembranous Laryngitis.

True Croup; Membranous Croup; Fibrinous Laryngitis.

Definition.—Inflammation of the mucous membrane of the larynx, resulting in the formation of a pseudomembrane or pellicle composed of a network of fibrin, embracing in its meshes leucocytes and necrotic epithelium.

Etiology.—Any agent capable of destroying the protecting epithelium of the laryngeal mucous membrane, thus permitting the escape of serum and white blood-corpuscles, may give rise to the formation of a pseudomembrane. Hence, this form of laryngitis may result, (a) from traumatism, as the inhalation of steam, hot smoke, or irritating and corrosive chemicals in the form of vapor or solution; (b) from the action of certain pathogenic micro-organisms.

Pseudomembranous laryngitis occurs at all seasons of the year. It especially affects young children between the ages of two and six. Cases in children under two and over seven years of age are much less common. Exceptionally the disease occurs at a later period of life. Boys are somewhat more liable than girls. This affection frequently occurs as a complication in scarlet fever and measles. In by far the greater number of cases it is a manifestation of diphtheria.

Symptoms.—The symptoms usually develop in the course of an attack of faucial diphtheria or of one of the exanthemata. Less frequently they arise as the manifestations of a primary laryngeal diphtheria. They point to progressive impairment of the functions of the larynx, with increasing obstruction to respiration and its consequences, and consist of hoarseness, aphonia, explosive and croupy cough, stridulous respiration, dyspnœa, recessions, restlessness, cyanosis, and stupor.

Diagnosis.—Acute progressive laryngeal stenosis in a young child is nearly always due to pseudomembranous laryngitis. If traces of the exudate can be discovered upon inspection of the throat, or if, upon physical examination, there can be detected coarse or whistling tracheal râles, or finally, if shreds of membrane are expectorated after paroxysms of explosive cough, the diagnosis becomes sure. It is equally so, in the absence of such confirmatory evidence, if the case occur in a locality already the scene of an epidemic of diphtheria. The fact that, even in pseudomembranous laryngitis, the signs of obstruction are at first paroxysmal and followed by intervals of partial relief must always be borne in mind. For this reason the early differential diagnosis between this disease and spasmodic laryngitis is not, in all instances, possible. In the latter, however, the intervals of relief are more complete and prolonged, the paroxysm not usually recurring until the succeeding night; the tendency is to progressive amelioration of the symptoms rather than progressive aggravation, and the signs of grave constitutional disturbance do not show themselves.

Prognosis.—Pseudomembranous laryngitis is, in the absence of treatment, an extremely fatal disease. The diphtheritic form, under the administration of antitoxin, frequently terminates in recovery. It is therefore imperatively necessary to at once employ this remedy.

vii. Tuberculous Laryngitis.

Laryngeal Phthisis; Throat Consumption.

Definition.—Inflammation of the tissues of the larynx caused by local tuberculosis.

Etiology.—Tuberculosis of the larynx may occur as a primary disease. Much more frequently, however, it is secondary to pulmonary tuberculosis. When the earliest symptoms are laryngeal, the disease remains for a time localized, but eventually the lungs become involved. Secondary tuberculous laryngitis occurs in more than 25 per cent. of the pulmonary cases. The laryngeal symptoms are pronounced and the lesions extensive and advanced in a much smaller percentage. A majority of the cases occur in males—a fact attributed to their greater liability to chronic catarrhal laryngitis, which acts as a predisposing cause. Not every case of chronic laryngitis in a consumptive individual is tuberculous. The mechanical irritation of frequent and severe cough and the contact of the sputum may cause chronic catarrhal laryngitis, which is aggravated by the condition of the patient, and which undoubtedly, after a time, predisposes to infection. In the tuberculous cases the mucous membrane is of a grayish, pale color, irregularly mottled and congested; it is at first swollen and studded with miliary tubercles, which by their coalescence form scattered tuberculous nodules. These nodules undergo caseation, as a result of which there form more or less extensive superficial ulcers, which show a tendency to spread. The floor of these ulcers is covered by a grayish exudation, and they are surrounded by a border of infiltrated and swollen tissue. They occur most frequently upon the arytenoids, in the interarytenoid space, upon the true cords, and on the epiglottis. The destruction of tissue extends deeply, implicating the submucosa, and in severe cases the perichondrium and cartilages, which undergo more or less extensive necrosis—tuberculous perichondritis and chondritis. The ulcers occasionally extend to the back of the tongue, to the pharynx, to the upper part of the œsophagus, and in severe cases to the pillars of the fauces and the tonsils. Complete erosion of the true cords not infrequently occurs, and the epiglottis is often destroyed throughout the greater part of its extent.

Symptoms.—The earlier symptoms are those of chronic laryngitis due to other causes. There is slight huskiness, which is at first intermittent and disappears after resting the voice. It soon becomes continuous, and gives place to a peculiar hoarseness, which in the advanced stages of the disease usually passes into complete aphonia. Cough is tickling, paroxysmal, and unproductive; it has the peculiar quality known as laryngeal, and may be distinguished in the same patient from the mere nervous cough of bronchial irritation. It is not at first distressing, but in cases of advanced ulceration it becomes husky and high-pitched, and is attended with pain. Spontaneous pain is not very common. There is often tenderness upon external pressure. Dysphagia is a prominent and most distressing symptom in advanced cases, especially when the epiglottis is involved, the arytenoids are extensively destroyed, or there is ulceration of the pharyngeal wall. In such cases the administration of nourishment is attended with difficulty, the attempt to take food of any kind giving rise to severe pain, urgent paroxysms of cough, and frequently to suffocative attacks. The difficulty in swallowing adds greatly to the sufferings of the patient and constitutes the most distressing symptom of the terminal stage of this form of tuberculosis. In the earlier stages the laryngoscope reveals the appearances due to chronic laryngeal catarrh. There is, however, greater pallor of the mucous membrane, together with some thickening over the arytenoids. Later the picture is characteristic. The vocal cords are thickened and eroded, and their motility is impaired; the epiglottis and arytenoid are infiltrated, and at various points superficial grayish ulcers with ill-defined borders are seen; finally, deep ulceration, with extensive loss of substance, occurs.

Diagnosis.—DIRECT.—In the earlier stages, especially in the absence of the evidences of pulmonary tuberculosis, the diagnosis of tuberculous laryngitis cannot always be made. Pallor of the laryngeal mucous membrane, thickening of the arytenoids, general failure of health on the part of the patient, and absence of response to local and constitutional treatment lead to the suspicion of tuberculous disease. This suspicion is confirmed by the appearance of the characteristic ulceration, the evidences of pulmonary tuberculosis, or the detection of tubercle bacilli in the sputum or the exudate scraped from the floor of the laryngeal ulcer. In selected cases the tuberculin tests may be made.

DIFFERENTIAL.—The diagnosis between tuberculosis and syphilis of the larynx is, in certain cases, attended with some degree of difficulty. In this connection, the greater tendency of syphilis to invade the pharynx, the fact that tuberculous ulceration of the larynx is, in general, progressive and continuously destructive, while syphilitic ulceration frequently shows a disposition to heal at one point while advancing at others, and, finally, the history of specific inflammatory or ulcerative lesions in other parts of the body in syphilis should receive due consideration.

Prognosis.—The course of tuberculous laryngitis is, as a rule, in the highest degree unfavorable. While, in the literature of the subject, cases of marked amelioration, or even of cure, especially in the primary form, are reported, the disease is so constantly fatal that the instances in which more than a temporary arrest occurs must be regarded as exceptional.

viii. Syphilitic Laryngitis.

Definition.—Inflammation of the larynx, occurring as a manifestation of syphilis, either hereditary or acquired.

Etiology.—The larynx is very frequently involved in syphilitic inflammation.

The catarrhal laryngitis of secondary syphilis presents nothing characteristic. Symmetrical superficial ulceration of the true and false cords occurs. Mucous patches, when present elsewhere, confirm the diagnosis, but they are not common in the larynx.

Much more frequent and important are tertiary lesions. Gummata, multiple or single, develop in the submucous tissues. They may undergo resolution, or, as is much more frequently the case, they break down, of these conditions—namely, swelling, spasm, and exudate—are present at the same time. Other views of less importance attribute the affection to spasm of the diaphragm or of all the inspiratory muscles. It has been suggested that the condition is similar to that in hay fever, with special manifestations due to differences in the anatomical site of the lesions. The sudden onset of the symptoms, the common association of asthma and hay fever, and the neurotic constitution of the subjects of these affections lend probability to this opinion.

Etiology.—PREDISPOSING INFLUENCES.—The neurotic temperament, which is transmitted from generation to generation, frequently carries with it the tendency to asthma. Asthma and epilepsy are sometimes associated. Males are more liable to the affection than females. Age plays an important part in the predisposition. Asthma usually begins early in life, often in childhood, and may continue throughout life. Pertussis is sometimes followed by asthma, and chronic bronchial catarrh is often accompanied by the attacks. These attacks are sharply differentiated from the dyspnœa which attends exertion, from which they are to be distinguished. Idiosyncrasy is important. The odors of certain plants or flowers, hay, artificial perfumes, ipecacuanha, and the emanations from animals, as the horse or cat, immediately cause the attack in certain persons. Violent emotions, especially if disagreeable, may act in the same way. Excesses at the table and certain articles of diet may be followed by the outbreak. Many persons remain free from the disease in the city, but at once suffer in the country or in some particular part of the country, or suffer in the city, but miss the attacks in the country or at the sea-shore. Others cannot use a feather pillow or sleep in a particular room. The most common source of reflex irritation is to be found in the mucosa of the upper respiratory tract. Forms of rhinitis, nasal polypi, hypertrophies of the inferior turbinated bones, enlarged tonsils, or adenoid vegetations are frequently present in asthmatics and relief very often follows their proper surgical treatment. The causal influence of uterine and ovarian disease is much less than at one time supposed. In old cases every "cold" may be attended with the paroxysm.

Symptoms.—The health is often excellent in the intervals between the attacks. The onset is sometimes preceded by prodromes, among which are chilliness, oppression in breathing, dyspeptic phenomena, vesical irritability, and mental depression. The attack mostly comes on at night, the patient waking from sleep with distressing difficulty in breathing, and oppression. There rapidly develops a paroxysm of the most urgent dyspnœa. Inspiration and expiration are both affected. The patient struggles for air. The respiratory muscles, and especially those which aid in expiratory efforts, are brought into forcible action. The abdominal muscles are tense and board-like. The expiration is prolonged. The face is pale, the expression anxious, and the patient refuses to talk. He may rush to the open window and gasp for air, with his arms fixed upon the frame. Small quantities of high-colored urine are passed at short intervals. There is a short, dry cough, with a peculiar, scanty, viscid
ration. The duration of the attack varies from a bad quarter of
half a day or longer. There are cases in which, with remissions

and exacerbations, the symptoms last for thirty-six or forty-eight hours. In severe paroxysms air hunger becomes urgent; restlessness, pallor, and cyanosis are accompanied by sweating, cold extremities, and a small, quick pulse. The intensity of the symptoms now abates; the cough becomes loose, the expectoration fluid and free, large quantities of urine may be passed, and in a short time there is complete relief. The patient now usually falls asleep. He may awake quite well or one or more further attacks may follow. Urticaria and in rare instances angioneurotic œdema have been observed during the attack. The sputum in the beginning of the attack contains Curschmann's spirals, Charcot-Leyden crystals, together with many leucocytes, mostly eosinophiles.

Physical Signs.—INSPECTION.—The chest has the inspiratory form. It appears large and suggests emphysema. The condition is, in fact, an acute emphysema, such as occurs also in pertussis. Under these circumstances the residual air is increased and the tidal air diminished, and in proportion as the ratio between them is deranged the chest becomes fixed and the dyspnœa urgent. The short, quick inspiration and the prolonged expiration are of diagnostic importance. PERCUSSION.—In mild attacks the signs upon percussion are little modified, the change amounting simply to a moderate hyperresonance, but in severe attacks the percussion sound is vesiculotympanitic. The pulmonary resonance extends downward two or three interspaces or more, the superficial cardiac dulness is much diminished, and the margins of the overdistended lungs scarcely change their position with the respiratory movements. AUSCULTATION.—The vesicular murmur is enfeebled. Great numbers of sibilant and sonorous râles are heard in all parts of the chest, and often from every part of the room. These râles constantly change in quality, pitch, and loudness, and are much more prolonged and intense upon inspiration than upon expiration. With free expectoration the râles become moist. The attacks recur at varying intervals. They sometimes come on in a series of three or four at night, with catarrhal symptoms in the daytime.

Diagnosis. — DIRECT. — The clinical picture is distinctive. Among the important criteria are sudden onset, mostly at night; expiratory dyspnœa; acute overdistention of the thorax as shown by the physical signs upon inspection and percussion; scanty expectoration with Curschmann's spirals and Charcot-Leyden crystals; loud wheezing and groaning râles; later abundant expectoration, with moist râles and relief of dyspnœa; eosinophilia.

DIFFERENTIAL.—*Emphysema and Chronic Bronchitis.*—The association of emphysema and bronchial asthma is a double one. The asthmatic tends to become emphysematous on the one hand, while, upon the other, attacks of asthma are common in emphysema. The obvious relationship between these conditions and chronic bronchitis has already been indicated. *Spasm of the Glottis.*—There may be true spasm, as in the laryngeal crisis of tabes. The dyspnœa is inspiratory and noisy, the respiratory movements of the larynx are extensive. The lungs are not overdistended, there is inspiratory retraction of the epigastric zone, and the peculiar cough, râles, and expectorati[illegible]thma are not present. Adductor spasm is of short du[illegible]roxysm of asthma is often prolonged. *Laryngismus Stridulus.*—This form of adductor spasm of children is characterized by apnœa. followed upon relaxation by a long-drawn inspiratory crowing sound. *Cardiac and Renal Dyspnœa.*—So-called cardiac and renal asthma have nothing in common with true asthma except dyspnœa. To call them asthma is a nosological error alike inconvenient to the teacher and misleading to the student.

Prognosis.—The symptoms are often alarming, but death does not occur during the attack. The removal of the sources of reflex irritation in the upper air-passages, improvement in the general condition of the patient, or permanent residence in a suitable climate is often followed by lasting relief.

IV. DISEASES OF THE PULMONARY TISSUE.

i. Circulatory Derangements.

(a) **Pulmonary Congestion.**—Congestion of the lungs is usually a symptomatic affection. There are two forms—active and passive.

1. **Active Congestion of the Lungs.**—The inhalation of overheated air, smoke, and other irritating substances, and overaction of the heart may cause this condition. The sudden death of firemen, open-air orators, and drunkards after exposure has been ascribed to it. The symptoms comprise great dyspnœa, oppression, feeble pulse, and cyanosis. The physical signs are restricted respiratory movements, impaired resonance, faint vesicular murmur, and fine râles. The mechanical interference with the circulation in pneumonia, intense bronchitis, pleurisy, and tuberculosis leads to overdistention of the capillaries in the adjacent lung tissue—collateral fluxion. The importance of this condition arises chiefly from the danger of œdema.

2. **Passive Congestion.**—Two forms are recognized—mechanical and hypostatic. MECHANICAL CONGESTION.—The condition is most marked in the dependent portion of the lungs. The essential factor is an obstacle to the return of the blood to the left ventricle. Its occurrence is favored by all conditions which restrict the respiratory expansion and contraction of the lungs and thus interfere with the normal movement of the blood current in the pulmonary vessels. Mechanical congestion of the lungs occurs in mitral stenosis and incompetency, emphysema, and in consequence of the pressure of tumors. The lung undergoes the changes known as brown induration. So long as compensation is maintained this condition is not marked by special symptoms of importance. When it is lost, dyspnœa, cough, and expectoration, often blood-stained and containing alveolar cells with blood pigment, occur. HYPOSTATIC CONGESTION.—The bases in this condition also are engorged with blood and serum. The condition is bilateral, one side being usually more deeply and more extensively congested than the other. Lobular patches may be airless, and bits of the affected tissue may sink in water. To this extreme condition are applied the terms splenization and hypostatic pneumonia. In fact there are frequently present in the congested regions foci of bronchopneumonia. Hypostatic congestion is common in protracted acute illness, as enteric fever; in chronic wast-

position, and is motionless upon inspiration, expiration, and attempts at phonation. In phonation the vocal cord and the arytenoid of the sound side pass beyond the median line. The voice is harsh, it easily breaks into a falsetto, and speaking is attended with effort. The cough is likewise harsh and brassy. Dyspnœa is not a symptom. In complete bilateral paralysis—a very rare condition—the cords occupy the median position and are immobile; their edges are slightly concave, as the aperture is sufficiently wide for respiration; dyspnœa is absent except upon exertion. Aphonia is complete and coughing is impossible.

2. BILATERAL PARALYSIS OF THE ABDUCTORS.—The posterior crico-arytenoids are involved. This form of laryngeal paralysis may occur as a central affection in the course of bulbar paralysis, multiple sclerosis, and locomotor ataxia. It may be produced by pressure upon both vagi or upon both recurrents. It is encountered as a rare form of hysterical palsy. Abductor paralysis may follow exposure to cold or may arise in the course of a laryngeal catarrh. The cords are approximated as in phonation. The glottis is not opened in inspiration; on the contrary, it acts like a valve, and is narrowed by the pressure of the air to a small slit. Inspiration is therefore difficult, prolonged, and stridulous, while expiration is unimpeded. Phonation is not affected. The ability to cough remains. This form of laryngeal paralysis is rare, but is attended with the danger of sudden suffocation. If the symptoms are progressive and the dyspnœa constant tracheotomy becomes necessary and the tube must be constantly worn.

3. UNILATERAL ABDUCTOR PARALYSIS.—One cord only may be affected in pressure-paralysis involving the recurrent of one side. Aneurism of the arch of the aorta, exerting pressure upon the left nerve, is by far the most common cause of this condition. The right nerve is especially liable to be involved in pleural thickening and retraction of the apex of the lung in the course of pulmonary tuberculosis. The vocal cord on the affected side remains fixed in the middle line during inspiration. The voice is sometimes unaffected; more commonly it is slightly harsh or rough. Dyspnœa and stridor are not often present. The movements of the other cord are normal.

4. ADDUCTOR PARALYSIS.—In the more common forms of adductor paralysis the lateral crico-arytenoids, the arytenoid, and the thyro-arytenoids, are implicated. It occurs as the result of exposure to cold or from overuse of the voice, and is very often the cause of loss of voice in catarrhal laryngitis; it is the usual form of paralysis in hysterical aphonia. The laryngoscope reveals the normal position and movement of the cords in respiration but their total failure to approximate on attempts at phonation. There is neither stridor nor dyspnœa; ability to cough is not affected, but aphonia is complete. Adductor paralysis may be partial. It is commonly bilateral, but in exceptional cases unilateral. In bilateral paresis of the thyro-arytenoids the glottis does not close completely on phonation, the margins of the cords being separated by an oval space. If one cord only is affected its margin remains concave. In paralysis of the arytenoid, which seldom occurs alone, the vocal cords are brought together in their anterior extent, but the failure of the arytenoid cartilages to approximate leaves a narrow triangular opening at the interarytenoid space.

Diagnosis.—DIRECT.—The diagnosis of general œdema of the lungs rests upon the occurrence of the above described symptoms in cases of œdematous or cachectic disease, cerebral disease or injury, angina pectoris, and impairment of the power of the left ventricle, especially when the power of the right heart is fairly well maintained. Collateral œdema may be at least provisionally diagnosticated when, in pneumonia or other inflammatory conditions, infarct, active circumscribed tuberculosis, or new growths, the symptoms are aggravated, the temperature rises, many moist râles are heard in the adjacent lung, and there is an abundant thin, blood-stained sputum. In rare instances acute œdema of the lungs follows the withdrawal of a pleural effusion by aspiration. The sputum is copious and has the characters above described. It is the result of the sudden removal of pressure upon the pulmonary vessels. Even more rare is the perforation of the lung by a serous pleural effusion. There are coarse râles usually confined to the affected side, and an abundant expectoration presenting the characters of the sputum in œdema but with a larger albumin content.

Prognosis.—General œdema of the lungs is frequently one of the manifestations of dissolution. There are, however, cases that recover under proper treatment. The outlook is at the best uncertain. Collateral œdema may mark an unfavorable turn in an acute illness or, as is frequently the case, subside under energetic management and be the point of departure for lasting improvement.

(c) **Pulmonary Hemorrhage.**—There are two forms. In the first the blood escapes into the bronchi and is expectorated—bronchopulmonary hemorrhage; in the second the blood is effused into the tissue of the lungs and air-cells—pulmonary apoplexy, hemorrhagic infarct.

1. **Bronchopulmonary Hemorrhage.**—*Bronchorrhagia; Hœmoptysis.* (See Part III, page 458.)

2. **Pulmonary Apoplexy.**—*Pneumorrhagia; Infarct.*—Anatomically two conditions are encountered: diffuse infiltration and hemorrhagic infarct.

Diffuse Hemorrhagic Infiltration of the Lungs.—The lung tissue and air-cells are densely and uniformly infiltrated with extravasated blood. The cut surface presents a smooth, somewhat gelatinous appearance and a blackish color. The condition is rare. It occurs more frequently in the hemorrhagic fevers, less often in sepsis and acute cerebral disease. The symptoms are dyspnœa, cyanosis, bloody sputum, blackish in color, and the nervous phenomena of collapse. Resonance is impaired. This form of hemorrhage is of no great diagnostic importance, since it constitutes the terminal event in an otherwise fatal malady.

Hemorrhagic Infarct.—The extravasation of blood is due to the arrest of circulation in a branch of the pulmonary artery by an embolus or thrombus. The anatomical condition and the symptoms differ greatly according to the location of the occlusion. If it occurs in the trunk or a main branch of the pulmonary artery, the whole or a large part of the blood is prevented from entering the pulmonary circuit, and there is dilatation of the right heart, a small, thready, arterial pulse, intense dyspnœa, cyanosis, and death from apnœa—*pulmonary apoplexy.* As the clinical manifestations om cardiac paralysis are the same the diagnosis remains an uncertain one.

When the obstruction takes place in a smaller branch of the pulmonary artery infarction usually occurs. These lesions are commonly at the periphery of the lung, and wedge-shaped, with the base resting upon the pleura, which is inflamed. Exceptionally they are located within the tissue of the lung and they are then irregularly oblong. Recent infarcts present the appearance of a blood-clot in the pulmonary tissue. The air-cells and their walls and the capillaries are packed with red blood-corpuscles. Infarcts are commonly multiple, exceptionally single. They vary in size from a pigeon's egg upward and may occupy a large portion of a lobe. In the arterial branch of supply may commonly be found the embolus or thrombus in the neighborhood of the apex. These obstructions, notwithstanding the fact that the pulmonary arteries are terminal, do not always cause infarction, owing to the width and free anastomosis of the capillaries and the ability of the bronchial vessels to maintain the circulation. The changes in the infarct are similar to those in blood-clots in other situations. The color becomes reddish-brown; the tissues contract, and are finally converted into a puckered, pigmented, fibroid nodule. The source of the embolus is to be sought in the right heart or peripheral venous system. The white thrombi which form in the right auricular appendix, the vegetations which develop upon the tricuspid leaflets in the rare cases of right-sided endocarditis, fibrin formations among the columnæ carneæ may be swept by the venous blood stream into the ramification of the pulmonary artery and become lodged. Any condition which tends to weaken the action of the right ventricle predisposes to this accident. Among these are valvular disease, especially mitral affections and myocardial degenerations. These emboli are not usually septic. Emboli from inflammatory or suppurating foci in various regions are infected and cause not a simple infarct but a metastatic abscess. When the general condition is septic, numerous small suppurating foci develop in the lung—*pyæmic abscesses*. These cannot always be recognized during life. When, however, a more extensive portion of the lung undergoes septic infarction pulmonary abscess results.

Symptoms.—The symptoms are neither constant nor distinctive. An initial chill may occur. It is, however, never so severe or prolonged as the ordinary chill of croupous pneumonia. Cyanosis, increased respiratory frequency, and dyspnœa at once develop. These symptoms vary in proportion to the number, and especially the size, of the infarctions. They may be slight or altogether absent. There is cough and the sputum contains blood (see Part III, page 459).

Physical Signs.—There is circumscribed dulness, more commonly in the lower lobes, especially on the right side, with bronchial breathing and high-pitched small mucous râles. Pleural friction over a limited area may very often be demonstrated.

Diagnosis.—DIRECT.—The diagnosis cannot always be made with certainty. The sudden occurrence of the above rational symptoms and physical signs in the course of chronic disease of the heart, or thrombosis of a crural or other vein, or some distant inflammatory or suppurative process warrants a provisional diagnosis.

DIFFERENTIAL.—*Croupous Pneumonia.*—There is a superficial resemblance in some of the cases; but the situation and outline of the consolidation, the character of the sputum in which the blood or hæmoglobin is more intimately mixed than in infarction, and the results of the laboratory examination of the sputum, which contains pneumococci, are distinctive. *Hæmoptysis in Mitral Disease, especially Mitral Stenosis.*—The occurrence of blood spitting, usually in small amounts and extending over a period of days or weeks, may suggest infarction. The differential diagnosis in the absence of physical signs or marked pulmonary symptoms is impossible. The blood may be due to engorgement of the pulmonary vessels. Its recurrence after exertion and at long intervals is in favor of the latter view. *Malignant Disease of the Lung.*—Blood spitting and signs of consolidation are present in both conditions. Pain, wasting, localizing physical signs, which gradually include more territory, cachexia, the presence of new growths elsewhere, and in particular of pigmented nævi or warts, with secondary nodules in the skin or subcutaneous tissues, implication of the lymph-nodes, or a history of the removal of a malignant growth, justify a provisional, and in well-marked cases a positive, diagnosis of cancer or sarcoma of the lung.

Air Embolism; Fat Embolism.—An embolus is any body transported by the circulating blood and capable, when arrested by the narrowness of the vessel, of obstructing the circulation. Emboli are usually too large to pass through the capillaries. They may be composed of fibrin masses, fragments of thrombi, vegetations or calcareous particles from endocardial vegetations, or fragments of neoplasms, which have penetrated the wall of a vessel. They may be infected or non-infected. There are two substances, differing from ordinary emboli in not consisting of solid bodies, which may give rise to urgent or fatal consequences when arrested in the capillaries of the pulmonary circulation; these are *air* and *fat.*

Etiology.—Air embolism occurs under certain circumstances when a vein is lacerated. Fat or oil embolism may follow fracture or injury to a bone, with escape of marrow into the tissues, or extensive laceration of adipose tissue, or its rapid breaking down in suppurative processes.

Symptoms.—The symptoms of air embolism are urgent or even fatal in cases in which the quantity of air is sufficient to form large numbers of bubbles which cannot pass the pulmonary capillaries—*an embolic shower.* They consist of an extreme degree of air hunger, loss of consciousness, convulsions, and collapse, and usually prove fatal in a brief period, sometimes instantly. In rare cases, however, these most alarming manifestations improve in consequence of the rapid absorption of the air, and prompt recovery takes place—a fortunate event not seen when multiple embolism, the embolic shower, is due to solid emboli. When the air bubble entering the vein is small or the air enters slowly the symptoms are commonly less urgent. No symptoms attend the presence of the most minute air bubbles which, having passed the wider pulmonary capillaries, are arrested in the capillaries of other organs.

Since the fat globules obstruct only the finest vessels in the lungs and only gradually enter the circulation, the sudden pulmonary symptoms seen in air embolism rarely present themselves. As a rule no symptoms

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Since the fat globules obstruct only the finest vessels in the lungs and only gradually enter the circulation, the sudden pulmonary symptoms seen in air embolism rarely present themselves. As a rule no symptoms

occur until some hours or days have elapsed from the time of the injury. Severe dyspnœa, œdema of the lungs, great depression, and coma may occur. As the fat emboli have no tendency to cause blood coagulation or thrombosis, they are gradually forced on under the pressure of the blood stream, or undergo resorption, and recovery takes place. The fat particles which pass the lungs may reach the cerebral capillaries or be arrested in the renal glomeruli. In the latter case they may be voided in the urine. Death is uncommon in fat embolism.

ii. Diseases Characterized by Changes in the Vesicular Structure of the Lungs.

(a) PULMONARY EMPHYSEMA.

Vesicular Emphysema; Substantive Emphysema; Pseudohypertrophic Emphysema.

Definition.—A chronic disease of the lungs, in which the infundibula and vesicles are dilated and their walls atrophied.

This is a well-defined clinical affection, and characterized by enlargement of the lungs, changes in the contour of the chest, incomplete aeration of the blood, and varying degrees of dyspnœa, especially upon exertion. It is to be distinguished from acute vesicular emphysema, compensatory emphysema, and interstitial and atrophic forms.

Etiology.—Heredity constitutes an important predisposing influence. The disease is frequently encountered in successive generations or in several members of a family, and is not uncommon in childhood. It has been ascribed to congenital defects in the development of the elastic fibres. Long-continued habitual intra-alveolar pressure, acting upon a congenitally defective alveolar structure, causes distention which tends to become permanent. The hyperinflation of the lungs which occurs in the paroxysms of whooping-cough and asthma is often the starting-point of emphysema. Repeated attacks of bronchitis or chronic bronchitis are often present. It is common also in players upon wind instruments, glass-blowers, and those whose work demands heavy lifting or prolonged muscular strain. The tension under these circumstances is expiratory. In violent attacks of cough and in straining, the glottis is closed and the intrathoracic tension greatly heightened. The parts of the lungs least supported by the chest wall, namely, the apices and anterior and inferior margins, show the most developed lesions of emphysema.

Anatomically the primary changes are in the lungs; the secondary changes in the wall of the thorax.

The lungs are voluminous, their margins meeting in the anterior mediastinum and extending downward to the extent of two fingers' breadth or more. The diaphragm is correspondingly displaced in a downward direction. They have lost their normal contractility and do not retract when the costal pleura is incised nor when withdrawn from the chest and laid upon the table. At the apices and borders there are seen beneath the pleura greatly distended air-vesicles, varying in diameter from

1 to 3 mm., and sometimes attaining the size of a pigeon's egg. There is marked diminution in the pigment usually found in the subpleural lymph-spaces. The atrophy of the distended vesicular walls leads to loss of their pumping function and permanent pressure upon the capillaries, and this to diminution in the intervesicular vascular supply. The infundibula are dilated, but bronchiectasis is not very common. The chambers of the right heart are dilated and hypertrophied. The pulmonary artery is in some instances dilated and atheromatous.

The chest permanently assumes the inspiratory form and the costal cartilages progressively undergo calcification and lose their elasticity.

Symptoms. — The derangement of function is twofold. First, the residual air is greatly increased and the tidal air correspondingly decreased in volume, and second, the pulmonary circulation is diminished. If these facts are borne in mind the significance of the symptoms is obvious. The lesions are gradually developed and it is only after they have made some progress that the characteristic symptoms and signs occur. At first there is merely dyspnœa and faint lividity upon exertion, and the chest merely looks full with the inspiratory contour. When the disease is fully developed the following symptoms are present: *Dyspnœa.*—The elasticity of the vesicular structure being to a great extent impaired, expiration is prolonged and difficult. The dyspnœa is chiefly expiratory. The loss of resiliency in the costal cartilages, the permanent maximum distention of the thorax, and the restricted play of the diaphragm render inspiration also difficult, even with the aid of the auxiliary muscles of respiration. The dyspnœa may be felt upon slight exertion or it may be continuous. It is increased upon the occurrence of the exacerbations of bronchitis, to which the patient suffering from emphysema is so liable. The breathing is puffy and wheezy. *Asthmatic Attacks.*—The dyspnœa under certain circumstances, and especially after "taking cold," often assumes a paroxysmal intensity, differing in no respect from true spasmodic asthma. *Cough* is a common symptom. It is due to the associated bronchitis. It is commonly wheezy and feebly explosive, and without much expectoration. It is usually less troublesome in warm, dry weather, and constitutes the recurrent winter cough of many elderly persons. *Cyanosis.*—The patients are frequently able to go about with lividity of high grade. This symptom is variable and may amount merely to a certain blueness of the lips and finger-nails while the patient is at rest.

Intermittent Cervical Hernia of the Lung of Spontaneous Origin.—C. B. Farr has reported a case of this kind and collected seven other instances from the literature. As a rule the condition accompanied chronic bronchitis with emphysema. It was unilateral in five cases and bilateral in three. These protrusions are of the form and size of a pear, with the base below and the apex extending upward. They are not present upon quiet breathing but prominent during cough and are the seat of a faint rustling murmur. They are resonant on percussion and may be reduced by gentle taxis. In several of the cases a hernial ring could be felt. They are to be differentiated from enlargements of the sinuses of the jugular veins, abscesses, and an empyema which presents in the neck.

is enfeebled or quite inaudible; bronchial breathing is absent; coarse dry or moist râles, often heard at a distance, decrease of vocal fremitus, and a faint or whispering voice are important phenomena of stenosis.

Diagnosis.—The differential diagnosis between laryngeal and tracheobronchial stenosis is of the highest practical importance. Among the symptoms which point to the larynx as the site of the obstruction are the abnormally increased respiratory movements of the larynx, the fixed attitude with the head somewhat thrown back, and the peculiar croupy, metallic quality in the respiration and cough—laryngeal cough. The question, however, is immediately settled by an examination with the laryngoscope.

Causal Diagnosis.—The examination of the trachea with the laryngoscope, when practicable, will determine the presence or absence of stenosis in that organ, and, when present, its nature. But there are often difficulties in the examination which are insurmountable. Goitre is manifest; tumor of the œsophagus is associated with dysphagia; mediastinal new growths and aneurism of the arch of the aorta are attended in common by signs of tumor, with displacement of the anterior border of the lung and venous obstruction, and separately by signs which, in the case of aneurism when present, are distinctive, as thrill, diastolic shock, and systolic pulsation. The presence of enlarged bronchial glands may be suspected when tuberculous lymph-nodes are elsewhere present. Malignant disease may be suspected when there are intermittent hemorrhagic sputum, enlarged lymphatics in the neck or axilla, and an otherwise inexplicable cachexia. A history of syphilis and specific lesions of the palate or larynx would lend importance to the assumption of cicatricial stenosis. The recurrence of attacks of fibrinous bronchitis with the expectoration of characteristic casts renders the diagnosis positive. Foreign bodies are easy of diagnosis. There is almost always a history, though under most unusual circumstances or in the case of the insane no history can be obtained. A fixed attitude, sudden attacks of suffocation, and the result of X-ray examination in the case of metallic and certain other substances are of diagnostic importance.

Prognosis.—In tracheobronchial stenosis the prognosis is as variable as the cause.

iv. Bronchial Asthma.

Nervous Asthma.

Definition.—A neurosis characterized by paroxysmal dyspnœa, a sense of constriction of the chest, and irregular recurrence. The symptoms and signs denote hyperæmia and swelling of the mucous membrane of the finer bronchial tubes, and the attack may be produced by direct or reflex irritation.

Asthma is not to be confounded with the dyspnœa of cardiac or renal disease, or that supervening upon exertion in emphysema and other chronic affections of the lungs and pleuræ.

Three principal hypotheses are advanced: (1) that the attack is due to spasm of the bronchial muscles; (2) that it is the result of hyperæmia and turgescence of the bronchial mucosa; (3) that it is caused by a peculiar inflammation of the bronchioles. It is probable that all three of these conditions—namely, swelling, spasm, and exudate—are present at the same time. Other views of less importance attribute the affection to spasm of the diaphragm or of all the inspiratory muscles. It has been suggested that the condition is similar to that in hay fever, with special manifestations due to differences in the anatomical site of the lesions. The sudden onset of the symptoms, the common association of asthma and hay fever, and the neurotic constitution of the subjects of these affections lend probability to this opinion.

Etiology.—PREDISPOSING INFLUENCES.—The neurotic temperament, which is transmitted from generation to generation, frequently carries with it the tendency to asthma. Asthma and epilepsy are sometimes associated. Males are more liable to the affection than females. Age plays an important part in the predisposition. Asthma usually begins early in life, often in childhood, and may continue throughout life. Pertussis is sometimes followed by asthma, and chronic bronchial catarrh is often accompanied by the attacks. These attacks are sharply differentiated from the dyspnœa which attends exertion, from which they are to be distinguished. Idiosyncrasy is important. The odors of certain plants or flowers, hay, artificial perfumes, ipecacuanha, and the emanations from animals, as the horse or cat, immediately cause the attack in certain persons. Violent emotions, especially if disagreeable, may act in the same way. Excesses at the table and certain articles of diet may be followed by the outbreak. Many persons remain free from the disease in the city, but at once suffer in the country or in some particular part of the country, or suffer in the city, but miss the attacks in the country or at the sea-shore. Others cannot use a feather pillow or sleep in a particular room. The most common source of reflex irritation is to be found in the mucosa of the upper respiratory tract. Forms of rhinitis, nasal polypi, hypertrophies of the inferior turbinated bones, enlarged tonsils, or adenoid vegetations are frequently present in asthmatics and relief very often follows their proper surgical treatment. The causal influence of uterine and ovarian disease is much less than at one time supposed. In old cases every "cold" may be attended with the paroxysm.

Symptoms.—The health is often excellent in the intervals between the attacks. The onset is sometimes preceded by prodromes, among which are chilliness, oppression in breathing, dyspeptic phenomena, vesical irritability, and mental depression. The attack mostly comes on at night, the patient waking from sleep with distressing difficulty in breathing, and oppression. There rapidly develops a paroxysm of the most urgent dyspnœa. Inspiration and expiration are both affected. The patient struggles for air. The respiratory muscles, and especially those which aid in expiratory efforts, are brought into forcible action. The abdominal muscles are tense and board-like. The expiration is prolonged. The face is pale, the expression anxious, and the patient refuses to talk. He may rush to the open window and gasp for air, with his arms fixed upon the frame. Small quantities of high-colored urine are passed at short intervals. There is a short, dry cough, with a peculiar, scanty, viscid expectoration. The duration of the attack varies from a bad quarter of an hour to half a day or longer. There are cases in which, with remissions and exacerbations, the symptoms last for thirty-six or forty-eight hours. In severe paroxysms air hunger becomes urgent; restlessness, pallor, and cyanosis are accompanied by sweating, cold extremities, and a small, quick pulse. The intensity of the symptoms now abates; the cough becomes loose, the expectoration fluid and free, large quantities of urine may be passed, and in a short time there is complete relief. The patient now usually falls asleep. He may awake quite well or one or more further attacks may follow. Urticaria and in rare instances angioneurotic œdema have been observed during the attack. The sputum in the beginning of the attack contains Curschmann's spirals, Charcot-Leyden crystals, together with many leucocytes, mostly eosinophiles.

Physical Signs.—Inspection.—The chest has the inspiratory form. It appears large and suggests emphysema. The condition is, in fact, an acute emphysema, such as occurs also in pertussis. Under these circumstances the residual air is increased and the tidal air diminished, and in proportion as the ratio between them is deranged the chest becomes fixed and the dyspnœa urgent. The short, quick inspiration and the prolonged expiration are of diagnostic importance. Percussion.—In mild attacks the signs upon percussion are little modified, the change amounting simply to a moderate hyperresonance, but in severe attacks the percussion sound is vesiculotympanitic. The pulmonary resonance extends downward two or three interspaces or more, the superficial cardiac dulness is much diminished, and the margins of the overdistended lungs scarcely change their position with the respiratory movements. Auscultation.—The vesicular murmur is enfeebled. Great numbers of sibilant and sonorous râles are heard in all parts of the chest, and often from every part of the room. These râles constantly change in quality, pitch, and loudness, and are much more prolonged and intense upon inspiration than upon expiration. With free expectoration the râles become moist. The attacks recur at varying intervals. They sometimes come on in a series of three or four at night, with catarrhal symptoms in the daytime.

Diagnosis. — Direct. — The clinical picture is distinctive. Among the important criteria are sudden onset, mostly at night; expiratory dyspnœa; acute overdistention of the thorax as shown by the physical signs upon inspection and percussion; scanty expectoration with Curschmann's spirals and Charcot-Leyden crystals; loud wheezing and groaning râles; later abundant expectoration, with moist râles and relief of dyspnœa; eosinophilia.

Differential.—*Emphysema and Chronic Bronchitis.*—The association of emphysema and bronchial asthma is a double one. The asthmatic tends to become emphysematous on the one hand, while, upon the other, attacks of asthma are common in emphysema. The obvious relationship between these conditions and chronic bronchitis has already been indicated. *Spasm of the Glottis.*—There may be true spasm, as in the laryngeal crisis of tabes. The dyspnœa is inspiratory and noisy, the respiratory movements of the larynx are extensive. The lungs are not overdistended, there is inspiratory retraction of the epigastric zone, and the peculiar cough, râles, and expectoration of asthma are not present. Adductor spasm is of short duration, while the paroxysm of asthma is often prolonged. *Laryngismus Stridulus.*—This form of adductor spasm of children is characterized by apnœa. followed upon relaxation by a long-drawn inspiratory crowing sound. *Cardiac and Renal Dyspnœa.*—So-called cardiac and renal asthma have nothing in common with true asthma except dyspnœa. To call them asthma is a nosological error alike inconvenient to the teacher and misleading to the student.

Prognosis.—The symptoms are often alarming, but death does not occur during the attack. The removal of the sources of reflex irritation in the upper air-passages, improvement in the general condition of the patient, or permanent residence in a suitable climate is often followed by lasting relief.

IV. DISEASES OF THE PULMONARY TISSUE.

i. Circulatory Derangements.

(a) **Pulmonary Congestion.** — Congestion of the lungs is usually a symptomatic affection. There are two forms — active and passive.

1. **Active Congestion of the Lungs.** — The inhalation of overheated air, smoke, and other irritating substances, and overaction of the heart may cause this condition. The sudden death of firemen, open-air orators, and drunkards after exposure has been ascribed to it. The symptoms comprise great dyspnœa, oppression, feeble pulse, and cyanosis. The physical signs are restricted respiratory movements, impaired resonance, faint vesicular murmur, and fine râles. The mechanical interference with the circulation in pneumonia, intense bronchitis, pleurisy, and tuberculosis leads to overdistention of the capillaries in the adjacent lung tissue—collateral fluxion. The importance of this condition arises chiefly from the danger of œdema.

2. **Passive Congestion.**—Two forms are recognized—mechanical and hypostatic. MECHANICAL CONGESTION.—The condition is most marked in the dependent portion of the lungs. The essential factor is an obstacle to the return of the blood to the left ventricle. Its occurrence is favored by all conditions which restrict the respiratory expansion and contraction of the lungs and thus interfere with the normal movement of the blood current in the pulmonary vessels. Mechanical congestion of the lungs occurs in mitral stenosis and incompetency, emphysema, and in consequence of the pressure of tumors. The lung undergoes the changes known as brown induration. So long as compensation is maintained this condition is not marked by special symptoms of importance. When it is lost, dyspnœa, cough, and expectoration, often blood-stained and containing alveolar cells with blood pigment, occur. HYPOSTATIC CONGESTION.—The bases in this condition also are engorged with blood and serum. The condition is bilateral, one side being usually more deeply and more extensively congested than the other. Lobular patches may be airless, and bits of the affected tissue may sink in water. To this extreme condition are applied the terms splenization and hypostatic pneumonia. In fact there are frequently present in the congested regions foci of bronchopneumonia. Hypostatic congestion is common in protracted acute illness, as enteric fever; in chronic wast-

ing diseases, as tuberculosis and cancer; in injury and disease of the brain, especially apoplexy, and in prolonged coma. There are no characteristic symptoms and the diagnosis rests upon the presence, over the lower lobes posteriorly, of impaired resonance, feeble respiratory sounds, patches of bronchovesicular breathing, and small mucous or subcrepitant râles.

(b) **Pulmonary Œdema.**—There are two forms of œdema of the lungs—general and collateral.

The termination of intense congestion of the lungs is, in many cases, the transudation of blood-serum from the overdistended capillary vessels into the vesicles themselves and their walls. The escape of serum into the small and later into larger bronchi follows and is of clinical importance. Pathologically the condition is one of serous infiltration of the pulmonary tissue, with accumulation in the air-cells and bronchi. The œdematous lung is heavy, pits on pressure, and exudes abundantly from the cut surface clear or blood-tinged serum.

1. **General Œdema.**—*Œdema from Engorgement; Stasis Œdema.*—The œdema is bilateral and involves the whole of both lungs. The bases are especially affected. Causal factors are overdistention of the capillary vessels, hydræmia which leads to nutritive changes in the walls of the vessels and a weakened left ventricle. The condition is very often a terminal one and accompanies the death agony. It is common in affections characterized by dropsies, as fatal anæmias, disease of the heart and kidneys, especially the cardiorenal affection, and cachexias generally. It occurs also without previous dropsy in cerebral diseases, acute pulmonary congestion, and angina pectoris. General œdema of the uninvolved portions of the lungs may occur in the *stadium ultimum* of croupous pneumonia.

2. **Collateral Œdema.**—*Local Œdema of the Lungs.*—This condition is the outcome of the collateral fluxion in the pulmonary tissue bordering on pneumonias, infarcts, active foci of tuberculous inflammation, or new growths. The cut surface exudes a bloody serum. The entire lung is not involved and the opposite lung may wholly escape. This constitutes the form known as inflammatory œdema.

Symptoms.—Pulmonary œdema may develop gradually or with great suddenness. The symptoms of the pre-existing malady are aggravated. Progressive dyspnœa, cough, copious, frothy, thin, fluid sputum which, in the case of collateral œdema, is often bloody, characterize the condition (see Part III, page 458). As it progresses cyanosis and the stupor and convulsive tremblings which indicate the action of carbon dioxide upon the nervous system occur. Fever does not usually accompany stasis œdema, but in inflammatory œdema there may be a rise in temperature.

Physical Signs.—The percussion resonance is usually somewhat impaired over the bases posteriorly and has the tympanitic quality. In very abundant serous transudation there may be dulness. Upon auscultation the respiratory murmur is enfeebled, and over the whole extent of the involved lung tissue are heard moist bronchial subcrepitant and crepitant râles. Vesiculobronchial or pure bronchial breathing may be heard in limited areas at the bases in intense œdema, and corresponds to the areas of dulness.

Diagnosis.—DIRECT.—The diagnosis of general œdema of the lungs rests upon the occurrence of the above described symptoms in cases of œdematous or cachectic disease, cerebral disease or injury, angina pectoris, and impairment of the power of the left ventricle, especially when the power of the right heart is fairly well maintained. Collateral œdema may be at least provisionally diagnosticated when, in pneumonia or other inflammatory conditions, infarct, active circumscribed tuberculosis, or new growths, the symptoms are aggravated, the temperature rises, many moist râles are heard in the adjacent lung, and there is an abundant thin, blood-stained sputum. In rare instances acute œdema of the lungs follows the withdrawal of a pleural effusion by aspiration. The sputum is copious and has the characters above described. It is the result of the sudden removal of pressure upon the pulmonary vessels. Even more rare is the perforation of the lung by a serous pleural effusion. There are coarse râles usually confined to the affected side, and an abundant expectoration presenting the characters of the sputum in œdema but with a larger albumin content.

Prognosis.—General œdema of the lungs is frequently one of the manifestations of dissolution. There are, however, cases that recover under proper treatment. The outlook is at the best uncertain. Collateral œdema may mark an unfavorable turn in an acute illness or, as is frequently the case, subside under energetic management and be the point of departure for lasting improvement.

(c) **Pulmonary Hemorrhage.**—There are two forms. In the first the blood escapes into the bronchi and is expectorated—bronchopulmonary hemorrhage; in the second the blood is effused into the tissue of the lungs and air-cells—pulmonary apoplexy, hemorrhagic infarct.

1. **Bronchopulmonary Hemorrhage.**—*Bronchorrhagia; Hæmoptysis.* (See Part III, page 458.)

2. **Pulmonary Apoplexy.**—*Pneumorrhagia; Infarct.*—Anatomically two conditions are encountered: diffuse infiltration and hemorrhagic infarct.

Diffuse Hemorrhagic Infiltration of the Lungs.—The lung tissue and air-cells are densely and uniformly infiltrated with extravasated blood. The cut surface presents a smooth, somewhat gelatinous appearance and a blackish color. The condition is rare. It occurs more frequently in the hemorrhagic fevers, less often in sepsis and acute cerebral disease. The symptoms are dyspnœa, cyanosis, bloody sputum, blackish in color, and the nervous phenomena of collapse. Resonance is impaired. This form of hemorrhage is of no great diagnostic importance, since it constitutes the terminal event in an otherwise fatal malady.

Hemorrhagic Infarct.—The extravasation of blood is due to the arrest of circulation in a branch of the pulmonary artery by an embolus or thrombus. The anatomical condition and the symptoms differ greatly according to the location of the occlusion. If it occurs in the trunk or a main branch of the pulmonary artery, the whole or a large part of the blood is prevented from entering the pulmonary circuit, and there is dilatation of the right heart, a small, thready, arterial pulse, intense dyspnœa, cyanosis, and death from apnœa—*pulmonary apoplexy.* As the clinical manifestations from cardiac paralysis are the same the diagnosis remains an uncertain one.

When the obstruction takes place in a smaller branch of the pulmonary artery infarction usually occurs. These lesions are commonly at the periphery of the lung, and wedge-shaped, with the base resting upon the pleura, which is inflamed. Exceptionally they are located within the tissue of the lung and they are then irregularly oblong. Recent infarcts present the appearance of a blood-clot in the pulmonary tissue. The air-cells and their walls and the capillaries are packed with red blood-corpuscles. Infarcts are commonly multiple, exceptionally single. They vary in size from a pigeon's egg upward and may occupy a large portion of a lobe. In the arterial branch of supply may commonly be found the embolus or thrombus in the neighborhood of the apex. These obstructions, notwithstanding the fact that the pulmonary arteries are terminal, do not always cause infarction, owing to the width and free anastomosis of the capillaries and the ability of the bronchial vessels to maintain the circulation. The changes in the infarct are similar to those in blood-clots in other situations. The color becomes reddish-brown; the tissues contract, and are finally converted into a puckered, pigmented, fibroid nodule. The source of the embolus is to be sought in the right heart or peripheral venous system. The white thrombi which form in the right auricular appendix, the vegetations which develop upon the tricuspid leaflets in the rare cases of right-sided endocarditis, fibrin formations among the columnæ carneæ may be swept by the venous blood stream into the ramification of the pulmonary artery and become lodged. Any condition which tends to weaken the action of the right ventricle predisposes to this accident. Among these are valvular disease, especially mitral affections and myocardial degenerations. These emboli are not usually septic. Emboli from inflammatory or suppurating foci in various regions are infected and cause not a simple infarct but a metastatic abscess. When the general condition is septic, numerous small suppurating foci develop in the lung—*pyæmic abscesses*. These cannot always be recognized during life. When, however, a more extensive portion of the lung undergoes septic infarction pulmonary abscess results.

Symptoms.—The symptoms are neither constant nor distinctive. An initial chill may occur. It is, however, never so severe or prolonged as the ordinary chill of croupous pneumonia. Cyanosis, increased respiratory frequency, and dyspnœa at once develop. These symptoms vary in proportion to the number, and especially the size, of the infarctions. They may be slight or altogether absent. There is cough and the sputum contains blood (see Part III, page 459).

Physical Signs.—There is circumscribed dulness, more commonly in the lower lobes, especially on the right side, with bronchial breathing and high-pitched small mucous râles. Pleural friction over a limited area may very often be demonstrated.

Diagnosis.—DIRECT.—The diagnosis cannot always be made with certainty. The sudden occurrence of the above rational symptoms and physical signs in the course of chronic disease of the heart, or thrombosis of a crural or other vein, or some distant inflammatory or suppurative process warrants a provisional diagnosis.

Differential.—*Croupous Pneumonia.*—There is a superficial resemblance in some of the cases; but the situation and outline of the consolidation, the character of the sputum in which the blood or hæmoglobin is more intimately mixed than in infarction, and the results of the laboratory examination of the sputum, which contains pneumococci, are distinctive. *Hæmoptysis in Mitral Disease, especially Mitral Stenosis.*—The occurrence of blood spitting, usually in small amounts and extending over a period of days or weeks, may suggest infarction. The differential diagnosis in the absence of physical signs or marked pulmonary symptoms is impossible. The blood may be due to engorgement of the pulmonary vessels. Its recurrence after exertion and at long intervals is in favor of the latter view. *Malignant Disease of the Lung.*—Blood spitting and signs of consolidation are present in both conditions. Pain, wasting, localizing physical signs, which gradually include more territory, cachexia, the presence of new growths elsewhere, and in particular of pigmented nævi or warts, with secondary nodules in the skin or subcutaneous tissues, implication of the lymph-nodes, or a history of the removal of a malignant growth, justify a provisional, and in well-marked cases a positive, diagnosis of cancer or sarcoma of the lung.

Air Embolism; Fat Embolism.—An embolus is any body transported by the circulating blood and capable, when arrested by the narrowness of the vessel, of obstructing the circulation. Emboli are usually too large to pass through the capillaries. They may be composed of fibrin masses, fragments of thrombi, vegetations or calcareous particles from endocardial vegetations, or fragments of neoplasms, which have penetrated the wall of a vessel. They may be infected or non-infected. There are two substances, differing from ordinary emboli in not consisting of solid bodies, which may give rise to urgent or fatal consequences when arrested in the capillaries of the pulmonary circulation; these are *air* and *fat.*

Etiology.—Air embolism occurs under certain circumstances when a vein is lacerated. Fat or oil embolism may follow fracture or injury to a bone, with escape of marrow into the tissues, or extensive laceration of adipose tissue, or its rapid breaking down in suppurative processes.

Symptoms.—The symptoms of air embolism are urgent or even fatal in cases in which the quantity of air is sufficient to form large numbers of bubbles which cannot pass the pulmonary capillaries—*an embolic shower.* They consist of an extreme degree of air hunger, loss of consciousness, convulsions, and collapse, and usually prove fatal in a brief period, sometimes instantly. In rare cases, however, these most alarming manifestations improve in consequence of the rapid absorption of the air, and prompt recovery takes place—a fortunate event not seen when multiple embolism, the embolic shower, is due to solid emboli. When the air bubble entering the vein is small or the air enters slowly the symptoms are commonly less urgent. No symptoms attend the presence of the most minute air bubbles which, having passed the wider pulmonary capillaries, are arrested in the capillaries of other organs.

Since the fat globules obstruct only the finest vessels in the lungs and only gradually enter the circulation, the sudden pulmonary symptoms seen in air embolism rarely present themselves. As a rule no symptoms

occur until some hours or days have elapsed from the time of the injury. Severe dyspnœa, œdema of the lungs, great depression, and coma may occur. As the fat emboli have no tendency to cause blood coagulation or thrombosis, they are gradually forced on under the pressure of the blood stream, or undergo resorption, and recovery takes place. The fat particles which pass the lungs may reach the cerebral capillaries or be arrested in the renal glomeruli. In the latter case they may be voided in the urine. Death is uncommon in fat embolism.

ii. Diseases Characterized by Changes in the Vesicular Structure of the Lungs.

(a) PULMONARY EMPHYSEMA.

Vesicular Emphysema; Substantive Emphysema; Pseudohypertrophic Emphysema.

Definition.—A chronic disease of the lungs, in which the infundibula and vesicles are dilated and their walls atrophied.

This is a well-defined clinical affection, and characterized by enlargement of the lungs, changes in the contour of the chest, incomplete aeration of the blood, and varying degrees of dyspnœa, especially upon exertion. It is to be distinguished from acute vesicular emphysema, compensatory emphysema, and interstitial and atrophic forms.

Etiology.—Heredity constitutes an important predisposing influence. The disease is frequently encountered in successive generations or in several members of a family, and is not uncommon in childhood. It has been ascribed to congenital defects in the development of the elastic fibres. Long-continued habitual intra-alveolar pressure, acting upon a congenitally defective alveolar structure, causes distention which tends to become permanent. The hyperinflation of the lungs which occurs in the paroxysms of whooping-cough and asthma is often the starting-point of emphysema. Repeated attacks of bronchitis or chronic bronchitis are often present. It is common also in players upon wind instruments, glass-blowers, and those whose work demands heavy lifting or prolonged muscular strain. The tension under these circumstances is expiratory. In violent attacks of cough and in straining, the glottis is closed and the intrathoracic tension greatly heightened. The parts of the lungs least supported by the chest wall, namely, the apices and anterior and inferior margins, show the most developed lesions of emphysema.

Anatomically the primary changes are in the lungs; the secondary changes in the wall of the thorax.

The lungs are voluminous, their margins meeting in the anterior mediastinum and extending downward to the extent of two fingers' breadth or more. The diaphragm is correspondingly displaced in a downward direction. They have lost their normal contractility and do not retract when the costal pleura is incised nor when withdrawn from the chest and laid upon the table. At the apices and borders there are seen beneath the pleura greatly distended air-vesicles, varying in diameter from

1 to 3 mm., and sometimes attaining the size of a pigeon's egg. There is marked diminution in the pigment usually found in the subpleural lymph-spaces. The atrophy of the distended vesicular walls leads to loss of their pumping function and permanent pressure upon the capillaries, and this to diminution in the intervesicular vascular supply. The infundibula are dilated, but bronchiectasis is not very common. The chambers of the right heart are dilated and hypertrophied. The pulmonary artery is in some instances dilated and atheromatous.

The chest permanently assumes the inspiratory form and the costal cartilages progressively undergo calcification and lose their elasticity.

Symptoms. — The derangement of function is twofold. First, the residual air is greatly increased and the tidal air correspondingly decreased in volume, and second, the pulmonary circulation is diminished. If these facts are borne in mind the significance of the symptoms is obvious. The lesions are gradually developed and it is only after they have made some progress that the characteristic symptoms and signs occur. At first there is merely dyspnœa and faint lividity upon exertion, and the chest merely looks full with the inspiratory contour. When the disease is fully developed the following symptoms are present: *Dyspnœa.*—The elasticity of the vesicular structure being to a great extent impaired, expiration is prolonged and difficult. The dyspnœa is chiefly expiratory. The loss of resiliency in the costal cartilages, the permanent maximum distention of the thorax, and the restricted play of the diaphragm render inspiration also difficult, even with the aid of the auxiliary muscles of respiration. The dyspnœa may be felt upon slight exertion or it may be continuous. It is increased upon the occurrence of the exacerbations of bronchitis, to which the patient suffering from emphysema is so liable. The breathing is puffy and wheezy. *Asthmatic Attacks.*—The dyspnœa under certain circumstances, and especially after "taking cold," often assumes a paroxysmal intensity, differing in no respect from true spasmodic asthma. *Cough* is a common symptom. It is due to the associated bronchitis. It is commonly wheezy and feebly explosive, and without much expectoration. It is usually less troublesome in warm, dry weather, and constitutes the recurrent winter cough of many elderly persons. *Cyanosis.*—The patients are frequently able to go about with lividity of high grade. This symptom is variable and may amount merely to a certain blueness of the lips and finger-nails while the patient is at rest.

Intermittent Cervical Hernia of the Lung of Spontaneous Origin.—C. B. Farr has reported a case of this kind and collected seven other instances from the literature. As a rule the condition accompanied chronic bronchitis with emphysema. It was unilateral in five cases and bilateral in three. These protrusions are of the form and size of a pear, with the base below and the apex extending upward. They are not present upon quiet breathing but prominent during cough and are the seat of a faint rustling murmur. They are resonant on percussion and may be reduced by gentle taxis. In several of the cases a hernial ring could be felt. They are to be differentiated from enlargements of the sinuses of the jugular veins, abscesses, and an empyema which presents in the neck.

Physical Signs.—*Inspection.*—In advanced cases the deformity of the chest is typical. It is barrel-shaped (see p. 64). The elevation of the sternum and ribs gives the neck a shortened appearance. The respiratory movements appear forcible, but the thorax does not expand. Many dilated superficial venules are seen along the line of attachment of the diaphragm. The cardiac impulse is not visible. Epigastric pulsation and dilated cervical veins, sometimes pulsating, are signs of a dilated and overdistended right heart. The deformity of the chest is less marked in those cases in which emphysema has commenced in advanced life at a period when the cartilages have already become calcified. *Palpation.*—The lack of respiratory excursus is very obvious upon palpation. The vocal fremitus is enfeebled. The impulse of the heart cannot be located. There is pulsation over the lower sternal and epigastric regions. *Mensuration.*—The restricted expansion of the chest is confirmed by careful measurement, and the rounded contour by the cyrtometer. *Percussion.*—The resonance is of vesiculotympanitic quality, especially sonorous over the lateral and posterior regions. The percussion sound has been compared with that elicited upon tapping a bandbox—*Schachtelton.* More important than the quality of the percussion sound are the borders of the expanded lungs which it marks. They are extended in every direction and may reach in front to the eighth rib and below it, behind to the level of the twelfth dorsal or even the second lumbar vertebra. The cardiac dulness may be completely obliterated. The liver and splenic dulness are much lowered. *Auscultation.*—The vesicular murmur is greatly enfeebled. Its very faintness is suggestive. The expiratory element is not often audible. Instead of it, however, there are many sibilant and wheezy râles. The sounds of the heart are faint and distant, the pulmonary second sound is often accentuated, and in advanced cases a tricuspid regurgitant murmur may be heard.

The effect of the lesions of emphysema upon the circulation is to obstruct the pulmonary circuit; to dilate and enfeeble the right ventricle; to diminish the arterial blood; and to increase the accumulation of venous blood. The enfeeblement of the right heart is, however, delayed by compensatory hypertrophy by which the circulatory faults are postponed. Ultimately, however, compensation fails and the results of venous engorgement become manifest. These are persistent cyanosis, pulsation in the veins of the neck, hepatic enlargement, diminished secretion of albuminous urine, œdema, anasarca and effusions into the great serous sacs, and gastric and intestinal catarrh. When these conditions are present there is almost always a complicating catarrhal bronchitis, manifest by the ordinary symptoms of cough and expectoration, the latter being usually mucoid and viscid, sometimes purulent. The presence of blood is not common and suggests either a complicating tuberculosis or pulmonary infarct. The general nutrition is impaired.

Diagnosis.—DIRECT.—In well-developed cases the condition may be at once recognized by the contour and diminished mobility of the chest, the dyspnœa and cyanotic lips, the spare frame, and a facies which is very suggestive to those who have observed many cases. The physical signs, and especially the displaced boundaries of the lungs as determined by percussion, are confirmatory.

DIFFERENTIAL. — *Acute Vesicular Emphysema; Acute Over-inflation of the Lungs.*—During the paroxysm of bronchial asthma and pertussis, and in bronchitis of the smaller tubes, the lungs are frequently much distended. The chest maintains the inspiratory form, and upon a single examination the condition might be confounded with emphysema. The anamnesis is important. The borders of the lung are enlarged, but not beyond the limits of normal full-held inspiration, and in the course of a little time after the termination of the primary disease they regain their normal position upon inspiration and expiration. In congenital weakness of the lung tissue such attacks may become the point of departure for true emphysema. A similar condition occurs in cases of cardiac dyspnœa and angina pectoris. The lungs are distended, their borders extended, and the expiration is prolonged and accompanied by wheezing râles. *Compensatory Emphysema; Vicarious Emphysema.*—When local lesions in the lung or pleura interfere with expansion upon inspiration, the unaffected tissue takes upon itself increased functional activity—*vicarious respiration.* This change may involve parts adjacent to the lesion, an adjoining lobe, or the opposite lung. It occurs in bronchopneumonia and around tuberculous foci and cicatrices, the air-cells of the unaffected lobules undergoing a vicarious distention; in pulmonary cirrhosis, the unaffected lung undergoing vicarious enlargement, and to a less extent in pleural adhesions and effusions and in pneumothorax. This process, at first truly compensatory and physiological, becomes after a time pathological. The vesicular walls and capillaries undergo atrophy; a circumscribed or partial emphysema comes to pass. In bronchopneumonia or pulmonary tuberculosis with scattered lobular lesions the distention of the adjacent air-cells masks the dulness and may render the diagnosis obscure; in retracted and cirrhotic conditions of one lung, such as follow the resorption of the pleural effusion, the borders of the opposite lung are much distended and may be followed by careful percussion, not only in their inferior extent, but also over the area of superficial cardiac dulness and beyond the median line toward the contracted side. *Atrophic Emphysema; Atrophy of the Lungs; Senile Emphysema.*—This is a purely senile change and is occasionally encountered in wizened old people with small, narrow chests which are permanently in the expiratory form. The alveolar walls and interalveolar capillaries are extensively atrophied, and the alveoli in places converted into series of large communicating cells. The lungs themselves are small and the thorax conforms to the changes in the contained organs. It is flattened, the shoulders droop, the costal angle is acute, the neck appears elongated. The diaphragm is high. The right heart does not show dilatation and hypertrophy as in large-lunged emphysema, because it also undergoes a corresponding senile involution. The respiratory muscles are atrophic. *Interstitial Emphysema.* — Small bubbles of air find their way into the interlobular and subpleural tissues. Their access is by way of tracheotomy or other surgical or accidental wounds of the neck or throat; less frequently through rupture of the alveolar walls by violent coughing, contusions of the chest, or ulceration. When the opening is near the root of the lung air may pass to the mediastinal connective tissue. The condition is rare. Mediastinal emphysema may be diagnosticated when the causal factors are present together with crepitating subcutaneous emphysema of the neck or chest, absence of cardiac dulness and impulse, obliteration of the sternal ends of the intercostal spaces, and upon auscultation a fine crepitus synchronous with the action of the heart. The veins of the neck are distended. Subpleural and interlobular emphysema not extending to the mediastinum does not usually present positive diagnostic phenomena. Rupture of an air bleb in subpleural emphysema may cause pneumothorax.

Prognosis.—Substantive emphysema is incurable, but much can be done to relieve the sufferings of the patient and to prolong his life. Treatment of the bronchitis, a favorable climate, and attention to the state of the heart are important. Death usually occurs in consequence of some intercurrent disease, as pneumonia or bronchopneumonia.

(b) PULMONARY ATELECTASIS.

Collapse of the Lung.

Definition.—An airless condition of lobules or parts of the lung, the vesicles being in a state of collapse and not occupied by fluid or solid exudate or other pathological products. It is congenital or acquired.

CONGENITAL ATELECTASIS.—The lung is airless at birth, or remains only partially expanded in consequence of deficient inspiratory efforts or obstruction of the respiratory passages by meconium or mucus. The respiration is feeble, ultimately gasping, shallow and rapid, the lower part of the thorax and the epigastrium are retracted during inspiration, there is universal deep, bluish-black cyanosis, together with muscular twitchings or general shivering convulsions.

ACQUIRED ATELECTASIS.—This is a secondary anatomical lesion. It is caused in two ways: (a) by obstruction of small bronchi by mucus or swelling of the mucosa—as in bronchopneumonia, the intra-alveolar air undergoing resorption by the capillaries and the vesicles collapsing by virtue of the elasticity of their walls; or (b) by pressure upon the lung such as occurs in pleural or pericardial effusions, pneumothorax and pneumopericardium, tumors of the lungs or pleura, mediastinal tumors or massive enlargement of the heart, scoliosis, and abdominal disorders which restrict the movements of the diaphragm, including persistent meteorism, large ascites, and visceral and other tumors. Diffuse atelectasis may arise in paretic conditions involving the muscles of respiration.

Symptoms.—The clinical phenomena vary according to the extent of lung tissue involved. In slight cases they are not distinctive. There may be moderate dyspnœa, cyanosis, a vesiculotympanitic percussion sound, and enfeebled vesicular murmur. When extensive atelectasis is present, especially if superficial, there are signs of consolidation, namely, dulness, increased vocal fremitus, bronchial respiration, and bronchophony. If there are in the affected region lobules which are incompletely collapsed, the percussion sound has the tympanitic quality and there is persistent fine crepitus. The clinical picture is usually, however, dominated by the symptoms of the primary affection. A condition showing the above symptom-complex which is transitory, that is, which passes off in the course of twenty-four hours, is from this very fact almost sure to be due to atelectasis.

(c) BRONCHOPNEUMONIA.

Lobular Pneumonia; Catarrhal Pneumonia; So-called Capillary Bronchitis.

Definition.—Inflammation of capillary or terminal bronchi and the air-vesicles which constitute the corresponding pulmonary lobules. There are several forms, all of which are caused by bacterial invasion of the lungs. The disease may be primary or secondary and is characterized clinically by symptoms of infection and interference with the respiratory function.

Etiology.—PREDISPOSING INFLUENCES.—Bronchopneumonia is common at the extremes of life. In children the relatively small size of the bronchi, their more abundant vascular supply, and the more rapid and exuberant growth of the epithelium of the bronchial mucosa constitute predisposing factors of great importance; while in the aged tissue relaxation, tendencies to passive congestion, and diminished reflex excitability on the part of the bronchial mucous membrane act in the same way. Bronchopneumonia is more prevalent in the winter and spring than at other seasons of the year. It is more common among the poorer classes. In the great majority of the cases bronchopneumonia occurs as a secondary or intercurrent process. There are two principal groups of cases: 1. Those in which it arises in the course of simple bronchitis or an acute infectious disease in which bronchitis forms part of the symptom-complex, as measles, pertussis, diphtheria, scarlet fever, influenza, and less frequently the variolous diseases, erysipelas, and enteric fever. It is a common complication of the acute intestinal diseases of infancy. Bronchopneumonia is a grave complication in all these affections and constitutes the cause of death in the majority of fatal cases. Its frequency corresponds closely to their epidemic prevalence. It is far less common in the acute febrile diseases of middle life. In the aged it is a common complication and frequently forms the terminal event in various acute and chronic diseases. The consolidating lesions of pulmonary tuberculosis are due to chronic localized tuberculous bronchopneumonia. 2. Aspiration or deglutition pneumonia. In the stupor of the low fevers and in comatose states of all kinds the reflex excitability of the larynx is lowered, and the secretions of the mouth, minute portions of drink, and particles of food during the act of swallowing are drawn into the trachea and bronchi. In this situation they set up an active bronchitis which by extension rapidly involves the smaller bronchi and gives rise to an intense bronchopneumonia. This accident is very common after operations upon the throat, nose, larynx, and trachea. Most cases of ether pneumonia arise in this way. The infecting material may come from within the chest itself; severe bronchopneumonia not rarely follows hæmoptysis and occasionally the aspiration of the contents of a bronchiectatic cavity, or pus from an empyema which has found its way into the lung by way of a bronchopulmonary fistula is followed by this disease. The extension of tuberculosis from one part of a lung to another, or to the opposite lung, is due in some instances to aspiration.

EXCITING CAUSE.—Various micro-organisms are associated with the lesions of bronchopneumonia. In the primary form which attacks young children in previous health the common organism is the pneumococcus, which may be found in pure culture. In the secondary forms the streptococcus is the common infecting agent, but mixed infections are usual. The organisms present are the Bacillus pneumoniæ of Friedländer. Streptococcus pyogenes, Staphylococcus albus et aureus. The Klebs–Löffler bacillus is frequently found in the bronchopneumonia of diphtheria. and Pfeiffer's bacillus in influenza pneumonias.

The lesions consist in interstitial inflammation of the bronchi and alveolar walls. The small bronchi are plugged with exudate composed of leucocytes and swollen epithelium; their walls are swollen. infiltrated with cells, and traversed by distended capillaries. and there is dense peribronchial infiltration. The vesicles are filled with leucocytes and swollen epithelium, and rarely show the dense accumulations of red corpuscles and the fibrillated fibrin seen in croupous pneumonia.

The pathological unit is the inflamed pulmonary lobule. Hence the descriptive term lobular pneumonia. According to the distribution of the lesions three anatomical forms exist: 1. There is more or less intense bronchitis extending to the finer tubes, without the gross evidences of lobular consolidation, but with the microscopic findings of inflammation. This form is bilateral. 2. The foci of inflammation are scattered throughout the lung tissue, with lobular collapse and infiltrated lobules felt as hard nodules. These patches of bronchopneumonia are sometimes isolated with strands of congested or uninflamed tissue intervening and areas of collateral emphysema, and sometimes massed in groups of considerable size. This form is also bilateral. 3. The greater part of a lobe is often involved—the pseudolobar form of bronchopneumonia. Even in this form the consolidation is not uniform as in croupous pneumonia, but there are more or less extensive tracts of deeply congested but still crepitant tissue scattered among the inflamed lobules.

The terminations are in resolution, suppuration or gangrene, or fibrosis. In the tuberculous forms, which are very common in previously apparently healthy children, bronchopneumonia terminates in caseation or chronic fibroid changes. Such cases often follow measles, pertussis, or diphtheria and may be the result of the lighting up of a latent tuberculosis or of tuberculous infection at the time.

Symptoms.—The onset of the primary form is abrupt. It is marked by convulsions and sudden rise of temperature. Cerebral symptoms are frequent and often intense. The defervescence may be critical. These cases, which run a course analogous to that of croupous pneumonia and present similar symptoms and localized physical signs, when they terminate fatally often show pseudolobar consolidation and the pneumococcus in the lesions.

The secondary forms are preceded by the signs of a bronchitis extending to the smaller tubes. The disease develops gradually without chill or convulsion. The fever is of variable intensity—102°–104° F. (39°–40° C.) —and does not conform to type, being irregularly remittent. The skin is hot and dry. There is cough, which is hard, dry, and distressing. The respiration frequency may reach 60 to 80 per minute, and cyanosis soon appears. Other evidences of deficient oxygenation of the blood are frequent, small, thready pulse, stupor, restlessness, and occasional convulsive tremor. The cough becomes less urgent, the child no longer struggles for air, the face becomes suffused and loses its anxious expression, and death occurs from cardiac paralysis. Sputum (see Part III, page 457).

Physical Signs.—Upon *inspection* there are early signs of obstruction in the terminal bronchi, inspiratory retraction of the intercostal spaces and lower sternal and epigastric regions, rapid, shallow, jerky respiration, and cyanosis of the lips and finger-tips. *Percussion.*—Death may occur before signs of consolidation develop. There may be merely vesiculotympanitic resonance. If consolidation is present impaired resonance, sometimes actual dulness, is found at the bases in scattered areas. To elicit it in the latter case demands very careful light percussion. Compensatory emphysema may mask small areas of dulness. *Auscultation* in the early stages reveals only the signs of the extension of the bronchitis to the finer tubes, namely, many diffuse small mucous and some crepitant râles. These are usually more intense and numerous at the bases posteriorly. Vesiculobronchial and, here and there, pure bronchial respiration may be heard over the patches of dulness. Failure of the right heart is followed by pulmonary œdema, which is usually the forerunner of the death agony.

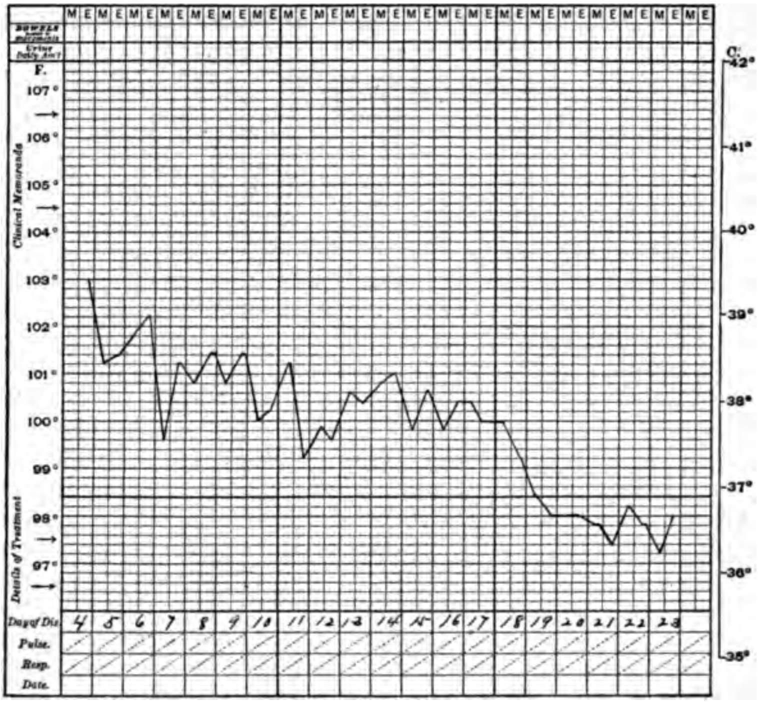

FIG. 316.—Bronchopneumonia in a man aged seventy-six.

Clinical Varieties. — 1. PRIMARY FORM. — It is a question whether many of these cases are not in truth irregular forms of croupous pneumonia—pneumococcus pneumonia. The primary form is rare in adults.

2. Masked Forms.—The actual condition may in infants be masked by cerebral symptoms such as also occur in croupous pneumonia at this period of life, namely, convulsions, drowsiness, retraction of the muscles of the back of the neck, and stupor; or by gastro-intestinal symptoms, such as nausea, vomiting, and looseness of the bowels. 3. Suffocative Catarrh.—The overwhelming cases were so designated by the earlier writers. The ordinary acute cases of this group, fatal in the course of two or three days, are most appropriately described under this term. 4. Secondary Forms.—Mild secondary bronchopneumonia may follow

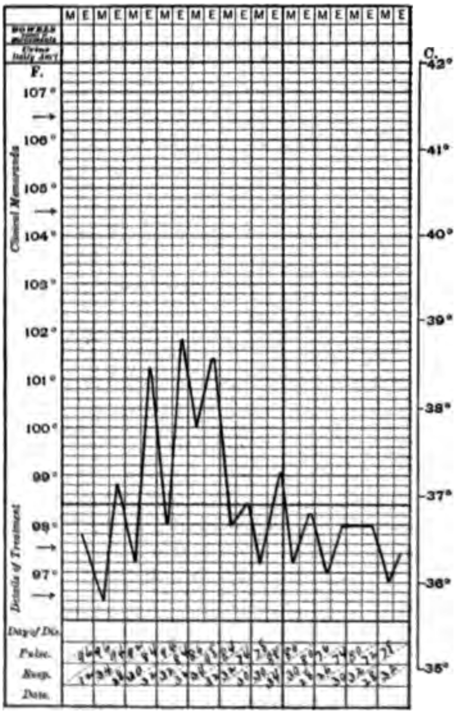

Fig. 317.—Bronchopneumonia occurring in the course of chronic nephritis.

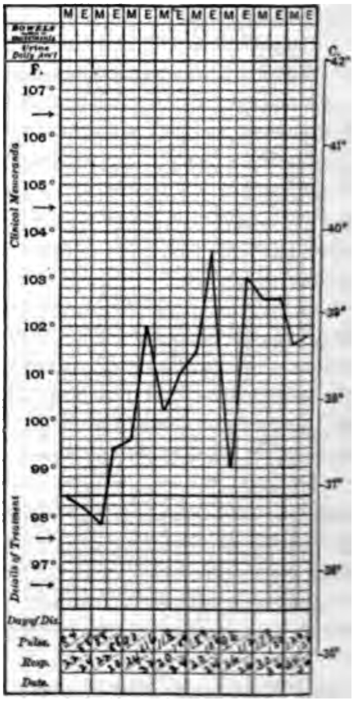

Fig. 318.—Fatal bronchopneumonia. Man aged seventy-six.

severe forms of bronchitis, both in children and adults. This form assumes great importance in diagnosis, since it may be merely a simple pathological condition, on the one hand, or an insidiously developing tuberculous bronchopneumonia, upon the other. 5. Ether Pneumonia; Postoperative Pneumonia.—This form of bronchopneumonia usually is pseudolobar and presents close resemblances to croupous pneumonia. The anæsthesia bronchitis which precedes the pneumonic symptoms, the low temperature range, the absence of bloody sputum, and the course of the attack are significant. In a majority of the cases it is an inhalation pneumonia.

Course and Duration.—Bronchopneumonia is not a self-limited disease. The primary cases, which suggest croupous pneumonia, run a short course and frequently terminate by crisis. The secondary cases are often prolonged and the defervescence is by lysis. Fatal cases in children often come to an end in from 3 to 5 days. The duration of favorable cases is from 1 to 4 or 5 weeks. Recovery may still take place after an illness of 8 or 10 weeks. In protracted cases tuberculosis is to be feared.

Diagnosis. — DIRECT. — A positive diagnosis cannot be made unless the signs of consolidation are present. Circumscribed areas of relative dulness, usually bilateral with vesiculotympanitic resonance interspersed, together with high-pitched, small mucous and crepitant râles, bronchial breathing, bronchophony, and increased vocal fremitus are diagnostic in the primary as well as in the secondary forms. In cases in which the lesions are massed in a single lobe, careful physical examination will almost always show a focus of consolidation upon the opposite side. The signs of consolidation are often absent, especially early in the attack. The diagnosis is even then probable if in bronchitis the temperature rises to 104° F. (40° C.), the cough becomes short, harassing, and painful, the respiration and pulse-frequency become high, cyanosis develops, and diffuse, high-pitched fine râles are heard.

DIFFERENTIAL.—*Croupous Pneumonia* (see page 728). *Acute Miliary Tuberculosis* (see page 790). *Acute Tuberculous Bronchopneumonia* (see page 804). *Atelectasis.*—The dulness and râles of this condition are modified and sometimes even disappear upon change of posture. Fever is not an essential concomitant. If present it is usually due to an associated bronchitis. *Meningitis.*—In infants marked cerebral symptoms sometimes occur, and the condition closely simulates meningitis. The differential diagnosis between tuberculous meningitis and bronchopneumonia with cerebral symptoms can in some cases only be made by time.

Prognosis.—The outlook is much more favorable in the primary than in the secondary forms. It is greatly influenced by the age of the patient. Within the first year almost every case dies; until the fifth year from 30 to 50 per cent.; in the aged the prognosis is ominous. Pale, fat, flabby children do not bear the disease well. Aspiration pneumonia is a very fatal disease, especially that form which follows operations upon the mouth and throat.

iii. Diseases of the Lungs Characterized by Interstitial Inflammation.

(a) CHRONIC INTERSTITIAL PNEUMONIA.

Cirrhosis of the Lung.

Definition.—Chronic inflammation of the lung with increase of the interstitial tissue and decrease in the vesicular tissue, with general induration and contraction. There are two forms, the local or circumscribed, and the diffuse, involving the greater part of one or both lungs.

1. LOCAL PULMONARY FIBROSIS.—This condition is very common. It occurs as a secondary process in inflammatory diseases of the lung, as bronchitis, croupous pneumonia, and bronchopneumonia. It constitutes an important feature in the complex lesions of tuberculosis and the chief final local process in obsolescent or cured tuberculous disease. It is the

termination of pulmonary atelectasis prolonged through the stages of compression and carnification until the tissue has become organized and expansion is no longer possible. Hence it is a feature of the lung tissue adjacent to aneurisms, tumors, abscesses, hydatids, and gummata. It is especially important as the outcome of neglected pleural effusion, because by the early withdrawal of the exudate the pressure atelectasis may be relieved and subsequent fibroid changes minimized. The clinical phenomena of local fibroid changes are subordinate to those of the primary disease which it accompanies. Retraction of the supra- or infraclavicular spaces, or circumscribed retraction elsewhere, is suggestive. Nevertheless the diagnosis must often be provisional rather than positive. The chief importance of the condition arises from its often constituting the point of departure for fibroid changes involving the entire lung.

2. Diffuse Pulmonary Fibrosis.—To a certain extent the etiological factors are the same as in the localized form. Thus the fibroid changes in chronic bronchitis and emphysema, and associated with bronchiectasis, are usually diffuse. When the condition follows pressure atelectasis it may be diffuse when the greater part of a lobe or lung is compromised. Other conditions which may be followed by chronic interstitial pneumonia are the irritation of a foreign body long retained in a bronchus, chronic bronchopneumonia, in rare instances unresolved croupous pneumonia, pleurisy, the connective-tissue overgrowth invading the lung from a chronic fibrinous pleural exudate—the pleurogenous form of chronic interstitial pneumonia—and syphilis.

Two forms are recognized. (a) *Lobar Interstitial Pneumonia.*—The disease is unilateral. The entire lung is usually involved. It frequently appears as a small, dense, airless mass of tissue lying along the spine. It is densely indurated, showing upon section a compact, grayish, fibroid tissue traversed by the bronchi and blood-vessels. In the pleurogenous form the lung is bound to the wall of the thorax by dense, thick, pleural adhesions. When the process has been intrapulmonary from the beginning the pleural adhesions may be of only moderate thickness. Bronchiectasis is common and often very extensive. In tuberculous cases vomicæ and caseating foci are present and the opposite lung shows evidences of tubercle. The right heart is hypertrophied, and atheroma and local arteriosclerosis are frequently encountered. The heart is displaced toward the affected side. The opposite lung is emphysematous—*vicarious emphysema*—and its border extends beyond the median line toward the affected side. (b) *Disseminated Interstitial Pneumonia.*—The condition is bilateral. There are circumscribed areas of fibrosis separated by lung tissue more or less emphysematous—*collateral vicarious emphysema.* These fibroid islets are deeply situated in the lung tissue and more frequently in the lower lobes. They surround bronchiectatic dilatations.

Symptoms.—Fever when present is due to an acute associated process or to tuberculosis. There are cough, dyspnœa upon exertion, and expectoration, which may show the special characteristics of the sputum of bronchiectasis. Hæmoptysis is fairly common, especially in the tuberculous cases. The affection is the very type of a chronic disease but the adaptations are remarkable. The general health is often remarkably good when the extent and nature of the lesions are considered, and the patients are capable of conducting their affairs.

Physical Signs.—Inspection.—The deformity of the chest in well-developed cases is conspicuous both in the unilateral and bilateral types of the affection, though in the latter retraction is more or less modified by compensatory emphysema. In the unilateral variety the affected side of the thorax is retracted and immobile. The ribs approach each other so that the intercostal spaces are obliterated, or the ribs may overlap. The shoulder droops and there is lateral curvature of the spine, the concavity in the dorsal region being toward the affected side. The respiratory muscles and those of the shoulder and arm are wasted. The semicircumference of the affected side is much diminished and remains uninfluenced by respiratory effort. The heart is strongly displaced toward the affected side, being drawn over by the contracting lung. The changes in the cardiac phenomena are much more apparent when the left lung is involved. Under these circumstances there may be an extensive area of pulsation in the second, third, and fourth interspaces to the left of the sternal border. When the right lung is affected the cardiac impulse may be wholly obliterated by the extending median border of the emphysematous left lung. The unaffected side is in strong contrast with its large size, wide intercostal spaces, and free respiratory excursus. Palpation.—If the bronchi are unobstructed the vocal fremitus is increased over the affected side. There is usually epigastric pulsation transmitted from the right heart. Cardiac pulsation is well felt in the second, third, and fourth interspaces in left-sided fibrosis, and in some cases there is a short diastolic thrill to the left of the pulmonary area. Percussion.—Dulness is usually marked and may be extreme at the apex and base. When the bronchi are extensively dilated the percussion sound has the tympanitic quality. This may be especially marked in the axillary region. Over the opposite lung there is hyperresonance. Upon linear percussion the anterior border of the unaffected lung is found to be displaced as far as, or even beyond, the sternal border on the affected side, and its lower border displaced downward, while the lower border of the affected lung and the related organ, *i.e.*, liver or spleen, are displaced upward and show no respiratory movement. Auscultation.—There is more or less widely extended bronchial respiration. At the apex the quality may be cavernous or amphoric; at the base feeble and distant. In some cases small mucous râles are heard. There is bronchophony and, if bronchial dilatation extends to the periphery of the lung, whispering pectoriloquy may be heard. The pulmonary second sound is accentuated and endocardial murmurs frequently appear toward the close of the disease as dilatation and failure of the right heart come on.

Diagnosis.—In the disseminated form the diagnosis may be obscure. The symptoms and signs may be simply those of bronchiectasis. In the unilateral form the diagnosis is unattended with difficulty. The underlying condition cannot, however, always be determined. The resemblance to fibroid phthisis is often very close. Signs of disease in the opposite lung, especially at the apex, fever, hæmoptysis, and foci of moist râles are in favor of a diagnosis of tuberculosis. Tubercle bacilli in the sputum are conclusive.

Prognosis.—The outlook as regards life is favorable; as regards recovery hopeless. The patients live many years and often have no great inconvenience except from cough, expectoration, and dyspnœa upon exertion. The powers of resistance are diminished and they readily succumb to intercurrent disease. Otherwise death is commonly due to progressive failure of the right heart or amyloid disease.

(b) PNEUMONOCONIOSIS.

Definition.—Disseminated fibrosis of the lungs caused by the habitual inhalation of a dust-laden atmosphere in various occupations.

Etiology.—Pneumonoconiosis is the very type of an occupation disease. Several varieties are described, according to the character of the work and the nature of the dust inhaled: *anthracosis* or coal miners' disease; *siderosis*, the form caused by inhaling metallic dust, especially iron oxide, and brass and bronze particles; *chalicosis*, due to mineral dusts, as stonecutters' consumption or the grinders' rot of the workers in cutlery. Similar affections occur in workers in flax and cotton and in grain shovellers.

The condition of the lungs in advanced cases of the disease caused by different substances is practically the same. The interstitial inflammation starts from the peribronchial lymph-nodes in which the dust particles excite proliferation of connective tissue, and in the early stages of the process, especially in anthracosis, is confined to these tissues. There is an associated chronic bronchitis to which many of the symptoms are due. Bronchiectasis is common and in a majority of the cases the clinical picture is that of a chronic bronchitis with emphysema. In anthracosis there is a carbon-laden black spit. In advanced cases softening occurs in the indurated nodules, and small cavities are formed which in some cases suppurate and discharge a purulent fluid by way of the bronchi. Notwithstanding the prevalence of anthracosis among coal miners tuberculous phthisis is comparatively rare.

Symptoms.—The disease does not show itself until after long exposure to the dust. There are the general signs of failing health, with cough, expectoration, often abundant, dyspnœa, and wheezing, especially upon exertion. The mucopurulent sputum in anthracosis is blackish. It is popularly known as "black spit." That of the other forms is light or grayish in color, without gross characteristics. In chalicosis glittering crystalloid particles of silicious material may be seen. Under the microscope the dust particles are seen in the alveolar epithelium. Tuberculosis may form the terminal condition.

Diagnosis.—The direct diagnosis is not as a rule difficult. The anamnesis, the gradual development of the affection after years of exposure, the symptoms of chronic bronchitis with emphysema and bronchiectasis, the mucopurulent sputum with the special characteristics mentioned, and the absence of tubercle bacilli are all of diagnostic importance.

Prognosis.—The outlook is favorable if the condition is recognized early and the patient can change his work. Otherwise the cases run a progressive though very chronic course.

iv. Diseases of the Lungs due to Suppuration and Necrosis.

(a) PULMONARY ABSCESS.

Definition. — Localized collections of pus in cavities formed by the disintegration of lung tissue.

Etiology. — Pulmonary abscess is a secondary process due to the intense action of various pyogenic organisms, among which streptococci and staphylococci are the most common. It may occur under the following conditions:

1. Acute Inflammation.—Suppuration may follow croupous pneumonia. It occurs in two forms: first, the purulent infiltration which constitutes an advanced stage of gray hepatization, and second, the much more rare condition of actual abscess cavity. The latter are usually small, multiple, with shreddy walls, and frequently contain necrotic tissue. They tend by fusion to form larger abscesses. Purulent infiltration and abscess are sometimes present in the same lung. Abscess is more common in bronchopneumonia, especially the form known as aspiration or deglutition pneumonia. In the low fevers and stuporous and comatose conditions, after wounds of the neck and operations upon the nose, throat, and mouth, and suppurative diseases of these parts, an intense acute bronchitis frequently arises, which by extension involves the distant tubules and causes purulent bronchopneumonia. Multiple abscesses, mostly minute but frequently attaining the size of an orange, may result. A similar condition may follow the inflammation caused by a foreign body lodged in a bronchus. Pulmonary abscess is a relatively common sequel of epidemic influenza. 2. Traumatism.—Perforation of the lung from without, as in stab or gunshot wounds, laceration of the lung by a fractured rib, and analogous accidents, may cause pulmonary abscess. 3. Perforation from Within.—Sudden invasion of the lung by purulent or otherwise infected substances from adjacent organs is a common cause of abscess. Cancer of the œsophagus, abscess of the liver, a suppurating hydatid cyst, or the aspiration of the pus in empyema suddenly rupturing into the lung may cause abscess. 4. Infective Emboli.—Metastatic abscesses in the lung are common in septic—pyæmic—states. The purulent foci are multiple, mostly subpleural, and at first wedge-shaped. They are commonly small, but occasionally extensive purulent infection occurs. The related pleura is inflamed and covered with a thick, greenish lymph. Occasionally softening and perforation are followed by pneumothorax. 5. Tuberculous Abscesses.—Circumscribed local suppurative processes enter largely into the complex lesions of pulmonary tuberculosis, especially in the later course of the disease. They are associated with caseation and cavity formation, and give rise to important and significant symptoms and physical signs.

Symptoms.—As in other suppurative processes there are irregular chills and fever. In pneumonia the general symptoms are aggravated, the sputum becomes purulent, and the signs of a cavity can sometimes be demonstrated. In pyæmia the local symptoms of pulmonary abscess are obscured by the general symptoms of sepsis, and the condition is usually overlooked. Aside from the presence of pus in large amounts the sputum presents characters of diagnostic importance (see Part III, page 457).

Physical Signs.—The signs of a cavity which empties itself under the stress of violent paroxysms of cough, and again refills, may be elicited upon physical examination when the abscess is of sufficient size and situated in the periphery of the lung. In small abscesses the signs are often wholly inconclusive.

Diagnosis.—DIRECT.—When the above symptoms and signs are present, especially when the etiological factors essential to diagnosis can be established and purulent sputum containing elastic fibres and the other characters named accompanies the cough, the diagnosis is positive. In many cases, however, it is at best provisional, and not rarely abscess in the lungs not suspected during life is found upon the post-mortem table.

DIFFERENTIAL.—*Collections of Pus Perforating into the Lung.*—Accumulations that may break into the lung occur in empyema, subphrenic abscess, hepatic abscess, suppurating hydatid cyst, in spinal caries, and other suppurative processes in adjacent viscera. The differential diagnosis rests upon the presence of symptoms and signs significant of such pathological conditions, and especially upon the absence of the elastic fibres from the expectorated pus. *Bronchiectasis.*—The etiological factors are different. Both are secondary affections, but bronchiectasis is a chronic, pulmonary abscess an acute, affection. The cough is more urgent and less constant in bronchiectasis, and the sputum more foul and does not as a rule contain elastic fibres, which, when present, are less abundant than in abscess. *Pulmonary Cavities in Tuberculosis.*—Etiological considerations are important. The slow development of phthisical cavities, their antecedent and concomitant phenomena, the characters of the sputum, and the presence of tubercle bacilli are of positive diagnostic significance. *Pulmonary Gangrene.*—The sputum in gangrene of the lung has an extremely intense, putrid odor not often present in the purulent expectoration of pulmonary abscess, and contains shreds of decomposing lung tissue, which can be readily detected when it is spread out upon a glass plate. These shreds frequently present the structure of the pulmonary alveoli.

(b) GANGRENE OF THE LUNG.

Definition.—Decomposition of lung tissue caused by the action of the bacteria of putrefaction. It may be diffuse or circumscribed.

Etiology.—Sphacelus of the lung occurs as an anatomical condition under a variety of circumstances. Impaired vitality of the tissues from general or local causes is a necessary predisposing factor. The subjects are commonly greatly debilitated by long-continued chronic or grave acute disease. Severe general disturbances of nutrition, such as are caused by prolonged infections or disease of the bones, or arise in malignant disease or diabetes mellitus, constitute predisposing influences of importance. Equally important are bronchiectasis, especially that form of bronchiectasis which occurs as the result of the pressure of a tumor or aneurism, tuberculous cavities and putrid bronchitis, since the putrid contents of the cavities or bronchi often cause secondary gangrene when aspirated into the adjacent tissues. Among the acute diseases in which gangrene of the lung occurs are croupous pneumonia, particularly when it affects debilitated or diabetic persons; aspiration bronchopneumonia, whether the infectious material be derived from the nose or throat of the patient, or from some focus of disease adjacent to the lung, as cancer of the œsophagus, abscess of the liver, or empyema; embolism of the pulmonary artery, especially when the embolus is infected, as in pyæmia or necrosis of the bones, and in enteric fever thrombosis of one of the principal branches of the pulmonary artery. No satisfactory explanation of the occasional occurrence of gangrene under these circumstances, when it remains absent in the majority of similar cases, has been advanced.

1. Diffuse Pulmonary Gangrene.—This form is very rare. It has been observed after croupous pneumonia and in the most intense cases of aspiration pneumonia. It may also occur after the occlusion of a large branch of the pulmonary artery by an embolus or thrombus. A large portion or the whole of a lobe may undergo putrefaction in the course of a day or two.

2. The Circumscribed Form.—The gangrenous portions of lung are surrounded by an intensely congested and œdematous border of tissue. The lesions may be single or multiple. They are more commonly situated in the lower lobes than the upper and in the peripheral than in the central parts of the lung. The greenish-black gangrenous tissue rapidly softens, with the formation of an irregular cavity with shreddy walls and an abundant, horribly offensive fluid.

Symptoms.—The expectoration is profuse, thin, and green or brownish in color. Its odor is exceedingly fetid, disgusting, and penetrating. In some instances it is mawkishly sweet. It is not only present upon the breath but also pervades the atmosphere of the patient's room (see Part III, p. 457). The symptoms of the primary affection and constitutional debility usually precede the characteristic phenomena of gangrene of the lung. There is commonly a moderate fever associated with rapid pulse and great general depression. Hemorrhage is not uncommon and may prove fatal.

Physical Signs.—The signs in the diffuse form are those of infiltration, impaired resonance, with the tympanitic quality, and vesiculobronchial or bronchial respiration. In the circumscribed form the signs of a cavity predominate. Very often in limited gangrene the signs may elude detection. Severe bronchitis is common.

Diagnosis.—Direct.—The sputum and the odor of the breath are characteristic. The presence of shreds of gangrenous alveolar tissue is conclusive.

Differential.—From putrid bronchitis and bronchiectasis gangrene of the lung cannot always be differentiated, except by the presence of lung tissue in the sputum, and when this is found in old standing cases it constitutes positive evidence of secondary gangrene.

Prognosis.—Pulmonary gangrene is almost always a terminal event. The patient sinks rapidly and dies from exhaustion. When the gangrenous area can be localized, and the condition of the patient is favorable, a surgical operation may be the means of saving life. In rare cases of circumscribed gangrene encapsulation has taken place, and still more rarely recovery has occurred after the expectoration of pieces of gangrenous lung of considerable size.

v. New Growths in the Lungs.

Neoplasms in the lungs are usually malignant. They are primary or secondary. The former are rare; the latter comparatively common.

1. Primary Tumors.—Carcinoma is the usual form. Much more rare are primary sarcoma and endothelioma. The growth usually invades one lung and forms a large mass, which may ultimately break down and give rise to a cavity. Diffuse cancerous infiltration may simulate tuberculous bronchopneumonia. Diffuse miliary infiltration has been described *carcinosis pulmonum miliaris*.

2. Secondary New Growths.—Every variety of malignant growth may by metastasis invade the lungs. In comparatively rare instances the secondary growth may be solitary, and chiefly involves the pleura. Usually they are multiple and occupy both lungs. Large tracts of pulmonary tissue may be densely invaded. The primary tumor is usually mammary, in the gastro-intestinal or genito-urinary tract, or in the bone. It may be epithelioma, scirrhus, colloid, sarcoma, enchondroma, or osteoma. In melanosarcoma the primary growth may have its starting-point in a pigmented mole. In Hodgkin's disease the growth may perforate the sternum and widely involve the lungs, or it may reach the lungs by way of the tracheal and bronchial lymph-nodes. In cancerous disease there is commonly secondary implication of the tracheal and bronchial glands, and less often of the cervical chains. Other superficial glands, including the inguinal, may be enlarged. Pleurisy is common. It may be cancerous or serofibrinous. Frequently both conditions are present. Not rarely the effusion is hemorrhagic.

Etiology. New growths in the lungs occur with greatest frequency in middle life. Males suffer more commonly from primary malignant disease of the lungs and pleura; women from the secondary form.

Symptoms. The clinical picture is not well defined, especially in the primary form. It sometimes suggests chronic pneumonia, sometimes a mediastinal tumor or thoracic aneurism, or again there may be nothing to suggest an affection of the lungs. Pain is usually present when the pleura is affected. It may be substernal. Cough is not constant. It is frequently aggravated in certain postures and may be dry and attended with pain. There is in many cases a jelly-like bloody sputum which is highly suggestive, though it may occur in other conditions. Dyspnœa upon exertion is a symptom of large growths or extensive infiltration, and may be paroxysmal. Pleural effusion, often found upon aspiration to be hemorrhagic, may develop. The lymph-nodes of the axilla or above the inner end of the clavicle are frequently enlarged. There is progressive wasting and cachexia, and toward the end irregular fever of remittent type. Pressure symptoms are common. These consist of distention of the large veins, with cyanosis of the face and one or both arms, enlarged and tortuous veins over the upper part of the chest, distressing dyspnœa or stridor from compression of the trachea or large bronchi, brassy cough, and aphonia from pressure upon recurrent laryngeal nerves. In large unilateral tumors the heart is displaced toward the opposite side and the diaphragm depressed.

Physical Signs.—The signs may be due to the tumor itself or to an accompanying pleural effusion. In the latter case the affected side is enlarged, the intercostal depressions obliterated, the respiratory movement restricted. The vocal fremitus is diminished. Upon percussion and auscultation the signs are modified by the presence of fluid and its amount, which is not usually great. Even when it is considerable, the tumor affects its distribution and upper lines. There is dulness which may be complete at the base. The breath sounds are feeble and distant. There may be well-characterized bronchial breathing.

Diagnosis.—Direct.—In primary cases the diagnosis may be difficult or impossible. Important criteria are strictly unilateral phenomena, irregular character and distribution of the physical signs, dark, jelly-like, mucoid, bloody expectoration, tendency to cachexia, and implication of superficial lymph-nodes. If carcinomatous tissue elements are found in the sputum or the growth perforates the chest wall—very rare events—the diagnosis is positive. The X-ray examination is important. In a case of single large growth in the lower lobe of the right lung, with multiple pigmented cutaneous lesions, occurring in a man aged sixty-four, the results of physical examination were fully confirmed. In the secondary form a probable diagnosis may be made with some confidence when the above described pulmonary symptoms arise in the course of several months after the recognition of a primary malignant tumor, as of the breast, womb, stomach, or bowel, with or without operation.

Differential.—The recognition of a mediastinal new growth or an aneurism of the aorta, in contradistinction from new growths in the lungs, may involve serious diagnostic difficulties.

Prognosis.—The course of malignant growths in the lungs is lethal, the end occurring within a year or two, and not rarely within a few months after the appearance of the symptoms.

V. DISEASES OF THE MEDIASTINUM.

Enlarged lymphatic glands, suppurative lymphadenitis, abscess, interstitial emphysema, chronic indurative mediastinitis, and new growths are to be considered.

1. **Enlargement of the lymphatic glands** in the mediastinum accompanies inflammation of the bronchi, bronchopneumonia and the specific infections, croupous pneumonia, measles, pertussis, and tuberculosis. This constant anatomical change does not commonly attain sufficient dimensions to cause definite symptoms or physical signs; exceptionally pressure upon the trachea may cause a paroxysmal, brassy cough, and dulness over the manubrium sterni, in the upper part of the interscapular region, or a modification of the sound upon direct percussion over the upper dorsal spines.

2. **Suppurative Lymphadenitis.**—Suppuration may occur in the tracheal or bronchial lymphatic glands as the result of ordinary inflammation. More commonly it is of tuberculous origin. The symptoms are obscure. Perforation into a bronchus, the œsophagus, or the aorta may occur. In other cases the fluid contents of the glandular abscess are absorbed, lime salts are deposited, and the condition becomes one of anatomical rather than clinical interest.

3. **Mediastinal Abscess.**—The abscess cavity usually occupies the anterior mediastinum. The condition may be acute or chronic. The acute cases are due to traumatism or occur in connection with the acute febrile infections; the chronic cases are as a rule tuberculous. Males are more commonly affected. The pus shows a disposition to burrow and may find its way through an intercostal space, into the abdomen, may rupture into the trachea or œsophagus, or may perforate the sternum.

Symptoms.—Substernal pain is constant. In the acute cases it is severe and throbbing, and accompanied with chills, fever, and sweating. Large abscesses may give rise to pressure symptoms and dyspnœa. There may be resorption of the fluid and inspissation.

Physical Signs.—A fluctuating tumor may appear in the episternal space or at the sternal border. In the latter situation it may suggest empyema necessitatis. Pulsating synchronously with the heart it may simulate a pulsating empyema or an aneurism. From pulsating empyema it may be distinguished by the absence of the signs of pleural effusion; from aneurism by absence of murmur, of diastolic shock, and of the expansile character in the pulsation. Exploratory puncture with a fine needle may be performed.

4. **Emphysema.**—The escape of air into the connective-tissue spaces of the mediastinum is an occasional event after traumatism or surgical operations upon the neck, as tracheotomy, and in pertussis. It may be associated with pneumothorax. There are no special symptoms. Crackling râles having the rhythm of the heart are heard over the sternal region. If the air finds its way into the subcutaneous tissue of the neck the crepitus may be recognized upon kneading the tissues lightly with the finger-tips. Etiological factors are important.

5. **Indurative Mediastinitis.**—Adherent pericardium with chronic proliferative mediastinitis is a rare condition. The heart is greatly enlarged. Its action is hampered by the extensive adhesions to the adjacent parts; its signs obscured by the greatly thickened fibrous tissue of the mediastinum. Friction sounds may sometimes be heard along the sternal borders. The nutrition of the heart muscle ultimately fails, and dyspnœa, cyanosis, and anasarca develop.

6. **Mediastinal New Growths.**—The common varieties are carcinoma and sarcoma. Dermoid cysts, hydatid cysts, and lymphomata, fibromata, lipomata, enchondromata, and gummata are of comparatively infrequent occurrence. The tumor may have its origin in the thymus, the lymph-glands, or the pleura or lung. Tumors of the anterior mediastinum originate from the remnants of the thymus of the connective tissue; those of the middle and posterior mediastinum from the lymph-nodes. Primary tumors are more commonly sarcoma than carcinoma. Among personal predisposing influences age and sex are important. Mediastinal tumor most frequently develops in the fifth decade of life and in men.

Symptoms.—The important manifestations of a tumor developing in or encroaching upon the mediastinal spaces are displacement and pressure symptoms. These symptoms relate to the heart, great vessels and nerves, the trachea, bronchi, lungs, and pleuræ, and the œsophagus. The symptoms depend upon the size of the new growth, and its immediate location. A small tumor may not be the occasion of any derangement of function. On the other hand, as the growth increases, remarkable adaptations take place, and life may be maintained despite great compression and dislocation of the heart and lung. The pressure of the tumor exerted upon the wall of the heart interferes with diastole and diminishes the volume of blood thrown into the aorta with the ventricular systole. The pulse is therefore small and frequent. The dislocation of the organ is backward or downward in tumors of the anterior mediastinum, with displacement of the apex beat to the left. The liver or spleen is also displaced downward by large mediastinal tumors. There may be cardiac dyspnœa from pressure. The presence of the tumor interferes with respiration and produces of itself a slight degree of cyanosis, which is increased by pressure upon large venous trunks. When the intrathoracic portion of the inferior vena cava is affected, venous distention and œdema of the abdomen and lower extremities result; when the superior vena cava is compromised, swelling and œdema in the face and both upper extremities may occur, together with signs of interference with the cerebral circulation, such as headache, vertigo, and ringing in the ears. A collateral venous circulation may be established in either case with great distention of the superficial veins of the abdomen or thorax. Pressure upon and obstruction of the right or left innominate vein is more common. This hinderance to the return of the venous blood manifests itself by œdema of the face and arm of the corresponding side, and enlargement of the superficial veins of the thorax. The arterial trunks yield less readily to the presence of the tumor than the veins. When the pressure becomes so great as to interfere with the lumen of the subclavian or innominate, the pulse upon the affected side is enfeebled. Pressure upon the recurrent laryngeal nerves causes dyspnœa, aphonia, and the severe, brassy, paroxysmal cough often present. The laryngoscope should be used. Pressure upon the phrenic may cause hiccough and shallow respiration; upon the vagus asthmatic attacks and dysphagia in the absence of direct pressure upon the œsophagus, and bradycardia or tachycardia. Implication of the sympathetic may cause dilatation or contraction of the pupil upon the affected side—inequality of the pupils. Dyspnœa is an early and constant symptom. It is due to various factors, as pressure upon the heart, the recurrent laryngeal nerves, the trachea, the lungs themselves, or the presence of a pleural effusion. It is often slight when the patient is quiet, but severe and distressing upon exertion. The mechanical compression of considerable portions of a lung manifests itself in dyspnœa of inspiratory type with slow and deep respiratory movements. In extreme conditions of pressure there is orthopnœa. When the pressure is unilateral there may be inspiratory depression of the intercostal spaces. Areas of fibrinous pleurisy are attended with local pains and friction sounds. Compression of the œsophagus renders the act of swallowing difficult or in extreme cases impossible.

Physical Signs. — *Inspection.* — Orthopnœa, œdema, and cyanosis of the face, arm, and upper part of the chest, and varicose enlargement of the superficial mammary and epigastric veins are often seen. But in many cases these signs are absent. In old cases there may be clubbing of the finger-tips and incurvation of the nails. There may be bulging of the sternum. The new growth may even erode the bone or perforate the chest wall at the sternal border. The impulse of the heart may be displaced to the left and downward. Respiratory derangements are conspicuous. In some cases inspiratory retraction of the soft parts of one side denotes unilateral compression of a main bronchus or the large part of the lung. Diminished respiratory excursus upon one side may be a sign of pleural effusion. *Palpation.*—Vocal fremitus is absent over the tumor and over pleural effusions. If the tumor pulsates it lacks the forcible, expansile impulse of an aneurism. *Percussion.*—Dulness of a high grade and increased resistance are present over the tumor. The borders of the area of dulness are irregular, and do not correspond to the outline of the heart or of the margin of an infiltrated lung. The dull area is continuous with the dulness of a pleural effusion when the latter is present and its persistence after the withdrawal of the fluid is of diagnostic significance. *Auscultation.*—The respiratory murmur retains its vesicular quality, in the main, but is enfeebled. At the borders of the tumor it may, owing to pressure atelectasis, have the bronchial quality. Stridor is the sign of compression of the trachea or a main bronchus.

Diagnosis.—Direct.—An area of dulness in the sternal region, with irregular and advancing borders, bulging of the breast-bone, dyspnœa of inspiratory type, absence of respiratory murmur, displacement of the heart and of the liver or spleen, signs of obstruction of the venous circulation, of pressure upon the vagi, the sympathetic, the phrenic, and the recurrent laryngeal nerves, the trachea, bronchi, or the lung itself, and dysphagia constitute a symptom-complex upon which the diagnosis of mediastinal tumor may be confidently made. The association of a number of them justifies a provisional diagnosis. When, however, to several of these signs are added the presence of enlarged superficial lymph-nodes and the visible and palpable evidences of a tumor perforating the sternum or the chest wall at the borders of the sternum, or advancing into the episternal notch, the diagnosis becomes positive.

Differential.—Obscure pressure symptoms may arise in consequence of the presence of a small mediastinal tumor, tuberculous tracheal or bronchial glands, gummata or syphilitic cicatrices, or a small aneurism. The differential diagnosis of these conditions cannot be made nor can a positive diagnosis of mediastinal tumor be reached until abnormal dulness associated with distinct evidences of intrathoracic pressure appear. With reference to the differential diagnosis, pericardial and pleural effusions, malignant disease of the pleura, and aneurism of the aorta demand special consideration. *Pericardial Effusion.*—Fluid pericardial and pleural exudates progressively displace the organs contained in the mediastinum; mediastinal new growths compress them. A flat percussion sound is the sign of both conditions; but the area of flatness in uncomplicated effusion into the serous sacs has definite and regular outlines, whereas in mediastinal tumor its borders are irregular and anomalous. In pericardial effusion the apex beat may be faintly palpable within the dull area and more distinct when the patient bends forward. In this attitude the dulness is also slightly increased in its transverse diameter. In mediastinal tumor, on the other hand, the cardiac impulse is at the left border of the dull area, which is not affected by change of posture. In cases in which the tumor extends between the apex of the heart and the wall of the chest no impulse can be detected. *Pleural Effusion.*—This condition is frequently associated with mediastinal tumor. Under these circumstances the physical signs are anomalous. The persistence of dulness of irregular outline and compression symptoms after the withdrawal of the fluid are significant. Dyspnœa upon exertion, and dislocation of adjacent organs, unilateral prominence of the chest wall with diminished respiratory excursus upon the same side, point to pleural effusion. In tumor the vocal fremitus is more commonly preserved, signs of pressure upon the recurrent are more frequent and more marked, and dysphagia is often present. An exploratory puncture will at once clear up any uncertainty. *Malignant Disease of the Pleura.*—More obscure is the differential diagnosis when this condition is present, especially if the new growth takes origin from the costal pleura and has attained considerable size. These tumors cause local bulging of the chest wall, dulness of irregular outline, effusion into the pleural sac, and compression of the lung, large venous trunks, and the œsophagus, and they may invade the mediastinal spaces. In the latter case they constitute a variety of mediastinal tumors. *Aneurism of the Thoracic Aorta.*—The differential diagnosis between mediastinal tumor and aneurism may be very difficult, especially in areas in which the outline of the aneurism is irregular and the sac more or less occupied by firm clot. The symptoms of both are due to pressure, and the effects of pressure are practically the same, whether it be exerted by a solid tumor or one distended with blood. The following data are in favor of mediastinal tumor: marked cyanosis, venous engorgement, and the enlarged superficial veins of collateral circulation; right-sided œdema of the face and arm; absence of diastolic shock; absence of tracheal tugging; relatively short duration; if pulsation be present, it is limited in extent, especially in large tumors, and not expansile in character; if murmurs be present, they are usually systolic only; enlargement of the lymph-nodes and a firm nodular, rather than an elastic, mass palpable at the sternal notch. The following render the diagnosis of aneurism probable: absence of œdema and cyanosis, and the absence of enlarged superficial veins upon the thorax and abdomen; the presence of diastolic shock and tracheal tugging; prolonged duration; expansile, heaving, and forcible pulsation, either in areas in which the tumor lies in relation with the chest wall or has perforated the sternum; double murmurs, inequality of the radial pulses, and severe boring pain radiating to the back, arms, and neck. Loud murmurs heard over a considerable area are common in aneurism, but murmurs may be wholly absent, whereas in mediastinal tumor murmurs are only occasionally heard.

Examination by the X-rays yields conclusive results when the shadow shows in any part the rounded expansile pulsation of an aneurismal sac. Rest in bed, a limited diet, the restriction of fluid, and the administration of potassium iodide may be followed by relief of pain in aneurism.

Diagnosis of the Location of Mediastinal Tumors.—Developing in the anterior mediastinum, new growths push forward and sometimes erode the sternum. They may frequently be felt in the suprasternal notch. The symptoms indicate compression of venous trunks. Dyspnœa is often urgent, and the lymphatic glands of the neck are often enlarged. When the growth occupies the middle and posterior mediastinum the physical signs may be obscure. Pressure is especially exerted upon the œsophagus and the recurrent laryngeal nerves. Dysphagia, urgent dyspnœa, and a brassy, laryngeal cough occur. In tumors springing from the pleura or lung symptoms of pressure upon the blood-vessels, nerves, and gullet are less marked; signs indicating compression of the lung itself more prominent. A complicating pleural effusion is common. There is a tendency to rapid emaciation and cachexia.

Diagnosis of the Character of Mediastinal Tumors.—The most common forms are carcinoma and sarcoma, the former developing as a rule later in life than the latter, but there are many exceptions to this statement. Rapidity of growth, metastatic tumors, glandular enlargement, loss of weight, anæmia, cachexia, subnormal temperature, and cutaneous pigmentation point to carcinoma. In sarcoma the weight and the appearance of fair nutrition may be long maintained.

Prognosis.—Acute enlargement of the tracheal and bronchial lymphatic glands usually subsides with the pulmonary disease of which it is a feature. Suppurating glands may rupture into contiguous structures or undergo retrogressive changes, with resorption of the fluid and deposition of lime salts. Tuberculous glands remain enlarged, with the tendency to caseation and softening. Abscess is a serious affection, but a fair proportion of the cases are amenable to surgical treatment. Chronic proliferative mediastinitis is a progressive disease tending to destroy life by impairing the function of the heart. The conditions under which mediastinal emphysema occur are usually of grave prognostic import. Finally, mediastinal tumors are in a large proportion of the cases malignant and without hope.

VI. DISEASES OF THE PLEURA.

i. Pleurisy.

Definition.—Inflammation of the pleura. The cases may be grouped according to various principles of classification. Etiologically primary and secondary forms may be recognized, but this distinction cannot always be made at the bedside; clinically the disease may run an acute or chronic course, but forms characterized by similar features differ greatly in intensity and duration. The anatomical division into dry or plastic pleurisy and pleurisy with effusion is most convenient for descriptive purposes.

(a) Fibrinous or Plastic Pleurisy.

Pleuritis Sicca.

Acute Dry Pleurisy.—The pleural membrane is the seat of a fibrinous exudate of varying thickness, arranged in a single layer or in superimposed strata. This form of pleurisy apparently occurs in some instances as a primary disease after exposure to cold or contusion of the chest. It is far more frequent as a secondary affection in acute and chronic diseases of the lung when the lesions extend to the pleura. It is an almost constant accompaniment of croupous pneumonia and very common in bronchopneumonia. Pulmonary infarct, abscess, gangrene, and malignant disease cause inflammation of the pleura when they extend to the periphery of the lung. Dry pleurisy is a constant accompaniment of chronic pulmonary tuberculosis, alike when the primary infection involves the lung or the pleura itself.

Symptoms.—The subjective symptoms of pleural irritation are present, namely, the pain known as *stitch in the side*, and dry cough. Fever of moderate intensity is usually also present. The pain is usually referred to the region of the nipple or the axilla. In diaphragmatic pleurisy the pain is often referred to the abdomen, especially in children. It is increased upon deep breathing. The respiratory movement is therefore consciously or unconsciously somewhat restricted, and the patient presses his hand upon the affected side when he coughs. Cough is sometimes absent.

Physical Signs.—*Inspection.*—The respiratory movement is somewhat limited upon the affected side. *Palpation.*—A distinct friction fremitus may very often be felt. This sign is due to roughening of the opposed pleural surfaces. *Percussion.*—There is no change in the sound, but upon linear percussion a limited inspiratory descent of the lower border of the lung upon the affected side may be made out. *Auscultation.*—A pleural friction rub, the almost constant and always distinct sign of dry pleurisy, is heard. When the plastic exudate affects the pleura in the neighborhood of the heart the friction sounds occur, not only synchronously with the respiratory movements, but also with the cardiac revolution—*pleuropericardial friction.* In miliary tuberculosis involving the pleura a fine, widely diffused friction sound may be heard.

Diagnosis.—DIRECT.—The friction sound is of positive diagnostic significance. When it is not heard the diagnosis of fibrinous pleurisy cannot be affirmed. Pain in the side, increased upon cough and deep breathing, occurs in other conditions. Even when the friction sound is present, we cannot in every case be sure that the pleurisy of which it is the sign is not associated with pneumonia, or the forerunner of pleural effusion. Time is therefore in certain cases essential to the diagnosis.

DIFFERENTIAL.—*Pleurodynia.*—Myalgia of the intercostal muscles of one side, intensified by cough and deep breathing, and by pressure often over a circumscribed area, may be mistaken for dry pleurisy. It is more common upon the left side. The absence of friction sounds and the constitutional condition under which myalgias arise render the differential diagnosis an easy matter. *Intercostal Neuralgia.*—The pain is limited to the course of nerve-trunks and is paroxysmal. There are tender points. The disease is common in neurotic and hysterical women. It is a chronic affection. Friction sounds do not occur. *Neuritis of Intercostal Nerves.*—Whether associated with herpes zoster, spinal caries, or disease of the cord, or due to the pressure of a tumor or aneurism, this painful affection of the chest is sometimes mistaken for pleurisy. The one essential diagnostic point is the presence or absence of friction sounds. Causal factors are important, and the distribution of the pain along nerve-trunks with *points douloureux* is significant.

Prognosis.—The outlook is mostly favorable. In the primary form after a few days adhesions take place, the friction murmur disappears, and the pain ceases. The secondary forms often run the same course.

Chronic Dry Pleurisy.—There are three forms: primitive dry pleurisy, that form which follows the resorption or withdrawal of pleural effusions, and tuberculous dry pleurisy.

1. PRIMITIVE DRY PLEURISY.—This variety may develop insidiously without marked symptoms, and be first recognized by the accidental discovery of the friction fremitus, or it may, as is commonly the case, be the outcome of the acute disease. Limited or general pleural adhesions take place. The respiratory function is but little affected. With general bilateral adhesions the respiratory play of the chest is restricted. Percussion is normal. The excursus of the lower border of resonance is diminished and the lessened movements of the diaphragm are confirmed by Litten's sign. This form of pleurisy may result in remarkable thickening and connective-tissue proliferation within the lung, with contraction and induration—*pulmonary cirrhosis.*

2. ADHESIVE PLEURISY FOLLOWING THE REMOVAL OF EXUDATES.—Upon resorption of an effusion, or its removal by aspiration or otherwise, the pleural surfaces unite and the fibrinous material becomes organized. This process is most marked at the base of the chest and gives rise to a characteristic deformity, in which there is flattening, with narrowing of the intercostal spaces and overlapping of the ribs, deficient expansion, enfeebled respiratory murmur, and dulness. The pathological change is largely due to prolonged pressure atelectasis, a fact of great importance as bearing upon the prompt removal of pleural effusions. The condition follows serofibrinous pleurisy, empyema, and traumatism of the chest, especially gunshot and stab wounds. It is of every grade, from the slight retraction of the chest wall following a rapidly removed serofibrinous effusion, relieved by respiratory gymnastics, particularly in young persons, to the gross and disfiguring deformity which is seen after neglected effusions of all kinds, especially old empyemata, and is associated with a permanently compressed, airless, fibroid and bronchiectatic lung.

3. TUBERCULOUS DRY PLEURISY.—The course of tuberculous dry pleurisy is from the onset essentially chronic and characterized by great thickening, together with implication of the connective-tissue framework of the lung. It may involve both sides and usually begins at an apex. Proliferating pericarditis or peritonitis may be also present.

Flushing or sweating of one cheek and dilatation of a pupil may occur when the pleural thickening implicates the upper thoracic ganglion.

The differential diagnosis between a circumscribed pleural effusion and great pleural thickening is in many cases extremely difficult, a fact not surprising in view of the physical condition and the occasional presence of small collections of residual fluid in the thickened pleura. Dulness, even flatness, feeble respiratory sounds, diminished vocal resonance, and absent vocal fremitus occur in both conditions. The use of the aspirator will usually at once determine the diagnosis.

(b) Pleurisy with Effusion.

Pleuritis Exudativa.

Definition.—Inflammation of the pleura in which fluid exudate is associated with the fibrin.

Pleural inflammation is due to microbic infection. The organisms present in the exudate are, with greatest frequency, the tubercle bacillus, the pneumococcus, and the streptococcus; far less commonly the staphylococcus, Bacterium coli commune, Friedländer's bacillus, the bacillus of Eberth, and the Klebs-Löffler bacillus. Mixed infections occur. According to the character of the effusion the following forms are recognized: serofibrinous, purulent, hemorrhagic, and chyliform.

SEROFIBRINOUS PLEURISY.

This term is not used to designate those cases in which small quantities of serum are entangled in the meshes of a loose plastic exudate, but to describe considerable collections of fluid which, unless prevented by adhesions, accumulate under the influence of gravity in the dependent parts of the pleural sac.

Etiology.—PREDISPOSING INFLUENCES.—The cases may be divided into idiopathic and secondary. Idiopathic or primary pleurisy often quickly follows a wetting or chill in an apparently healthy person. The majority of the cases are, however, tuberculous. Serofibrinous pleurisy may follow injury to the chest. With reference to personal predisposition, hospital statistics show a greater liability on the part of males—5 to 1—and in middle life, especially between 40 and 50. The disease occurs, however, at all ages. Secondary pleurisy occurs not only in connection with tuberculous disease of the lung, or tuberculous lesions in distant parts of the body, but also in croupous pneumonia and bronchopneumonia, malignant disease of the pleura or lung, pericarditis, rheumatic fever, enteric fever, diseases of the liver, and chronic nephritis.

EXCITING CAUSES.—Exposure to cold and damp and traumatism lower local tissue resistance to pathogenic micro-organisms.

Morbid Anatomy.—The serous and fibrinous exudates are present in varying proportions. Fibrin may be scanty, or form thick, shaggy layers upon the pulmonary and costal pleuræ and curd-like masses or flocculi which float in the serum and collect in the most dependent part of the pleural sac. The fluid is clear or slightly turbid, according to the relative abundance of cells and fibrin masses which it contains. It is of a pale citron or lemon color, but may be darker. It coagulates on boiling, but sometimes on standing undergoes spontaneous coagulation. Chemically it resembles blood-serum. It may show the presence of cholesterin, uric acid, or sugar. Microscopically there are seen leucocytes, endothelial cells, fibrin shreds, and erythrocytes. The fluid, according to its volume, exerts pressure upon the lungs and adjacent organs. In small effusions the lower lobe is compressed and partially atelectic; in large effusions the entire lung may be reduced to a flat, airless, carnified mass lying against the spine. In large effusions the mediastinum and heart are displaced toward the opposite side, the diaphragm is depressed, and with it the liver or spleen, as the case may be.

Symptoms.—Serofibrinous pleurisy may begin insidiously or with acute symptoms. The former mode of onset is more common in children and aged persons and in the secondary forms which develop in acute or chronic disease. The chief symptoms are shortness of breath on exertion and rapid anæmia. The latter may be preceded by prodromes, or a chill with fever and pleural pain may suddenly occur. If relatively mild these symptoms suggest acute plastic pleurisy; if severe, croupous pneumonia. The pain is severe, lancinating, and aggravated by deep breathing. It is referred to the nipple or axillary region; sometimes, probably when the diaphragmatic pleura is involved, to the umbilical region or the hypochondriac region of the affected side, suggesting gastralgia, gastric ulcer, or an acute inflammatory infradiaphragmatic inflammation, as cholecystitis or appendicitis. In rare cases the pain is located in the lumbar region. The temperature rises gradually rather than rapidly and attains an average of 102°–103° F. (39°–39.5° C.). The fever is atypical and irregular and of varying duration. Surface observations show in the early course of the disease a slightly higher elevation upon the affected side. Cough as a rule is present. It is accompanied by scanty, mucous expectoration. When this contains blood, a larval pneumonia is to be suspected. Dyspnœa is at first due to the fever and pain; later to circumscription of the respiratory surface in consequence of the compression of the lung. The more rapidly the fluid accumulates the more urgent the shortness of breath. A large effusion if slowly formed may cause little or no dyspnœa so long as the patient lies quietly in bed. A moderate leucocytosis—12,000 to 15,000—is present during the febrile period. In a small proportion of the cases the leucocytes are below normal.

Physical Signs.—INSPECTION.—The patient prefers to lie upon his back slightly propped up on pillows or upon the affected side. In large effusions the contour of the affected side appears to be abnormally full, and the chest may show upon measurement, due allowance being made for the normal disparity, an increase of 2 or 3 cm. in the semicircumference. The intercostal furrows are absent. The immobility of the affected side is often in striking contrast to the movement of the sound side, which is exaggerated by vicarious function. In right-sided effusion the apex of the heart may be displaced to the fourth interspace beyond the mammillary line, or even as far as the left anterior axillary line; in left effusions the apex may lie behind the sternum and no impulse be seen; or there may be a visible impulse in the third or fourth interspaces as far to the right as the nipple line. PALPATION.—The signs obtained upon inspection are confirmed by the sense of touch. The vocal fremitus is diminished or absent. In children the fremitus attendant upon crying is sometimes transmitted along the chest wall to the affected side. It may be present in circumscribed areas over large effusions when there are localized old pleural adhesions. Fluctuation is not a sign of simple serofibrinous pleurisy, and œdema of the chest wall scarcely ever occurs. MENSURATION.—The difference in the contour between the sides as determined by the cyrtometer is very striking. There is an increase in the anteroposterior diameter, together with an increase in the semicircumference. The difference in respiratory expansion may be accurately measured by the saddle-tape. **PERCUSSION.**—The percussion sound over the effusion is flat, and the percussing finger perceives an absence of elasticity which is very suggestive. Above the level of the fluid, Skodaic resonance—the sign of relaxation of vital intrapulmonary tension—may be elicited in front and to a less degree behind. The upper line of flatness is not horizontal, but rises in a curve resembling the italic letter *S*, starting at its lowest point from the spine and rising to the axilla, from which it descends obliquely in a straight line to the sternum. This line, known as "*Ellis's line of flatness*," has been established by abundant clinical and experimental studies. It is much modified when the patient has been confined to bed during the accumulation of the fluid, when there are lesions in the lung which modify its shape and consistence, and when the compression of the lung is interfered with by pleural adhesions. It is effaced when the fluid rises above the third rib. The flat percussion sound on the right side is continuous with that of the liver, from which it cannot be discriminated; on the left in the mammillary line it extends to Traube's semilunar space, the convex upper border of which becomes gradually flattened as the fluid increases. The rising and falling of the upper line of dulness, as the effusion increases or undergoes resorption, may be demonstrated by careful, light percussion and markings upon the skin at intervals of two or three days. In moderate effusions in which the lung is not confined by adhesions movable dulness may be demonstrated by marking the upper line of dulness in the anterior surface while the patient is in the erect or sitting posture, and again after some time spent in the dorsal decubitus. Massive effusions reach the clavicle and even extend to the sternal border of the opposite side. The downward dislocation of the liver or spleen may be demonstrated by linear percussion, which enables us to demonstrate the lower borders of these organs respectively. The liver is depressed in very large left effusions by reason of the dislocation of the heart toward the right, the crowding of the lung in the right pleural cavity, and the general depression of the diaphragm.

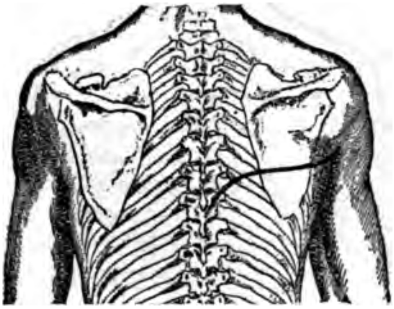

FIG. 319.—Ellis's curve; moderate pleural effusion; patient in upright posture.

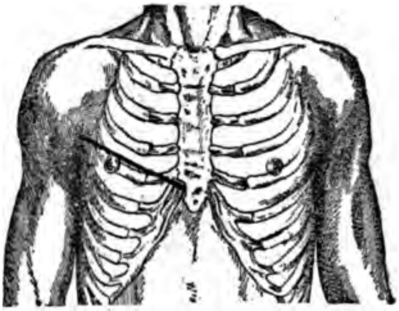

FIG. 320.—Anterior line of flatness.

Normal Paravertebral Triangles of Relative Dulness.—Upon percussion over the spine from above downward, the resonance is progressively diminished in the lower thoracic region. This impairment of resonance also extends laterally in such a manner as to form on each side of the lower thoracic spine a narrow triangle of relative dulness, the base of which corresponds to the lower limit of normal pulmonary resonance.

Koranyi's (Grocco's) Sign; Abnormal Paravertebral Triangles of Dulness.—These triangles appear upon the sound side in pleural effusion, and may differ from the normal triangles only in respect of the degree of dulness. More commonly they differ also in extent. The procedure is as follows: (1) The borders of the effusion are determined by percussion. (2) The base of the lung upon the sound side is ascertained by percussing from above downward. (3) The degree of resonance over the spinous processes is learned by percussion also from above downward, and the point at which relative dulness begins is noted. This commonly is at the level of impaired resonance, or slightly above the level of flatness on the side of the effusion. (4) Percussion upon the sound side in a direction toward the spine in serial horizontal lines from above downward reveals a paravertebral right-angled triangle of dulness, the vertical side of which corresponds to the spine and rises to or slightly above the level of the effusion on the opposite side, the base to the lower border of the lung, while the hypothenuse extends from the apex to the outer and lowest point of dulness. The base line varies with the volume of the effusion and may reach 6 or 10 cm. in length. The triangle is usually larger in right- than in left-sided effusions. The respiratory murmur, vocal resonance, and vocal fremitus are enfeebled over this area of dulness. The phenomenon occurs alike in hydrothorax and in serofibrinous and purulent effusions. The base line is longer in purulent than in serous effusions. The triangle in free effusions disappears upon change from the upright to the recumbent posture. The explanation of the paravertebral triangles of dulness is not clear. It is probable that under normal conditions the vibrations of the lung tissue are to some extent inhibited by the bodies of the vertebræ against which it rests, and that an effusion upon the opposite side acts as a "mute" or damper and still further interferes with the vibrations of the lung in the costovertebral recess of the sound side.

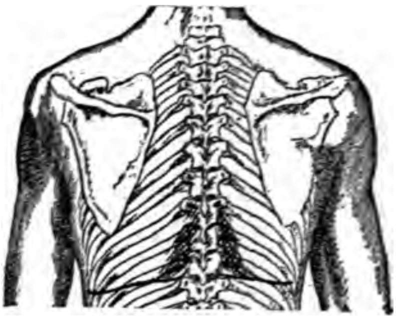

FIG. 321.—Normal paravertebral triangles.

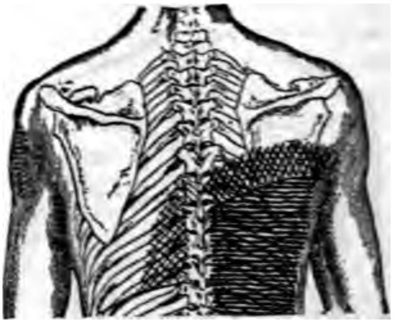

FIG. 322.—Right pleural effusion with triangle of dense paravertebral dulness on left side.

AUSCULTATION.—In the beginning of the attack friction sounds having the quality and situation of the friction signs in acute fibrinous pleurisy are heard. When the case has been under observation from the beginning, the rapid replacement of this sign by flatness is of the highest diagnostic significance. As the fluid undergoes resorption and the pleural surfaces once more come in contact, friction sounds are again heard. At this period they are grating or creaking, or fine and moist, like the crepitus of pneumonia, and are heard just above the level of flatness. As expansion of compressed vesicular tissue accompanies the process of resorption, there are crepitant râles to be heard. As the fluid accumulates, the respiratory murmur becomes at first feeble and distant. Later, while retaining its distant quality it assumes the bronchial character and may be amphoric. Cavernous respiration associated with râles, especially in children, may suggest a cavity. Over large effusions the respiratory sounds may be wholly absent. Above the level of the fluid the respiration is vesiculo-bronchial or bronchial. The vocal resonance is usually diminished or absent. In rare instances there is bronchophony. Ægophony is sometimes heard in the scapular region in medium-sized effusions. The whispered voice is better transmitted through a serous than a purulent effusion—*Bacelli's sign.*

The Heart.—The diastole is restricted by pressure. The sounds are therefore usually less distinct than normal. There is a diminished flow of blood into the arteries, which causes small pulse and a tendency to cyanosis and oliguria. Murmurs are not uncommon in the displaced heart, and a pleuropericardial friction may be detected in many cases.

Clinical Course of Serofibrinous Pleurisy.—The cases may be grouped according to the amount of fluid, which varies up to 4 litres. Less than 500 c.c. cannot be satisfactorily demonstrated in an adult. There are many cases in which the effusion does not exceed this amount and manifests itself by limited dulness at the base of the chest and immobility of the lower border of pulmonary resonance. Spontaneous arrest of the process occurs and resorption begins—*slight effusion.* In another group the fluid reaches to the level of the fourth rib in front and resorption is more tardy—*moderate effusion.* Again the upper line of flatness may reach the second rib—*large effusion;* and finally there are cases in which the outpour of the serous exudate appears to be limited only by the capacity of the pleural sac, and the whole side is distended and flat, the signs of fluid reaching to the clavicle and beyond the opposite sternal border—*massive effusion.*

In slight effusions the fever subsides, the cough ceases, and recovery takes place in the course of a week or ten days. Some impairment of resonance with feeble respiratory sounds usually persists for a longer period. In moderate and large effusions the tendency is to spontaneous resorption, but the process is slow and permanent damage to the lung results from prolonged pressure atelectasis. In this group there is commonly, but by no means invariably, a gradual subsidence of fever. The exceptional cases in which fever persists impair the usefulness of this symptom in the differential diagnosis between serofibrinous and purulent pleurisies. Massive effusions when the intrapleural pressure is extreme are usually attended with distressing pressure symptoms. In cases in which the accumulation of fluid has been slow, the patient may experience merely a sense of weight and oppression, with shortness of breath upon exertion. Large effusions arrest the pumping function of the thoracic organs, and thereby diminish the outflow of arterial blood from, and the inflow of venous blood to, the heart, and the movement of the lymph. They show little or no tendency to undergo resorption. Serofibrinous effusions in very rare instances have perforated the lung or the chest wall. In massive effusions with great dislocation of the heart there is danger of sudden death, an accident attributed to various causes, as heart-clot, embolism of the pulmonary artery, paralysis of the heart muscle, and twists or kinking of the great vessels. The last of these explanations is purely hypothetical. Sudden œdema of the functionating lung occurs when death is not immediate.

PURULENT PLEURISY: EMPYEMA.

This designation is applied to those cases of pleural inflammation characterized by the formation of pus. When pus finds its way into the pleural sac by perforation from neighboring structures the condition is known as pyothorax.

Etiology. — PREDISPOSING INFLUENCES. — Empyema is mostly a secondary affection. It occurs as a sequel to the infectious febrile diseases, especially scarlet fever, and is common after croupous pneumonia and bronchopneumonia and in connection with abscess and gangrene of the lung. It constitutes a rare complication of pulmonary tuberculosis, occurring in tuberculous bronchopneumonia and less frequently in consequence of pleural infection from a caseating lesion or a subpleural tuberculous abscess. Direct infection from without may also occur, as in fracture of a rib or a penetrating wound of the chest. It occurs at every age and is common in young infants, in whom a false diagnosis of pneumonia is frequently made.

EXCITING CAUSE.—The usual organisms in their order of frequency are the pneumococcus, the ordinary pyogenic bacteria, and the tubercle bacillus. The influenza bacillus and the Bacterium coli communis have been found in rare instances. Empyema is not a stage in the course of serofibrinous pleurisy. The conversion of the serous into the purulent effusion is unusual. In very rare instances serofibrinous effusions have been infected in aspiration.

Morbid Anatomy.—The lung is compressed to an airless mass. The pleural surfaces are thickened, and are the seat of a grayish-white granular exudate. Upon the costal layer may be seen superficial erosions and sometimes the openings of fistulous tracts. The fluid has the gross and microscopical characters of ordinary pus, varying from a thin to a thick or creamy consistence. Its odor is sometimes sweetish, but in many cases, especially those following wounds or associated with gangrene, it is horribly fetid.

Symptoms.—An abrupt onset with acute symptoms is rare. The common beginning is insidious, with an intensification of the symptoms of the primary affection. Cough is neither frequent nor urgent; there is little or no expectoration, and dyspnœa, except in large effusions, is present only upon exertion. The symptoms of sepsis, as pallor, chilliness, irregular fever, and more or less profuse sweating, are very common, especially in children. There is a high leucocytosis, 40,000 or more per cubic millimetre.

Physical Signs.—The signs elicited upon physical examination are the same as in serofibrinous pleurisy, with the following superadded: marked bulging of the affected side with obliteration, even prominence, of the intercostal spaces in the lower segment of the chest, especially in children. In many cases œdema and cyanotic discoloration of the lower part of the chest with dilatation of the venules. Whispering pectoriloquy is not heard over the effusion—*Baccelli's sign.* Distinct bronchial breathing, transmitted along the chest wall, is often heard over the effusion in young children, a sign which may lead to a false diagnosis of pneumonia. Displacement phenomena, affecting the heart, liver, and spleen, are more pronounced than in serofibrinous effusion, a fact attributed to the greater weight of the fluid, but probably due to the greater impairment in the tonicity of the tissues from the imbibition of toxin laden fluids.

VARIETIES OF PURULENT PLEURISY.

Empyema Necessitatis.—The pus by erosion of the costal pleura finds its way through an intercostal space and forms a subcutaneous, fluctuating tumor. This tumor may appear at various parts of the chest, but is usually situated anteriorly from the third to the sixth interspace. After a time if left to itself it opens, and an oblique fistulous communication with the pleural cavity is established, which continues to discharge pus for an indefinite time. When near the heart the tumor may pulsate. It is usually hemispherical and diminished in size upon full inspiration.

Pulsating Pleural Effusion.—The pulsation is synchronous with the cardiac revolution, and may be intrapleural and manifest in the lower intercostal space, in the anterolateral aspect of the chest, or show itself merely in an empyema necessitatis. The pulsation occurs in old cases, almost always upon the left side, and with one exception among the reported cases the effusion has been purulent. Various explanations have been advanced, none of which has met with general acceptance.

Encysted or Circumscribed Pleural Effusion.—The effusion is limited by pleural adhesions. The encysted fluid may vary in amount and suggest abscess of the lung, or two or more loculi may communicate with each other by narrow openings. It is sometimes serofibrinous but usually purulent. These collections may be situated between the pulmonary and costal pleuræ, especially in the posterolateral region of the chest between the base of the lung and the diaphragm, or they may be interlobar.

HEMORRHAGIC PLEURISY.

The exudate is mixed with blood. The condition is to be distinguished from hæmothorax, which arises in the absence of pleural inflammation when blood escapes into the pleural sac from traumatism, the rupture of an aneurism, or the compression of thoracic veins by a new growth.

Etiology. — Hemorrhagic effusion is of comparatively infrequent occurrence. It is encountered in pleurisy under the following conditions: in the malignant and hemorrhagic forms of the acute febrile infections; in visceral diseases associated with extensive vascular changes, as chronic nephritis and cirrhosis of the liver; in tuberculous disease, both miliary tuberculosis of the pleura and the more chronic pleural tuberculosis which accompanies chronic ulcerative phthisis; in primary and secondary malignant disease of the pleura,—carcinoma and sarcoma,—very rarely in so-called idiopathic or primary serofibrinous pleurisy, in which, however, red corpuscles are always to some extent present.

CHYLIFORM PLEURAL EFFUSIONS: HYDROPS ADIPOSUS.

The exudate has a milky appearance due to the fatty metamorphosis of endothelial and other cellular elements. The condition is not to be confounded with chylous effusion which it closely resembles. The fact is not to be overlooked that a mixture of chyliform exudates and chylous transudates may be present—as in a case recently under my care.

Etiology.—Chyliform effusions owe their peculiar appearance to, (a) the presence of cells that have undergone fatty degeneration, as in carcinoma of the pleura, tuberculous pleurisy, non-tuberculous exudate, pleurisy and abscess of the lung, and (b) to abnormal fat in the blood—lipæmia.

Diagnosis.—The nature of the effusion cannot be suspected during life unless it is withdrawn by aspiration. The fluid is yellow, whey-like, and cheesy. Upon standing there collects upon the surface a cream-like layer, showing under the microscope small globules, mostly in the form of collections of highly refractive granules with large indistinct nuclei. In the underlying fluid are leucocytes and larger cellular elements which, in consequence of differences in the amount of fat, show all possible transitional forms. (Compare this description with that of chylous effusion, p.1103.) Pseudochylous effusions have been ascribed to the presence of lecithin, and Edsall has described a non-fatty pleural effusion in which the opacity was due to altered globulins.

The Diagnosis of Pleurisy with Effusion.—DIRECT.—The diagnosis rests upon the physical signs. In large effusions the physical examination yields conclusive results. The signs may be divided into primary, or those dependent upon the presence of the fluid *per se*, and secondary, or those due to the pressure of the fluid upon adjacent organs—*displacement signs*. Among the more important of the primary signs are restricted respiratory movement, flat percussion, absence of vocal fremitus, feeble and distant breath sounds, and diminished or absent vocal resonance. The important secondary signs are displacement of the heart toward the opposite side, as shown by a visible or palpable impulse, or, in its absence, by the point of maximum intensity of the first sound; downward dislocation and immobility of the liver when the pleural effusion is right-sided; flattening of the convex upper border of Traube's semilunar space; and displacement of the spleen when the effusion is left-sided. Linear percussion shows restriction or absence of movement of the borders of the lung on the affected side, and inspection increased—vicarious—respiratory movement upon the opposite side. Difficulties arise in moderate effusions. Here the primary symptoms are usually characteristic but the valuable aid afforded by displacement phenomena is lacking. The methods of physical diagnosis must be employed with great nicety in doubtful cases. The *S*-shaped upper line of dulness, movable dulness when present, linear percussion, flatness below and Skodaic resonance above the border line, absent or enfeebled breath sounds, and absent or enfeebled vocal resonance and vocal fremitus are significant. When several or all of these signs are present the diagnosis of effusion can be made with some confidence. In small effusions the diagnostic problem becomes more difficult and more interesting. The same signs are present, but to recognize them demands the highest skill. Finally, we have the aspirator needle which can be used in any case of doubt. There are several reasons why the aspirator should be used in exploratory puncture rather than the hypodermic syringe. The needles are longer and of larger calibre, an important matter in encysted effusions or where there is thick pus; when the exudate is serofibrinous the exploratory puncture becomes at once a therapeutic procedure and a single operation takes the place of two, and when pus is present the ocular demonstration prepares the patient for the necessary later surgical operation of drainage.

The apparatus and spot selected must be sterilized, according to surgical requirements, directly before the operation. The needle should be introduced at a level in which the ordinary signs of effusion, as dulness, absent or enfeebled respiration, and absent or diminished fremitus, are well defined. As a rule, in ordinary effusions the sixth or seventh interspace in the midaxillary line, or a spot just below the angle of the scapula, may be chosen. If the puncture is made too low the needle simply penetrates the costodiaphragmatic reflexion of the pleura and may enter the liver; if too high it will be inserted into the compressed lung above the level of the effusion. The point selected for an exploratory puncture in a circumscribed lesion will be determined by the physical signs. It is an imperative rule to test the instrument with sterile water immediately before it is used. (See p. 458.)

DIFFERENTIAL.—It is in the atypical cases that special difficulties arise.

Croupous Pneumonia.—The general rule that increased vocal fremitus occurs in pneumonic consolidation and diminished or absent vocal fremitus in effusion is subject to exceptions. In consolidation a plug or mass of tough mucus may obstruct a main bronchus and arrest the vibrations, while in effusion they may be distinctly transmitted along bands of old pleural adhesions, or in children from the opposite side along the elastic walls of the chest. The occurrence of bronchophony and bronchial respiration in certain cases add to the difficulty. The following points are to be considered:

(1) In pleurisy, onset with moderate fever and no rigor; at most chilliness or slight chill; (2) dulness increasing to flatness at the base and posteriorly, and extending upward and forward; a peculiar sensation of inelasticity to the percussing finger; (3) vocal fremitus, enfeebled or abolished in the great majority of cases; (4) bronchial respiration, if heard at all, at the upper level of dulness or in patches; usually distant and

faint; (5) bronchophony not intense, ægophony common in the scapular region; (6) friction sounds when the case is seen early and at the upper border of dulness upon resorption of the fluid, when crepitus may be present; (7) in large effusions displacement signs; (8) sputum, mucoid when present, very rarely blood-tinged; (9) fever of irregular remittent type.

In pneumonia, (1) onset abrupt with chill, often prolonged and severe; (2) dulness rather than flatness, coextensive with the borders of a lobe or lobes; (3) vocal fremitus, marked and corresponding to the dulness, and especially when, if feeble or absent, it reappears after cough and the expectoration of tough mucoid sputum; (4) bronchial breathing most marked over area of greatest dulness and often whiffing or snoring in character; (5) bronchophony marked; ægophony rare; (6) crepitant râles, high-pitched and in "showers of crackles" diffused over an area of dulness and disappearing when bronchial breathing becomes intense; (7) displacement phenomena absent; (8) rusty or prune-juice sputum the rule; (9) high temperature of typical range, self-limited course, and critical defervescence. Pleurisy with effusion is frequently associated with croupous pneumonia and bronchopneumonia.

Pericardial Effusion.—When large this condition may simulate left-sided pleural effusion. The outline of the area of dulness anteriorly, its convexity to the right of the sternum, Skodaic resonance at the base and in the axillary region, absence of cardiac impulse on the right, and a degree of dyspnœa and cardiac feebleness not seen in moderate pleural effusions are of diagnostic importance.

Hydrothorax.—When unilateral, this condition cannot always be differentiated from serofibrinous pleurisy by the ordinary methods of physical examination. It occurs in heart disease with great enlargement or dilatation. The diagnosis rests upon concomitant conditions and character of the fluid.

Intrathoracic Tumors.—New growths of the lung, pleura, and mediastinum may be mistaken for pleural effusion. The situation of the dulness and its irregular outline, the signs of marked compression of the large venous trunks, important nerves, and hollow organs, as the trachea, bronchi, and œsophagus, indications of malignant disease in other parts of the body, and enlargement of superficial lymph-nodes should prevent this error. Intrathoracic tumors are very often complicated by pleural effusion.

Aneurism.—Pulsating empyema necessitatis may suggest aortic aneurism. The location of the tumor, usually at the base of the chest, the absence of murmurs, diastolic shock, and tracheal tugging, and the fact that on deep inspiration the tumor diminishes in size and tension are against the diagnosis of aneurism. A fine exploratory needle may be introduced.

Extrapleural Abscess.—This rare condition is to be differentiated from pleural effusion by the absence of the signs of compression of the lungs and the displacement of adjacent organs. When such an abscess is opened pneumothorax does not occur and a probe does not enter the pleural cavity.

Subphrenic Abscess.—This condition may suggest a moderate pleural effusion, from which it may be differentiated by the persistence of the respiratory movement of the lower border of the lung, the presence of food particles in the aspirated fluid when the condition is due to gastric ulcer perihepatic friction, and the absence of pneumothorax upon puncture.

Tumors of the Liver.—Abscess, hydatid cyst, and carcinoma in the right lobe of the liver may displace the diaphragm upward, compress the lung, and cause dulness and feeble respiratory murmur. If large they may also dislocate the cardiac impulse slightly to the left. The diagnosis of hepatic enlargement rests upon the retention of the respiratory movement of the lower border of the lung, friction sounds over the area of dulness, and demonstrable convexity of the upper line of dulness. Exploratory puncture may be performed.

Perforation of the diaphragm may, when adhesions to the liver have taken place, cause a condition not to be differentiated clinically from hepatic abscess, unless the case has been observed from its onset, or pus which is characteristic of hepatic abscess is expectorated or obtained by operation.

The Diagnosis of the Character of the Effusion.—This may be readily settled by the use of the aspirator needle. With this means at our disposal the clinical symptoms, which are somewhat uncertain, assume secondary importance. A serofibrinous effusion is suggested by comparatively mild onset, the absence of the evidence of previous disease, the transmission of the whispered voice, and in general a mild course characterized merely by malnutrition, anæmia, and dyspnœa upon exertion. That the effusion is purulent is rendered probable by the presence of pneumonia, influenza, sepsis or phthisis, irregular chills, high fever and copious sweating, nontransmission of the whispered voice, œdema and cyanosis of the lower portion of the affected side, and a high leucocytosis. But pus may be present in default of several of these symptoms, on the one hand, and, on the other, severe septic phenomena may accompany a moderate, even a small circumscribed serous effusion. Hemorrhagic and chyliform fluids can be recognized only when withdrawn.

The Diagnosis of the Pathological Process.—The examination of the fluid is of great service both as regards diagnosis and prognosis. A majority of serofibrinous effusions are of tuberculous origin. Tuberculous foci may or may not be present in the lungs. The methods of examination comprise microscopy, which may be employed at the bedside, and cytodiagnosis, animal inoculation, and culture methods, which are available only in the laboratory. Lymphocytes generally predominate in tuberculosis effusions; a polynuclear leucocyte preponderance suggests acute infection, and a large number of endothelial cells is found in mechanical effusion or transudate. Inoculation methods, with small amounts of the fluid, as usually practiced are negative, but when larger quantities, as 15 c.c., are used, the result has confirmed the clinical and pathological findings in regard to the preponderance of tuberculous cases in serofibrinous effusions. If actual fragments of cancerous tissue are present the diagnosis is positive. Bacteria are present in small numbers in clear exudates. In purulent exudates they are present in great numbers, sometimes a single variety, sometimes several varieties. Streptococci are most commonly present. The infection may be direct from the lung, as in bronchopneumonia or streptococcus pneumonia, or from distant foci. Less common is pneumococcus infection, which is usually secondary, exceptionally primary.

The Prognostic Value of the Bacteriological Examination of the Fluid.—A sterile fluid usually may be regarded as of tuberculous origin. The presence of the pneumococcus is relatively favorable, since the cases generally run a satisfactory course and recovery may take place after a single aspiration. Streptococcus pleurisy is the most unfavorable of all forms. It is frequently associated with general septicæmia and leads up to the fatal issue. The mixed infections are of unfavorable import.

The Prognosis of Purulent Pleural Effusions.—Empyema is an essentially chronic disease. If neglected the outlook as to recovery is extremely unfavorable, and when spontaneous recovery occurs it is only partial. Early and efficient drainage is followed by a large proportion of satisfactory recoveries. The most unfavorable cases are those which arise in the course of general streptococcus infection. Untreated cases may terminate: 1. In small empyemata, by gradual resorption of the fluid and the deposition of lime salts. 2. By the discharge of the pus through the lung, more commonly after the establishment of a bronchopulmonary fistula, very rarely by soakage without the formation of a demonstrable fistula. In the former case pneumothorax almost always occurs, in the latter probably never. If sudden rupture occurs life may be destroyed by suffocation. 3. By the perforation of the costal or diaphragmatic pleura and the formation of empyema necessitatis which, though usually in the anterior surface of the chest, may be at any point, including the lumbar region and the iliac fossa, where it simulates a lumbar or psoas abscess. Under these circumstances there is usually permanent atelectasis of the lung with fibroid changes, great pleural thickening, and contraction and deformity of the chest. A fair degree of health may be maintained for a varying period, but if the patient survive there is clubbing of the finger-tips, amyloid disease develops, and ultimately tuberculosis in a large proportion of the cases.

Morbid States Characterized by the Transudation of Serum or Chyle, or the Eruption of Pus, Blood, or Air Into the Pleural Sac.

(a) HYDROTHORAX.

Definition.—The accumulation of simple non-inflammatory fluid in the pleural cavities. It occurs as a secondary affection in many diseases, chiefly those attended by dropsy.

Etiology.—The primary disease may involve the kidneys, the heart, or the blood. There is usually more or less anasarca, exceptionally merely slight œdema of the feet. Hydrothorax is in many cases the precursor of death. In disease of the kidneys it is commonly bilateral, the effusion being greater on one side, usually the right. In chronic valvular disease with hypertrophy and dilatation, the effusion is always more marked and sometimes solely upon the right side, and it promptly returns after repeated aspiration. The larger right-sided effusion of cardiac disease has been ascribed to pressure upon the azygos veins, but it is probably due to the larger space in the left thorax occupied by the enlarged heart. Extensive old pleural adhesions may prevent accumulation upon one side. The pleural membranes are not the seat of a fibrinous exudate, being smooth and glistening. The fluid is clear and free from fibrin flocculi. It is usually moderate in amount. The symptoms are dyspnœa, often amounting to orthopnœa, and an aggravation of those due to the primary disease. The physical signs are those of pleural effusion.

Diagnosis.—The condition may be differentiated from serofibrinous pleurisy by the nature of the primary disease, the absence of fever, of displacement symptoms, of friction sounds, and the relatively prompt change in the line of dulness with change of posture.

(b) CHYLOUS PLEURAL EFFUSION—HYDROPS CHYLOSUS.

Definition.—An accumulation of chyle from the thoracic duct or the lacteals by transudation or direct discharge into the pleural sac.

Etiology.—The special causes of chylous effusion into the pleural sac are, (a) conditions leading to an escape of chyle, as external violence, disease or occlusion of the chyliferous vessels, carcinoma of the pleura, occlusion of the left subclavian vein, compression of the duct by a tumor, malignant lymphoma, disease of lymphatic vessels, sclerosis, lymphangiectasis, and filariasis, and (b) the discharge of a chylous ascites into the pleural cavity by way of the lymph spaces.

Symptoms.—The symptoms and physical signs do not differ in any particular from those of a pleural effusion.

Diagnosis.—There are no means by which the nature of the fluid can be determined *intra vitam* except by the withdrawal of a portion of it. In many of the reported cases its presence was first recognized at the autopsy. The fluid bears the closest resemblance to milk, is literally milk-like. It is opaque white in color, with a faint yellowish or creamy shade, slightly alkaline, and of a specific gravity of about 1,017. Microscopically there are seen great numbers of minute, dust-like granules in active, molecular movement, a few larger fatty bodies scattered separately or in groups, a few leucocytes, larger cells containing distinct, highly refracted granules, and a very few erythrocytes. Shaken with ether in a test-tube after the addition of a few drops of potassium hydroxide, the fluid becomes transparent and almost colorless. Upon the addition of osmic acid it becomes black in color. The morphological elements are almost exclusively leucocytes and, in great preponderance, lymphocytes. The fluid is sterile. The foregoing characters serve to distinguish chylous effusion—transudates—from chyliform effusions—exudates (see p. 1098). The presence of grape-sugar is without diagnostic importance, since the fact has recently been established that this substance may frequently be demonstrated in ordinary serous transudates and exudates, and may therefore be expected in chyliform exudates.

(c) PYOTHORAX.

The sudden rupture of a hepatic, subphrenic, mediastinal, or pulmonary abscess into the pleural sac may take place. This accident is usually prevented by more or less extensive pleural adhesions. When it occurs, general infection of the pleura immediately follows with the conversion of pyothorax into purulent pleurisy. Communication with the bronchi or with a subphrenic pneumothorax will give rise to the association of air with the pus—pyopneumothorax.

(d) **HÆMOTHORAX.**

Hemorrhage into the pleural cavity results from trauma, the rupture of an aneurism, the pressure of a tumor upon the thoracic veins, and in rare instances from pulmonary gangrene. The sudden manifestation of the symptoms of internal hemorrhage, pallor, collapse, small, thready pulse, coupled with the physical signs of pleural effusion justify a provisional diagnosis. The withdrawal of blood upon exploratory puncture renders the diagnosis positive.

(e) **PNEUMOTHORAX.**

Hydropneumothorax; Hæmopneumothorax; Pyopneumothorax.

Definition.—Air in the pleural cavity. This condition is extremely rare. Infection of the pleura takes place in almost every instance, and in the course of a short time the air is associated with fluid—*hydropneumothorax, hæmopneumothorax*, or *pyopneumothorax.*

There exists in the normal pleural cavity a negative pressure, by reason of which the lung fills the chest in a state of vital tension. When, through any communication with the external atmosphere, the tension is relieved, the distended lung collapses to the limits of its inherent elasticity, and a volume of air, equivalent to the differences in the mass of the lung under normal distention and under balanced intrapulmonary and intrapleural pressure, enters the pleural sac—*pneumothorax.* This balance is, however, maintained only in the case of the communication remaining freely open as in some external wounds or perforation through consolidated lung tissue. Under other circumstances a valvular action is established, particularly in the perforation through the lung, and the intrapleural pressure gradually becomes positive. While the balance is maintained, the mediastinum is *drawn* toward the opposite side, and the diaphragm somewhat depressed; when the pressure becomes positive, displacement phenomena become more marked, the mediastinum is *pushed* further toward the sound side, and the diaphragm pushed downward.

Etiology. — Pneumothorax is caused by: 1. Perforating wounds of the pleura: (a) through the chest wall, as in the case of stabs and gunshot injuries, aspiration and other surgical operations; (b) internal trauma, as when sharp or pointed foreign bodies are swallowed, or an emphysematous lung or one tied down by local adhesions is torn in violent efforts at lifting or in paroxysms of cough. The accident may even occur in the absence of straining. The air may gradually undergo resorption. More commonly pleurisy with effusion follows. 2. Perforation of the pleura by ulceration or necrosis: (a) from without, as in (i) diseases of the lung: (*α*) tuberculosis, by far the most common cause, the perforating lesion being the softening of a caseous mass or the rupture of a rapidly forming cavity before limiting pleural adhesions have taken place. (*β*)

Necrosis of lung tissue in septic conditions, as septic bronchopneumonia, gangrene, and very rarely infarctions. (ii) Malignant disease of the œsophagus. (iii) Infradiaphragmatic lesions, as (*a*) subphrenic abscess, (β) abscess of the liver, (γ) malignant disease of the stomach or colon. (b) From within, as in empyema, with the formation of a bronchopulmonary or pleurobronchial fistula. 3. As the result of the development in pleural exudates of the gas-producing bacillus—B. aërogenes capsulatus of Welch.

In rare cases pneumothorax is double, and recurrent cases have been reported. The condition is common in adults, exceptional in children.

Morbid Anatomy.—The air space is usually large, the lung compressed and carnified, the pleura inflamed, and serous or purulent effusion present. The confined air may escape through a cannula with a whistling sound and force enough to blow out a candle.

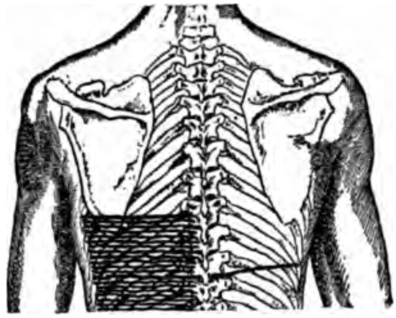

FIG. 323.—Horizontal line of surface of effusion in pyopneumothorax; patient in the upright position. This line shifts with change of posture.

Symptoms.—The occurrence of pneumothorax is usually attended by sudden pain in the side, distressing shortness of breath, slight cyanosis, and feeble pulse. In old tuberculous cases it may occur insidiously.

Physical Signs.—The results of physical examination are characteristic. INSPECTION.—The affected side is enlarged, the intercostal spaces bulge, and the respiratory excursus is greatly diminished. The impulse of the heart is displaced toward the opposite side. PALPATION.—Vocal fremitus is absent or greatly diminished. PERCUSSION.—The signs depend upon the degree of intrapleural tension and the amount of fluid present. When tension is moderate the physical conditions necessary to the production of tympanitic resonance are present, and as these conditions vary the quality of tympany changes from clear, high-pitched hyperresonance to the flat, woodeny tympany of Skodaic resonance. When tension is extreme the physical conditions underlying tympany no longer exist, and the percussion sound is muffled and dull—a fact of great importance in diagnosis. There is flatness at the base due to effused fluid, its upper horizontal line indicating the height to which the effusion rises and changing with change of posture—*movable dulness.* AUSCULTATION.—The breath sounds are feeble and distant and have the amphoric quality. They are in strong contrast with the loud puerile vesicular murmur of the sound side. The voice has also a peculiar, amphoric quality. The ringing musical râle known as *metallic tinkling* or *gutta cadens* is heard upon deep breathing or coughing. The coin test is also present and of positive diagnostic value. Finally, the swash of the free fluid within the pleural cavity upon energetic sudden changes of the patient's body—*Hippocratic succussion*—may often be heard at a distance, or even by the patient himself. In that form of pneumothorax in which there is free communication with a bronchus through consolidated lung, the bulging of the intercostal spaces and displacement signs are less marked, coarse, gurgling râles are observed at times, and there is the occasional expectoration of a thin, purulent fluid. In rare instances of left-sided pneumothorax the heart sounds may have a metallic echoing quality.

Diagnosis.—DIRECT.—The signs are characteristic and the diagnosis, even when in consequence of old adhesions the air space is limited, may be made with confidence.

DIFFERENTIAL.—The following conditions may give rise to uncertainty: *Cirrhosis of the Left Lung.*—The high position of the diaphragm with a dilated stomach yields tympany in the lower part of the left chest, amphoric sounds, and sometimes gastric succussion. The various sounds are little influenced by respiration. Percussion when the stomach is filled with fluid and the fact that the impulse of the heart is displaced toward the left are conclusive. *Diaphragmatic Hernia.*—This condition when congenital may be misleading. After a crushing accident the metallic sounds are related to peristaltic rather than respiratory movements, and the difficulty in passing the tube at the cardiac orifice, owing to the dislocation of the stomach, is suggestive. *Pyopneumothorax Subphrenicus of Leyden.*—The anamnesis is important—symptoms of gastric or duodenal ulcer, chronic intestinal disease, hepatic or splenic abscess usually precede this condition. Cough and sputum are not commonly present; the heart is slightly displaced, the liver much lowered. The lower border of the lung rises and falls, as shown by percussion and auscultation, upon deep respiratory efforts, and movable dulness cannot be made out. *Large Intrapulmonary Cavities.*—Two conditions are to be considered: first, the breaking down of the greater part of a lung—a very rare event—in which the physical arrangement closely resembles ordinary pneumothorax; and second, the cavities resulting from pulmonary abscess, gangrene, or bronchiectasis, or the ordinary cavities of phthisis, which may simulate circumscribed pneumothorax, which is likewise of extremely rare occurrence. In the first of these conditions the amphoric quality of the respiratory and voice sounds may be intense, but the succussion splash, the coin sound, and displacement phenomena are absent. In extremely rare cases, however, coin percussion may yield the bell-like resonance over a cavity. Smaller cavities may be differentiated from circumscribed pneumothorax by the presence or increase of the vocal fremitus, absence of chest distention, sinking of the intercostal spaces over the cavity, and changes in the physical signs, upon percussion and auscultation, after severe cough with copious expectoration. The location of the cavity is without diagnostic importance, since sacculated pneumothorax may occur at the apex, while the cavities following abscess and gangrene are usually in the lower lobe, those of bronchiectasis may occupy any portion of the lung, and those of phthisis, while usually apical, are sometimes situated at the base.

Prognosis.—Spontaneous pneumothorax, occurring upon effort in a person suffering from emphysema or with local pleural adhesion, frequently terminates in recovery with resorption of the air. The traumatic and surgical cases also do well. There is a group of tuberculous cases in which the occurrence of pneumothorax appears to arrest the progress of the disease. There are chronic cases of open pneumothorax, the fistula being either pleurobronchial or external, which last for years, the patients being able to go about and attend to their affairs. In pneumothorax acutissimus death may take place within an hour or in the course of the first day, though the catastrophe may be averted by the use of the trocar and cannula. As a rule, the cases occurring in tuberculosis die in a few days or weeks.

Masked Pneumothorax.—This term has been applied to cases in which the symptoms of pneumothorax, namely, intense pleural pain, dyspnœa, pressure phenomena, and displacement of the mediastinal organs and diaphragm, have suddenly occurred in the course of advanced tuberculosis in the absence of the usual signs of pneumothorax upon auscultation and percussion. In the course of some days these signs gradually appear, and a circumscribed pneumothorax may be demonstrated. They are at first obscured by the deep situation of the collection of extrapulmonary air, which has escaped by way of an opening into the mediastinum, an interlobar space, or a space between the base of the lung and the diaphragm, and is retained by previously formed pleural adhesions. Deep-seated circumscribed pneumothorax is sometimes encountered post mortem in cases in which neither the signs nor symptoms have been observed during life.

XI.

THE DIAGNOSIS OF DISEASES OF THE KIDNEYS.

I. ANATOMICAL ANOMALIES OF THE KIDNEYS.

The kidneys may be displaced, with or without deformity; the displacement may be congenital or acquired. They may vary in number: congenital absence of one or both kidneys, supernumerary kidneys, atrophy of one kidney. They may be anomalous in form: general departures from type, as lobulation; hypertrophy of one or both organs, and fusion —horse-shoe kidney, sigmoid kidney, disk-shaped kidney. Finally, there may be variations in the blood-vessels, pelvis, and ureters.

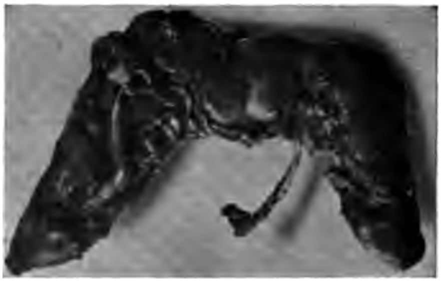

FIG. 324.—"Horse-shoe" kidney —German Hospital.

Of these abnormal conditions the hypertrophied kidney can be diagnosticated only when the affected organ is movable and is recognized upon palpation through the abdominal wall; the horse-shoe kidney only when it has descended to a position just above the promontory of the sacrum and can be felt through thin abdominal walls as a prevertebral tumor with a non-expansile pulsation communicated from the underlying aorta, upon which it in part rests; a single kidney may be suspected when tympanitic percussion resonance is constantly elicited in one lumbar region and no movable kidney is palpable, or when, after an attack of renal colic with impaction, complete anuria and ultimately fatal uræmia occur. In rare cases the impaction of a calculus upon one side may be followed by anuria when both kidneys are present. Other anomalies cannot be recognized during life.

II. MOVABLE KIDNEY.

Ren Mobilis; Palpable Kidney; Floating or Wandering Kidney; Nephroptosis.

Etiology.—The condition may be congenital, the kidney being surrounded by peritoneum which forms a mesonephron. Far more commonly it is acquired. It is probable that congenital defects in the mechanism of attachment are at fault in all cases. Wasting of the perirenal fat is a factor. Movable kidney has been observed at all ages, but is most usual in middle life. It is more common in women than men in the proportion of 7 to 1—a fact attributed to compression of the base of the chest by the corset and the change in the position of the uterus and the relaxation of the abdominal wall after repeated child-bearing. It occurs, however, in women who have never borne children. It is mostly unilateral, several times as often on the right side as on the left, and occasionally double. The greater frequency on the right side is attributed to the relation of the right kidney to the liver and the respiratory movement communicated to it by the latter organ. In the anamnesis there is sometimes an antecedent history of injury or strain. The kidney undergoes dislocation together with the other abdominal viscera in Glénard's disease—*enteroptosis.*

Symptoms.—In a large proportion of the cases there are no definite or characteristic symptoms. Neurasthenic and gastro-intestinal symptoms are common. Constipation is frequent and fecal obstruction may occur. Dragging pains in the lumbar region, especially upon prolonged standing, are observed. Neuralgic pains in the abdomen occur. The tumor, which is often accidentally discovered by the patient, is not tender upon gentle pressure, but when firmly compressed there is a dull, sickening pain. *Dietl's Crises.*—In some cases of floating kidney there are paroxysmal attacks, characterized by abdominal pain, nausea, and vomiting, with chills, fever, and collapse. These attacks have been mistaken for renal colic, acute intestinal disease, and appendicitis, but the kidney may be felt and is tender, swollen, and less freely movable than usual. The urine during the attack may contain uric acid or calcium oxalates in excess, and intermittent hæmaturia may occur. These paroxysms have been ascribed to torsion of the renal vessels. Intermittent hydronephrosis sometimes occurs. The nervous symptoms of movable kidney are important. In women hysterical manifestations, in men hypochondriasis are common. Such patients are very susceptible to suggestion, and their sufferings are often much increased when the diagnosis is communicated to them. In other cases a plain statement of the cause of the trouble is followed by relief.

Physical Examination.—The patient should be placed upon his back, with the abdominal muscles relaxed. In well-marked cases the tumor is plainly visible and palpable in the erect posture. Ordinarily, upon bimanual palpation in the dorsal decubitus, one hand being placed in the lumbar region, the other in the hypochondrium, with gentle manipulation during full respiratory movements, the kidney, if movable, may be recognized by the fingers upon the abdomen as an oval, smooth mass. By this manœuvre various degrees of mobility may be determined. (1) PALPABLE KIDNEY.—The lower end of the organ may be felt upon deep palpation just below the edge of the ribs in the nipple line—a condition of little or no clinical import. (2) MOVABLE KIDNEY.—Upon deep inspiration the fingers upon the abdomen may, if there is little abdominal fat, be pressed over the upper end of the kidney, which can be thus fixed for the time but has no wider excursion—a degree of dislocation the importance of which is frequently overrated. (3) FLOATING OR WANDERING KIDNEY.—The organ may be felt as an oval, smooth, solid tumor, having the size and contour of the kidney. In some instances the hilum and pulsating renal artery can be recognized. This tumor is freely movable, and sometimes lies just above Poupart's ligament, or may by gentle pressure be displaced to the median line or beyond it. In different postures the wandering kidney changes its position, falling forward in the knee-elbow position, and away from the abdominal wall in the dorsal decubitus, when it is often possible to slip it upward into its normal place. To this degree of displacement belong the more distressing symptoms of *ren mobilis.* Dilatation and downward displacement of the stomach can be demonstrated in a large proportion of the cases, especially in women.

Diagnosis.—DIRECT.—A positive diagnosis can usually be made in palpable and movable kidney by the position of the smooth, rounded, firm tumor, which descends with deep inspiration and can be made to disappear by pressure upward and backward, particularly when, as sometimes happens upon repeated examination, flattening or tympanitic resonance is found in the renal region upon the same side. Floating kidney rarely presents difficulty in diagnosis.

DIFFERENTIAL.—*Tumor of the Gall-bladder.*—The mass presents at the border of the ribs, has the respiratory movement of the liver, cannot be grasped from above, and when forced backward immediately returns to its former position. Its movement is less extensive than in floating kidney and is, roughly speaking, in the arc of a circle having its centre in the normal position of the gall-bladder. Furthermore, in tumors of the gall-bladder the upper margin reaches and is continuous with the liver. Other tumors of the liver and tumors of the bowel are fixed and do not present the characteristic contour of the kidney. *Movable Spleen.*—Any doubt as to whether a movable tumor upon the left side is the kidney or spleen is at once settled by the shape of the tumor and the presence or absence of the normal area of dulness in the splenic region. *Tumor of the Pylorus.*—Carcinoma in this region may be freely movable. Under such circumstances the shape of the tumor, its relation to the stomach, filled and emptied by means of the stomach tube, dulness in both renal regions, and the prominence of gastric symptoms are of diagnostic value. *Ovarian Cysts.*—The facts that the tumor arises from the pelvis, that its outline is round or globular, that it is elastic rather than firm, and that it cannot be made to disappear into the normal position of the kidney, readily settle any doubt as to the differential diagnosis.

Prognosis.—The outlook as to permanent fixation is less hopeful than as to relief by the adjustments that follow improvement in the general health. Nephropexy and nephrorrhaphy, with and without decapsulation, have many successes and many failures to their credit. Relief in many cases may be obtained by a suitable belt and pad and treatment of the neurasthenia.

III. CIRCULATORY DERANGEMENTS.

Theoretically anæmia and congestion occur.

(a) **Renal Anæmia.**—The oncometric observations of Mendelsohn indicate that the kidneys are small and bloodless in acute fever. This investigator holds that the scanty, high-colored urine of febrile states is due to anæmia. Clinicians have generally attributed it to renal congestion. No positive diagnosis of anæmia of the kidneys can be made.

(b) **Congestion of the Kidneys.**—ACTIVE CONGESTION.—*Etiology.*—Certain drugs, as the terebinthinates and cantharides, when taken in overdoses, are accredited with causing congestion of the kidneys. Exposure to damp and cold, various poisons and irritants have the same effect. Active hyperæmia is characteristic of the onset of acute nephritis, from which it cannot be clinically differentiated. Post mortem the kidney is large, dark, and soft, and upon section drips blood. The condition is typical in postscarlatinal nephritis. The urine is scanty, densely albuminous, and contains red blood-corpuscles and tube-casts.

PASSIVE CONGESTION.—*Etiology.*—The hyperæmia is mechanical. It results from the transference of blood-pressure from the arterial to the venous side of the circulation, which occurs in cardiac disease and emphysema, and locally from pressure upon the renal veins by the pregnant uterus, abdominal tumors, and large ascites. The condition found post mortem is known as cyanotic induration, and is a form of chronic diffuse nephritis. The urine is diminished, dark red in color, of high specific gravity, and contains albumin in moderate amount, with hyaline tube-casts. A few red blood-corpuscles may be present in the sediment. The line between congestion and nephritis cannot always be drawn at the bedside. Hyaline casts only, moderate albumin, isolated red corpuscles, total absence of uræmic symptoms, cynosis rather than pallor, and improvement upon the administration of digitalis suggest, in a heart case, the diagnosis of congestion rather than nephritis. *Prognosis.*—The causal conditions in chronic hyperæmia of the kidneys are such as to render the prognosis unfavorable. Congestion tends to pass into nephritis.

(c) **Hemorrhagic Infarct of the Kidney.**—*Etiology.*—Embolism of renal arteries occurs in valvular disease, endarteritis, and traumatism involving the renal artery. *Symptoms.*—Sudden pain in the region of the kidney upon one side, with corresponding tenderness upon pressure, and hæmaturia constitute the symptoms in well-marked cases. These symptoms are all transient, the pain and tenderness subsiding in the course of a day or two, the blood disappearing from the urine in three or four days, and the albumin a short time later. In the majority of instances in which infarcts are found post mortem no clinical symptoms have been noted. *Diagnosis.*—Hemorrhagic infarct of the kidney cannot, as a rule, be diagnosticated during life. When the above symptoms occur in a patient in whom the etiological factors are present, or in whom embolic processes elsewhere can be demonstrated, the diagnosis is positive. *Prognosis.*—The outlook is that of the underlying morbid condition. Old infarcts of the kidneys are often found in post-mortem examinations.

IV. URÆMIA.

Definition.—A toxæmia developing in the course of acute or chronic nephritis and other conditions characterized by deficient urinary secretion or complete anuria, and manifested by irregularly associated nervous and gastro-intestinal symptoms.

Various hypotheses have been advanced regarding the pathology of uræmia, among which the following are important: 1. That it is due to the accumulation of excrementitious substances normally eliminated by the kidneys, especially urea, salts, and nitrogenous bodies. 2. That it is caused by toxins evolved in the course of abnormal tissue metabolism, of the nature of which nothing positive is known. Uræmia has been attributed to derangements of a hypothetical internal secretion of the kidney. 3. That the nervous symptoms are largely due to local cerebral œdema.

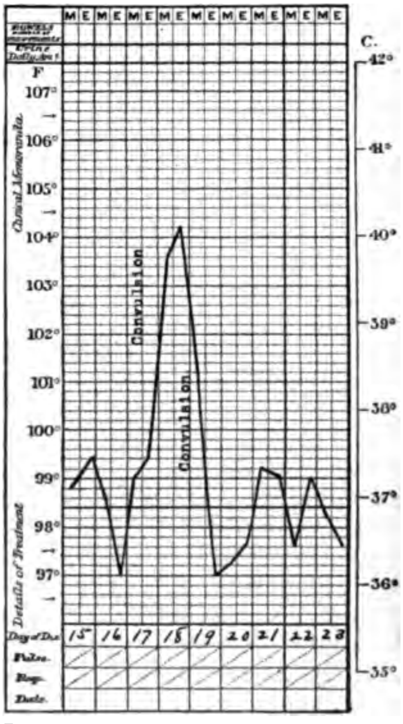

FIG. 325.—Chronic parenchymatous nephritis. Uræmia; convulsions; recovery.

Symptoms.—Uræmia may be of every grade of intensity and of the most variable duration. Latent, acute, and chronic forms are therefore described.

THE LATENT FORM.—This form has been especially studied in cases of complete anuria. The patient may suffer very little inconvenience. Preliminary headache and the alternation of convulsions and coma seen in acute uræmia are often absent. The mind remains clear, the pupils are contracted, muscular twitchings and vomiting occur. The temperature is subnormal.

THE ACUTE FORM.—The onset is preceded by headache, mental confusion, dulness, and drowsiness. The attack begins abruptly with vomiting and diarrhœa, or convulsions alternating with or followed by coma, or coma may develop in the absence of convulsions. Such an attack very often occurs in persons in whom no previous indications of nephritis have been observed. Fever of irregular type is frequently present, and may be, in acute nephritis, a manifestation of the underlying disease or symptomatic of some complication, as an intercurrent inflammatory or infectious process, itself the cause of the uræmia, or the fever may be part of the uræmic symptom-complex.

THE CHRONIC FORM.—The patient may go about and in a way attend to his affairs. He suffers, however, from headache, vertigo, confusion, drowsiness, and pruritus, and very often has transient muscular twitchings. Dyspnœa, which may be continuous or paroxysmal, and is frequently nocturnal only, is a common symptom in chronic uræmia. It is often regarded as asthma. The respiration is sometimes Cheyne-Stokes in type. Itching, numbness, and cramps in the calves of the legs also occur. Local palsies, hemiplegias, and monoplegias occur, and are frequently transient. The psychoses of chronic uræmia are important. They very often occur in persons not known to have nephritis. Mania and delusional insanity are common. Delusions of persecution, suicidal tendencies, and melancholia occur. The alienist may be in doubt whether an insane person has nephritis, or a patient suffering from nephritis has an uræmic psychosis—an uncertainty which emphasizes the artificial character of nosological classifications.

The convulsions of uræmia may occur abruptly or after a spell of headache and restlessness. They closely resemble the epileptic seizure, though the epileptic cry is said not to occur. The repetition of the general convulsion with unconsciousness in the intervals may suggest "status epilepticus." Jacksonian epilepsy may occur. The temperature sinks as a rule after the attack. Uræmic amaurosis may occur after a convulsive attack, or in the absence of convulsions. It may pass off in the course of a few days. The ophthalmoscopic findings are negative. Uræmic deafness of the same fleeting character has also been observed.

Persons suffering from chronic uræmia frequently have no appetite and a foul tongue and breath. The stomatitis sometimes present has no special characters.

In grave cases of acute uræmia a frost-like efflorescence of urea has sometimes been observed upon the skin. Acute inflammations of the serous membranes, endocarditis, pericarditis, pleurisy, peritonitis, and, much more rarely, meningitis occur as terminal events in patients suffering from conditions in which chronic uræmia has developed.

Diagnosis.—DIRECT.—The diagnosis of uræmia depends upon the association of nephritis and nervous symptoms of more or less irregular character and combination. In cases of anuria from any cause a direct diagnosis is justified. In other cases an examination of the urine yields definite data. The nervous symptoms are often such that a diagnosis by exclusion becomes necessary. The copious vomitus in some cases may have the odor of ammonia, since the urea in the gastric contents may have undergone the change into ammonium carbonate. The determination of the urea output in the urine cannot be relied upon as indicating the approach of uræmic symptoms.

The diagnosis of the latent uræmia of Roberts rests upon the association of certain of the milder symptoms of the condition with more or less complete anuria. In acute fully developed uræmia with vomiting, convulsions, coma amaurosis, and stertorous or Cheyne-Stokes respiration, the symptom-complex is so characteristic that errors in diagnosis seldom occur. If some of these symptoms suddenly develop as the result of intoxications or severe infectious processes, in persons not suffering from nephritis, the presence of albumin in relatively small amounts and hyaline casts only, without red blood-corpuscles or other tube-casts, particularly if the specific gravity of the urine be not abnormally low, is of diagnostic value. Nor is it always easy to recognize the nausea, vomiting, and diarrhœa of the gastro-intestinal form as uræmic. Chronic uræmia is even more difficult of recognition. Asthma-like attacks with shortness of breath, especially at night, may lead to an incorrect diagnosis in cases in which the nervous symptoms are slight and ill-defined. The acute attack very often occurs in the course of the chronic condition. The pupils are inconstant. They may be dilated or normal. The presence of albuminuric retinitis may be of positive diagnostic significance.

DIFFERENTIAL.—1. CEREBRAL DISEASE.—(a) The uræmic attack, with sudden loss of consciousness, and hemiplegia, especially when these symptoms are associated with convulsions, may present the clinical picture of *apoplexy* from cerebral hemorrhage or thrombosis. In favor of the latter diagnosis are the abruptness of the onset, the completeness of the loss of power, conjugate deviation, and persistence of the symptoms. It is characteristic of the nervous symptoms of uræmia that they are incomplete and transitory. (b) *Meningitis.*—Sudden coma following headache and vomiting, without localizing phenomena but attended by albuminuria, may present great difficulties in diagnosis. The results of spinal puncture are important Stiffness of the neck, paralysis of cerebral nerves, retraction of the abdomen, and Kernig's sign occur in meningitis. (c) *Tumors.*—The symptoms of coarse lesions of the brain may suggest uræmia. Jacksonian and general convulsions, vomiting, headache, vertigo, and hemiplegia and monoplegia occur in both conditions. But in anatomical lesions the symptoms are chronic and usually though not always progressive, while in uræmia characterized by such symptoms the attack is sudden and frequently transitory. 2. SEVERE INFECTIONS.—There are cases of uræmia in which stupor, a dry tongue, rapid, feeble pulse, muscular twitching, and fever persist for weeks, and the appearance of the patient suggests an acute specific disease with secondary infection. These cases may resemble: (a) *Enteric Fever.*—The differentiation from uræmia depends upon the presence of a pulse relatively slow, as compared with the rise of temperature, splenic tumor, rose rash, a temperature range conforming to type, and a positive Widal reaction. (b) *Miliary Tuberculosis.*—The pulmonary symptoms and signs, the signs of an associated pleurisy or pericarditis, and choroidal tubercles when present suffice to establish the true nature of the affection. (c) *Septic Conditions.*—Local necrotic processes, multiple foci of inflammation, irregular chills, fever and sweating, embolic phenomena are diagnostic. 3. INTOXICATIONS.—Uræmic coma may be mistaken for poisoning by alcohol or opium. The anamnesis is important. In all cases the urine must be drawn and examined. The circumstances in which the patient is found and the odor of the breath may be suggestive. (a) *Alcohol.*—The temperature is subnormal, the pupils usually dilated, the coma often incomplete; if it alternates with delirium the latter is of peculiar type and attended with tremor. (b) *Opium.*—Contracted pupils, slow pulse and respiration, profound stupor from which the patient cannot be roused favor the diagnosis of opium poisoning rather than uræmia.

Prognosis.—As uræmia is a secondary toxæmia the prognosis depends upon that of the primary disease. Latent uræmia dependent upon anuria may disappear when the flow of urine is re-established. The uræmia of acute nephritis disappears with recovery from the renal condition; that of the chronic forms may be transient and recurrent. A patient in the Pennsylvania Hospital was unconscious for several days, with convulsions alternating with coma, but recovered and lived four or five years, working as a laborer. Sudden uræmic coma is common in chronic interstitial nephritis, and may be the first indication of renal disease. It is frequently fatal.

V. INFLAMMATION OF THE KIDNEYS.

The inflammations of the kidneys comprised under the general term Bright's disease cannot be satisfactorily classified, either from the clinical or the pathological stand point. Still less can the clinical varieties be closely coördinated with the post-mortem findings. Clinically the cases may be grouped as acute and chronic; anatomically they are all diffuse; that is to say, epithelial, vascular, and intertubular tissues are involved, but, since the changes in these structures vary in degree, parenchymatous, glomerular, and interstitial forms are recognized, according as one or the other of these groups of tissues is particularly affected.

(a) Acute Nephritis.

Acute Parenchymatous Nephritis; Acute Bright's Disease.

Definition.—Acute diffuse inflammation of the kidneys, caused by the action of cold, poisons, or the toxins of the infectious diseases, and characterized by scanty urine containing albumin, blood-corpuscles, and tube-casts, a tendency to dropsy, and evidences of toxæmia.

Etiology.—Exposure to cold and wet is very often followed in the course of a day or two by the evidences of acute nephritis. Trench diggers and other laborers in low wet places are especially liable. It is common after the exposure incident to a debauch. Certain drugs, as cantharides, internally administered or externally applied, turpentine, balsam of Peru, potassium chlorate, naphthol, and certain acids, as sulphuric acid, salicylic acid, and phenol, in excessive doses are sometimes followed by this form of nephritis. The nephritis which frequently follows scarlet fever is typical. Less common and usually less intense is the acute nephritis associated with pneumonia, enteric fever, influenza, and diphtheria. The acute nephritis of yellow fever and cholera is of severe type. Nephritis may occur in association with variola, varicella, meningitis, syphilis, septic conditions, purpura, and angina tonsillaris. The acute nephritis of pregnancy is probably caused by toxins of unknown nature, and the remarkable form which occurs after extensive burns and other cutaneous lesions probably belongs to this group.

Symptoms.—The general symptoms after exposure to cold and wet usually develop suddenly; after poisoning and the infections, gradually. The onset in children may be attended with convulsions; in adults, by a chill or chilliness. Much more common are such initial symptoms as pain in the back, nausea, vomiting, and headache. Pallor, puffiness about the eyes, and œdema of the ankles are very often the first symptoms to call attention to the kidneys. Fever is not constant. It is more common in children than adults. The temperature may reach 102°-103° F. (38.9°-39.5° C.). Its range does not conform to type.

The urinary changes are characteristic. The quantity is at first greatly diminished. Anuria may occur. Usually a few ounces—100 to 200 c.c.—are secreted in twenty-four hours. The specific gravity is high—1.020 to 1.030. Later, when the secretion is re-established, the specific gravity falls to normal or below it. The percentage of urea is high, but the total quantity is greatly reduced. Owing to the excess of solids the urine is not transparent. It varies in color from a mere smokiness to the dense, opaque brown of porter. These changes are due to the presence of blood, but the urine is never bright red. Upon standing an abundant, dark, coarse sediment is precipitated, which consists of red blood-corpuscles, epithelium from the urinary tract, uric acid and other crystals, and hyaline, granular, blood, and epithelial tube-casts. Albumin is abundant and upon testing precipitates in coarse, curdy flakes. Upon the application of heat the urine may solidify in the test-tube. The foregoing urinary changes are of highest grade in the beginning of the attack. They are to some extent a measure of the severity of the disease and they gradually lessen as improvement occurs in favorable cases.

Dropsy, though exceptionally absent, is a frequent and important symptom. It varies from mere puffiness about the eyelids to a general anasarca with effusion into the serous sacs. It is a peculiarity of the dropsy of acute nephritis that it is irregular in its distribution and does not always gravitate according to the posture of the patient. The degree of œdema is greater after colds, in pregnancy, and after scarlatina than after the other infections. In the nephritis of diphtheria there may be little or none. There are cases of post-scarlatinal nephritis in which effusion into the serous cavities occurs with scanty subcutaneous œdema. Pulmonary œdema and œdema of the glottis may occur. Anæmia is an early and marked condition. Epistaxis is common and symptomatic purpura not infrequent. The pulse tension may be increased and the aortic second sound accentuated. Acute dilatation of the heart may occur. Albuminuric retinitis is comparatively infrequent, though retinal hemorrhages are occasionally encountered.

Uræmic symptoms, among which we include the preliminary anorexia, dulness, and headache, and the initial nausea and vomiting, are almost constant. When to these minor symptoms caused by the retention of excrementitious substances are added convulsions and coma, the condition of acute uræmia is fully established. This may occur at any period in the course of the attack.

Diagnosis.—DIRECT.—The general symptoms are variable and by no means characteristic. Pallor, with slight puffiness of the ankles or eyelids, may be present in the absence of subjective sensations of impaired health; or the symptoms of the causal affection may mask those of the nephritis. This is apt to be the case in pregnancy. It is therefore imperative that the urine be examined at intervals of two or three weeks as a matter of routine during gestation. The clinical picture of acute nephritis in the acute cases following cold, or occurring after scarlatina, is such as to justify a positive diagnosis. In the insidiously developing cases the conditions may be less obvious. The urinary findings as given above are of diagnostic significance. The presence of blood-corpuscles with blood and epithelial casts is characteristic.

DIFFERENTIAL.—1. *Febrile or toxic albuminuria* cannot in all cases be distinctly differentiated from an infectious nephritis. In favor of the former is the absence of special symptoms, pallor, slight œdema, or uræmic phenomena, and certain characters of the urine, namely, albuminuria of lighter degree and transitory duration, lower specific gravity and larger quantity of the urine, and the absence of blood-corpuscles and blood and epithelial casts.

2. *Intercurrent acute nephritis* in the course of chronic nephritis. This condition is by no means infrequent. The acute attack has the clinical phenomena of the primary affection and is often regarded as primary. Attention to the anamnesis, which shows antecedent poor health, characterized by weakness and lassitude, headache, gastric derangements, pallor, and slight or transitory œdema, and usually the absence of any recent definite causal factor, may explain the occurrence of acute dropsy with toxic phenomena. The urinary changes are less sharply defined than in the primary cases, and a tense pulse with cardiac hypertrophy and accentuated aortic second sound, and particularly albuminuric retinitis, render the diagnosis of coexistent chronic nephritis certain.

3. *Glomerular Nephritis.*—The attempt to differentiate cases in which vascular changes are primary and severe, while the epithelium and intertubular tissues are affected to a less degree, constitutes an extreme refinement of diagnosis. This condition is present in post-scarlatinal nephritis, a variety characterized by anuria, extreme dropsy, and acute uræmic symptoms. The absence of epithelial casts might under such circumstances have some degree of diagnostic significance.

Prognosis.—The outlook depends more upon the course than upon the immediate condition of the patient. It is more unfavorable in post-scarlatinal and puerperal nephritis than in other forms. Acute nephritis following cold is less dangerous. That which follows the various infections other than scarlet fever is usually of milder type. Complete and rapid recovery may follow the intense forms associated with yellow fever and cholera. The death-rate in infancy is not lower than 33 per cent. The prognosis as to entire recovery is uncertain. Acute nephritis is very often the point of departure for the chronic form. Even with apparent recovery there remains an especial liability to attacks later in life. At the onset neither the dropsy, the amount of urine, nor the proportion of albumin which it contains justifies a positive prognosis. Urgent uræmic symptoms are always alarming. In the gravest acute nephritis dropsy may be absent. Complete anuria lasting for a day or two may occur in cases terminating favorably, and dense albuminuria often gradually disappears. Low arterial tension, intense anæmia, persistence of dropsy, effusion into the serous sacs, continuing albuminuria of high grade, and chronic uræmic symptoms are of unfavorable prognostic import. Cases thus characterized are liable to an acute fatal exacerbation, or escaping that, to a chronic course. The absence of these conditions is favorable. Recovery may be practically complete in four or six weeks. In other cases a favorable termination may occur at the end of several months.

(b) Chronic Nephritis.

Chronic Bright's Disease.

Anatomically two principal forms are encountered, namely, chronic parenchymatous nephritis and chronic interstitial nephritis, and these respectively manifest themselves by a more or less well-defined symptom-complex.

1. CHRONIC PARENCHYMATOUS NEPHRITIS.

Chronic Desquamative Nephritis; Chronic Tubal Nephritis.

Definition.—Chronic diffuse inflammation of the kidneys, occurring as a sequel of acute nephritis or developing insidiously, and characterized by albuminous urine of high specific gravity, containing tube-casts.

Etiology.—This form of nephritis frequently follows the acute variety and is due to the same causes. In many cases it insidiously develops after an acute attack in the course of a chronic infection. It is common in persons who have suffered from repeated attacks of malarial fever, and in chronic alcoholism. It is met with at all ages, but is especially common in early adult life, and in children as a sequel of scarlatinal nephritis. In adult life it is more frequent in males than females.

The kidneys may show the changes which constitute the large white kidney, the small white or pale granular kidney, or the kidney of chronic hemorrhagic nephritis. Whether the pale granular kidney is a condition consecutive to the large white kidney, or an independent pathological process from the beginning, is not positively known. These two conditions cannot be differentiated clinically either by the symptoms or by the composition and contents of the urine. A protracted course, with the symptoms and urinary changes of parenchymatous nephritis passing by degrees into those suggestive of the chronic interstitial form, would support the opinion that the small white kidney may be a later stage of the large white kidney.

Symptoms. — When consecutive to the acute form, chronic parenchymatous nephritis presents similar, though less urgent, symptoms. A majority of the patients, after a period of failing health with ill-defined symptoms, become pallid and puffy about the eyelids and ankles, or the albumin and casts are discovered upon routine examination of the urine.

The urine, especially in the earlier course of the disease, is diminished in quantity, averaging in the adult 750 to 1000 c.c. in the course of twenty-four hours. With increasing dropsy the output usually diminishes, to again increase as the dropsical effusion undergoes resorption. It is dirty yellow in color and turbid from the presence of urates. Upon standing it precipitates a sediment, which reveals upon examination leucocytes, often red blood-corpuscles, epithelium from the urinary passages, and many tube-casts, hyaline, granular, fatty, and epithelial. Albumin is usually abundant, showing a relative decrease during repose and an increase after exercise. It varies in amount from .5 to 2 per cent. The specific gravity is above normal, varying from 1.025 to 1.035. As the case progresses, the average daily quantity of urine augments, while the average daily amount of albumin excreted declines, and the specific gravity falls. The daily amount of urea fluctuates, but the average is below the normal. The presence of blood in considerable quantities is suggestive of chronic hemorrhagic nephritis.

The tendency to dropsy is characteristic of this form of nephritis. The eyelids and ankles early in the disease, the subcutaneous tissues generally later, and toward the close the serous sacs become the seat of dropsical effusions. The loose tissues of the genitalia in both sexes, and dependent parts in general, become in advanced cases highly distended and œdematous. Uræmic symptoms, especially headache and drowsiness, are common. Gastro-intestinal symptoms, anorexia, nausea, vomiting, and diarrhœa belong to this group. Convulsions may occur as the end approaches. Albuminuric retinitis is not common early in the disease. Hypertrophy of the heart is of less frequent occurrence and less marked than in the chronic interstitial form. The longer the duration of the disease the greater the tendency to cardiac enlargement. The pulse tension is usually increased, the aortic second sound accentuated. and some degree of arteriosclerosis gradually develops. In chronic parenchymatous nephritis there is a peculiar tendency to bronchitis, pneumonia, pleurisy, and pericarditis.

Diagnosis.—DIRECT.—The recognition of this form of nephritis depends upon the association of the above-described urinary changes, the tendency to dropsy, and mild uræmic symptoms, as headache, drowsiness, loss of appetite, and nausea. In mild cases the diagnosis must rest upon the condition of the urine. The facies, which exhibits marked pallor, a dull, pasty complexion, and puffy eyelids, is suggestive.

Prognosis.—The prognosis is grave, both as to life and as to recovery. A limited proportion of cases under very strict and judicious management, especially among children, recover in the course of a year or two. The greater number have already, when the diagnosis is made, entered upon a life of chronic invalidism. The scene closes with increasing and unmanageable dropsy, acute uræmic symptoms, or intercurrent acute inflammation, usually of the pleuræ or pericardium.

2. CHRONIC INTERSTITIAL NEPHRITIS.

Contracted or Granular Kidney; Sclerosis of the Kidney; Gouty Kidney.

Definition.—Chronic diffuse inflammation of the kidneys, associated with overgrowth of intertubular connective tissue, and characterized by insidious development, increased urine of low specific gravity, albuminuria, which is usually slight and often intermittent, arteriosclerosis, and little tendency to dropsy.

Anatomically, there is marked increase in the connective tissue, with degeneration of the parenchymatous structures. The process is essentially atrophic, the atrophy probably beginning in the glomerules and tubules, and being followed by increase in the connective tissue. Three forms are described: (a) The pale granular kidney, to which reference has already been made—*secondary atrophic kidney;* (b) the contracted kidney, occurring as an independent affection—*primary atrophic kidney;* and (c) sclerosis of the kidneys, developing in connection with cardiovascular disease—*arteriosclerotic atrophy of the kidney.*

Etiology. — The pale granular kidney probably constitutes a later stage of the large white kidney, in which atrophic changes in the secreting structures have been followed by connective-tissue overgrowth. The primary contracted kidney is the result of a gradual degenerative process. Many of the cases occur in the absence of discoverable cause and may be due to premature senile involution. Hereditary influences, early arteriosclerosis, syphilis, and gout are causal factors. Chronic intoxications, especially alcohol and lead, play an important part in the etiology of sclerosis of the kidney. Habitual excesses at table, especially the eating of large quantities of meat, are particularly injurious. The arteriosclerotic form is associated with cardiac hypertrophy and sclerosis of the arterial walls. It is common in active, energetic men, who work hard and eat and drink too much. Habitual anxiety and worry, and inability to endure the stress of life favor the development of the vascular changes of which contracted kidney is the outcome. This form of renal disease is much more common after forty than in early life, and in men than in women. In this country arteriosclerosis develops at an earlier age among the Slav immigrants than in persons of other nationalities.

Symptoms.—The changes in the kidneys are insidious and frequently reach an advanced stage before they cause symptoms that attract attention. Acute uræmic symptoms frequently constitute the first marked manifestations of the disease. The earlier symptoms, headache, frequent micturition, and digestive disorders are often disregarded by the patient. More commonly the general health becomes gradually impaired. The patient is weak and breathless upon exertion. He rises frequently to pass urine, suffers from habitual headache, and complains of nausea and occasional vomiting.

The composition of the urine varies to some extent according to the variety of the renal atrophy. In the secondary form the quantity of the urine is less and the amount of albumin greater than in the primary form. The specific gravity is more nearly normal, that is, higher. There are various kinds of casts and a few red blood-corpuscles in the sediment. The tendency to dropsy is more marked. In the primary form the quantity of urine is much increased above normal and frequently reaches as much as four litres. This polyuria is the cause of an abnormal thirst. The color is pale yellow, the transparency clear, and the specific gravity low—1.005 to 1.012. A scanty sediment is deposited, in which are found a few hyaline and granular tube-casts, granular epithelial cells, leucocytes, and rarely red blood-corpuscles. The amount of albumin is small, especially after repose. It increases during attacks of intercurrent disease or with cardiac weakness, when the quantity of urine is diminished. It is characteristic of this form of nephritis that there are often albumin-free periods, especially in the early part of the day. The casts in some instances disappear even while the urine remains albuminous. The urinary solids are decreased, especially urea. Temporarily the urea may reach normal, and uric acid, phosphoric acid, the chlorides, and ammonia may approach normal. In the arteriosclerotic form polyuria is less common, the color of the urine is normal, the albumin is more abundant and more constant. and there are hyaline and granular casts, which may at times disappear.

Dropsy, so long as the power of the hypertrophied heart and the polyuria are maintained, is absent or scanty. Pretibial œdema may be noted, or slight puffiness of the ankles. The heart is hypertrophied, the left ventricle being first affected. The apex is displaced to the left and downward. The impulse is forcible and sometimes heaving. The aortic second sound is accentuated. There may be reduplication of the first sound. or an apex systolic murmur transmitted to the axilla. Toward the close the hypertrophy fails and the signs of dilatation are pronounced, together with lessened urine, increased albumin, and mounting dropsy. The pulse is hard and tense. The superficial arterial walls are thickened and incompressible. The radials can be rolled with the finger like a whip-cord upon the underlying bone. The temporals are prominent and tortuous. There is early and persistent increase of blood-pressure. Epistaxis is common and may be troublesome. Hemorrhages into the skin occur. Headache is a very common symptom. The symptoms in advanced cases are mostly due to the cardiovascular conditions, or to uræmia. To the former group are to be referred sudden œdema of the glottis or lungs, pleural effusion. and some cases of cardiac dyspnœa. This symptom may resemble asthma and is often troublesome at night. Cerebral hemorrhage is not uncommon. Fully 40 per cent. of the cases of apoplexy occur in persons suffering from contracted kidneys. To uræmia, either in its chronic or acute forms, must be referred certain of the cases of nocturnal dyspnœa—so-called renal asthma, Cheyne-Stokes respiration, nausea, vomiting, which is often uncontrollable, and diarrhœa. The complexion is usually pallid and muddy. Sweating is uncommon. The urea "frost" may be deposited after free perspiration. Pruritus and eczema are common. Muscular cramps occur. especially on waking in the morning. Albuminuric retinitis occurs more frequently than in any other form of nephritis. Visual troubles, in a large proportion of the cases, lead to the discovery of the actual condition. Sudden blindness without ophthalmoscopic findings—uræmic amaurosis—is sometimes observed. Hemorrhages beneath the conjunctivæ or into the eyelids occur. Tinnitus aurium or cerebri, vertigo, and nervous deafness are encountered.

Persons subject to chronic interstitial nephritis are peculiarly liable to severe intercurrent diseases. Bronchitis and pneumonia are common. Inflammatory affections of the serous membranes, as acute pleurisy and pericarditis, occur.

Diagnosis.—The early stages of chronic interstitial nephritis present no characteristic clinical phenomena. The anatomical condition may be advanced in cases unattended by evidences of ill health prior to the occurrence of the acute disease which has been the cause of death. The association of cardiac hypertrophy, sclerotic arteries, high pulse tension, accentuated aortic second sound, with copious urine of low specific gravity containing an inconstant trace of albumin and a few hyaline and granular casts, justifies a positive diagnosis. The urine should be repeatedly examined, specimens being taken at night and in the morning. The condition is often discovered accidentally in examination for life insurance, and sometimes overlooked under the same circumstances.

The diagnosis of small granular kidney cannot be positively made from the symptoms and urinary composition. It is rendered probable by a previous history of acute or chronic parenchymatous nephritis. Nor can the arteriosclerotic form be distinguished from the other varieties with certainty. The diagnosis becomes probable when the patient is past forty and has marked cardiac hypertrophy, hardened arteries, increased pulse tension, and uræmic symptoms, and particularly when the progress of the case is comparatively rapid.

Prognosis.—The outlook as regards recovery is hopeless. The disease is incurable. As regards prolongation of life and a fair degree of health, the prognosis is not altogether without encouragement. Many of the cases, under careful management and with a self-denying and regular manner of living, make slow progress and continue for years without passing into invalidism. The symptoms of chronic uræmia are danger signals; those of acute uræmia heralds of catastrophe. The signs of cardiac failure are usually the beginning of the end.

VI. PYELITIS.

Definition.—Inflammation of the pelvis of the kidney, due to direct bacterial infection by way of the blood or the ureters and lymphatics.

When the inflammation extends to the substance of the kidney the condition is designated pyelonephritis; when the entire organ is involved, pyonephrosis or renal abscess; the form due to tuberculosis is known as nephrophthisis.

Etiology. — Under ordinary conditions the kidneys are capable of eliminating, without damage to themselves, the pathogenic organisms reaching them by way of the blood stream or ureters and lymph channels in constitutional or local infection. When, however, their resistance to pathogenic influences is diminished by such general causes as prolonged malnutrition, anæmia, cold, or over-exertion, or by local conditions, as congestion, nephritis, pressure upon the kidney or ureter, twisting of the ureter in displacement or operation, infection occurs. Whether this takes place from the side of the blood current or from the urinary tract, the pelvis of the kidney is first affected—*pyelitis*. The colon bacillus, Bacillus proteus, streptococcus, and staphylococcus albus have been found in pure cultures. The tubercle bacillus is the cause of a special form of pyelitis. That form which occurs in gonorrhœa is caused not by the gonococcus but by associated pyogenic organisms.

Morbid Anatomy.—Pyelitis may be catarrhal or suppurative. The tuberculous form begins locally, the kidney gradually becoming infil-

[illegible] and softening, with ultimate [illegible] from the resorption of fluid elements [illegible]. It is associated with tuberculosis of [illegible] and testicles, or the ovaries or Fallopian tubes— [illegible]. Pyelitis due to local causes usually affects [illegible] conditions may involve one [illegible] following enlarged prostate [illegible] operation, is bilateral and [illegible]. The acute suppurative pyelonephritis [illegible]

Symptoms.—[illegible] accompanies the acute infections [illegible] can be recognized during life. [illegible] tenderness over the affected [illegible] sometimes acid, sometimes [illegible] transitional epithelial cells, and red [illegible]. Recurrent attacks occur in which [illegible] the patient has had clear urine and no [illegible] suddenly becomes turbid and smoky, and [illegible] the change being accompanied by pain [illegible] fuse sweating.

[illegible] varies in amount and may at [illegible]—[illegible] the blocking of the ureter [illegible] associated in some cases with the [illegible]. In acute pyelonephritis shreds of [illegible] together with tube-casts which may [illegible]. The urine is usually increased [illegible] proportion to the pus and blood [illegible] alkaline, but sometimes acid. [illegible]. It is commonly acid in the clear [illegible] and alkaline when there is an [illegible]

[illegible] type and associated with chills [illegible] are sometimes ague-like and [illegible] they closely simulate malaria. [illegible] assumes the hectic type. Chronic [illegible] anæmia, and progressive [illegible] abscess formation may [illegible] in some cases suggest enteric [illegible] criteria of that disease [illegible] group of cases familiar to [illegible] persistent pyuria, fairly good [illegible] this fact is important in [illegible] dryness of the [illegible] vomiting, [illegible] with dyspnœa— [illegible] sometimes constitute a terminal [illegible] Alkaline by ammoniacal [illegible] has been regarded as [illegible] products of decomposing [illegible] differs from uræmia in the absence of convulsions and retinitis. Paraplegia, variously ascribed to myelitis, peripheral neuritis, or reflex causes, is not uncommon.

The local swelling in the renal region varies in size from time to time. It may attain large size and give rise to signs of fluctuation in pyonephrosis —*abscess of the kidney.*

Diagnosis.—DIRECT.—The constant or intermittent presence of pus and blood in the urine, the occurrence of renal tissue, the absence of tube-casts, a tumor in the renal region inconstant in size or showing deep fluctuation in the absence of œdema, one-sided lumbar pain and tenderness, and chills, fever, sweating, wasting, and anæmia justify a positive diagnosis. In the absence of several of these clinical phenomena a provisional diagnosis may be made.

DIFFERENTIAL.—Tuberculous pyelonephritis may be diagnosticated when, with the above symptoms, tubercle bacilli are present in the purulent urine. In doubtful cases laboratory methods must be employed, especially the inoculation of guinea-pigs. Evidences of tuberculous disease in the urinary passages or genital organs are of diagnostic importance. The discrimination between pyelitis and pyelonephritis cannot always be made with precision. The presence of minute bits of renal tissue in the urine or a tender tumor in the region of the kidney would point to the latter condition. A deep fluctuating tumor points to renal abscess. Abscess within the capsule of the kidney is to be distinguished from perirenal abscess by the more circumscribed outline of the tumor, the absence of œdema, and the anamnesis, but the differential diagnosis is sometimes impracticable.

Cystitis and pyelitis are frequently associated. The polyuria, intermitting pyuria when present, the pain, tenderness, and tumor mass in one lumbar region, and the absence of frequent micturition and vesical tenesmus are in favor of the latter affection. The anamnesis is important. The cystoscope and catheterization of the ureters may be employed in doubtful cases.

Prognosis.—The cases associated with the acute febrile infections usually recover with the convalescence from the primary disease. The tuberculous form, when the kidney only is infected, may terminate in recovery, with cretaceous masses replacing more or less renal tissue. In abscess the outlook is unfavorable. Amyloid disease, fatal sepsis, or peritonitis from perforation may occur. The diagnosis assumes importance in view of the possibility of relief by surgery.

VII. PERINEPHRIC ABSCESS.

Paranephritis; Perirenal Abscess.

Definition. —Suppurative inflammation of the connective tissue surrounding the kidney.

Etiology.—Perinephric abscess may follow blows and injuries, the acute febrile infections, especially in children, inflammation of the kidney or ureter, perforation of the appendix or bowel, or result from a perforating empyema or spinal caries.

Morbid Anatomy.—The pus cavity is usually extensive, and the adjacent tissues œdematous. The accumulation is usually posterior, but may be anterior, to the kidney. It shows a strong tendency to burrow, and may perforate into the pleura, the bowel, the peritoneum, the bladder, or vagina, or follow the direction of gravitating spinal pus along the sheath of the psoas muscle or the iliac fascia, or finally the abscess if left to itself may burst externally.

Symptoms.—Pain in the region of the kidney, aggravated by pressure, or referred to the hip-joint, or inside of the thigh, and associated with retraction of the testicle, a limping gait, flexed thigh, stooping posture, and rigid spine, deep induration and œdema, and a tumor mass upon palpation between the last rib and the crest of the ilium, normal urine unless the primary pus depot is within the capsule of the kidney, and the constitutional evidences of pus make up the clinical picture.

Diagnosis.—The DIRECT DIAGNOSIS is justified by the above association of symptoms. Pus-free urine, and œdema overlying the tumor, and deep fluctuation are significant. It is not always possible to determine whether the infection comes from the kidney or some source outside of it. Here the history is important.

DIFFERENTIAL.—The pain and attitude may closely simulate hip-joint disease; but the essential symptoms are wanting, and the tumor and œdema in the region of the kidney are conclusive. When the abscess points in the inguinal region spinal caries must be excluded.

VIII. NEPHROLITHIASIS.

Renal Calculus; Renal Infarct.

Definition.—A condition characterized by the presence in the substance or pelvis of the kidney, or in the ureter, of concretions formed by the deposition of certain of the constituents of the urine normally held in solution.

RENAL INFARCT.—The deposition of urinary salts in the substance of the kidneys occurs under the following circumstances: In the new-born in the shape of uric acid crystalline masses in the tubules, and especially at the apices of the pyramids—*uric acid infarcts;* in chronic gout, sodium and ammonium urate in whitish linear deposits, chiefly in the pyramids—*sodium urate infarcts;* and dense white linear deposits of calcium phosphate or carbonate in the pyramids, chiefly in aged persons—*calcareous infarcts.*

RENAL CALCULUS.—The concretions which form in the pelvis and calices constitute, according to their size, sand, gravel, or stone. Renal sand consists of gritty particles of a size not too large to traverse the urinary passages without arrest. These minute calculi are frequently voided in the urine in considerable quantities at intervals for years, without causing symptoms. They form a characteristic coarse urinary sediment. The term gravel is applied to larger concretions, ranging in size from a canary seed to a pea, usually multiple, sometimes single, round and smooth, or irregular and rough, which form in the pelvis and calices, and passing into the ureter cause renal colic. Kidney stones, dendritic or coral calculi, are larger concretions, which attain dimensions in the pelvis which prevent their entering the ureter. Gradually increasing in size, they often form remarkable branching casts or moulds of the pelvis and calices. *Ureteral Calculi.*—The orifice of the ureter may be blocked by a large calculus, formed in the pelvis of the kidney, or a calculus may be arrested at any point in its course from the kidney to the bladder.

CHEMICAL COMPOSITION OF RENAL CALCULI.—These concretions do not represent mere precipitations of crystallizable or other solid constituents of the urine. They are composed in the first place of mixtures of various substances of this kind, arranged irregularly or in concentric layers, and in the second place they contain various proteid substances present as the result of inflammatory irritation of the tissues with which they are in contact, and finally bacteria are frequently found in the somewhat irregularly differentiated central nucleus. The principal varieties consist of: *Uric Acid and Urates.*—This is the common form and constitutes renal sand, small single calculi, and the large branching kidney stones. They are of a yellowish or brownish-red color, smooth or slightly irregular surface, and dense consistence. The large stones are composed of concentric strata and are very hard. They consist of uric acid and urates in varying proportions. In urinary sand the crystalline particles may consist of uric acid alone; in children calculi composed of urates occur. *Calcium Oxalate.*—Mulberry calculi are of a brownish or black color, rough and mammillated or pointed surface, and very hard. They are composed of oxalate of lime and uric acid, the former predominating. *Calcium Phosphate and Ammoniomagnesium Phosphate.*—Phosphatic calculi are composed of these salts, together with small quantities of calcium carbonate. They are of a whitish or pale fawn color, crystalline or chalk-like surface, and light consistence. They are sometimes friable. They are common, and the substances of which they are composed are deposited as an outer layer upon uric acid or oxalate calculi. *Cystin, Xanthin, and Indigo.*—Calculi chiefly composed of these substances are occasionally met with.

IMMEDIATE EFFECTS UPON THE KIDNEY.—In many cases the kidney manifests a remarkable tolerance for the slowly forming calculus. Single or multiple stones may be found post mortem without lesions of the kidney or a history of renal symptoms. Urinary sand or small round uric acid calculi are often passed at intervals by persons otherwise in good health. Large dendritic calculi cause induration and atrophy of the kidney substance. When infection takes place, calculous pyelitis and pyonephrosis result.

Etiology.—The subjects of uric acid and calcium oxalate crystals are usually adults of good constitution, active, and given to the pleasures of the table. Many of them are gouty. The urine is highly acid and contains uric acid in excess. Phosphatic calculi are met with in anæmic persons in poor health, often women with alkaline, sedimentary urine. In general, renal calculus is much more common in men than in women; in infancy and late adult life than in the middle periods. Dyspepsia, migraine, and a sedentary life are predisposing influences.

Symptoms.—Nephrolithiasis may be latent. Persons pass renal sand occasionally without local or general derangement of health. Sometimes a large dendritic calculus is unattended by symptoms. As a rule two sets of definite symptoms occur.

1. Symptoms of Stone in the Kidney.—These are: (a) *Pain*, which may be dull and continuous, or paroxysmal. It is usually referred to the lumbar region of the affected side, and sometimes radiates to the scrotum or glans penis. It may extend to the opposite side of the back. It may be aggravated by a misstep or sudden jarring of the body. Nephralgia is common in movable kidney, and occurs in the absence of renal calculus. (b) *Hæmaturia.*—Microscopic blood in the urine is very common. The amount of blood may cause smokiness, or, exceptionally, give a bright red tinge to the urine. Hæmaturia may be continuous, or it may recur at intervals, coming on spontaneously or after exertion, and ceasing upon rest. Bleeding is more common when the calculus is lodged in the ureter than when it remains in the pelvis of the kidney. (c) *Pyuria.*—Pus in the urine is the indication of calculous pyelitis, which may exist for a long time without causing serious symptoms, or may result in pyelonephritis or renal abscess. (d) *Septic Phenomena.*—There are cases in which paroxysms of intense pain, with chills, high fever,—104° to 106° F. (40°-41.1° C.),—and sweating occur at varying but irregular intervals. The urine becomes turbid and contains blood and transitional epithelium, but remains free from pus. These attacks, often regarded as malaria, are more like hepatic fever. Their recognition depends upon the positive evidence of nephrolithiasis and negative signs as regards malaria, *i.e.*, absence of blood parasites and failure of quinine. When calculus is established, irregular chills, fever and sweating, anæmia, wasting, pyuria, and smoky urine are commonly present.

2. Symptoms of Renal Colic.—The attack begins with dull pain in the renal region, which presently extends to the flank and toward the groin. This pain is continuous with excruciating exacerbations and points of focal intensity in the glans penis and testicle, which is retracted. It is accompanied by a most urgent continuous vesical tenesmus and desire to pass water, which is without result beyond a few drops of bloody urine, the voidance of which is attended with distressing scalding sensations. Rectal tenesmus and intense nausea and vomiting frequently add to the distress of the patient. Restlessness, anxiety, pallor, shivering, cold sweats, feeble pulse, and other collapse symptoms are usually also present. Fever may occur, 101°-103° F. (38.3°-39.5° C.). The attack ceases abruptly, with sensations of relief as the calculus passes into the bladder or returns into the pelvis of the kidney. Its duration varies from about an hour to a day or more. In the longer attacks there are periods of remission.

During the attacks an acute hydronephrosis develops upon the affected side, which is relieved upon the escape of the calculus into the bladder with the discharge of a large quantity of urine. A large amount of clear urine may be discharged from the sound kidney during the course of the attack. Renal colic does not always terminate in complete relief. The calculus may become impacted in the ureter and cause hydronephrosis and hæmaturia; in extremely rare cases it may rupture into the peritoneal cavity or the intestine, or may form an abscess and perforate the skin. Again, anuria may occur under the following circumstances: with a normal kidney on the opposite side, from functional arrest, in consequence of nervous irritation; with a previously diseased kidney from the same cause; with a single kidney. These conditions are rare.

Death occurs from uræmia, as a rule within ten or twelve days after complete anuria has set in, exceptionally not for two or three weeks.

Diagnosis.—DIRECT.—Other forms of paroxysmal pain may be mistaken for renal colic, but the seat of the pain, its definite extension toward the groin, with local intensification in the testicle and glans, together with hæmaturia, are characteristic. When direct evidence of the existence of a calculus, as previous attacks with the passage of one, or the voidance of one subsequent to the attack, can be secured, the diagnosis becomes certain. The recognition of a calculus in the pelvis of the kidney or in the ureter, or the grating of several calculi upon palpation, has been mentioned, but I have no personal knowledge of such signs.

DIFFERENTIAL.—Similar paroxysms of colicky pain are attributed to accumulation of renal sand, uric acid, or oxalates in the pelvis of the kidney. Dietl's crises, the nephritic crises of tabes, and clots of blood in renal hemorrhage, such as occur in cancer, may closely simulate renal colic. Biliary colic and intestinal colic rarely give rise to uncertainty. The differential diagnosis must be reached by a careful study of the individual case. A diagnosis by exclusion may be necessary.

The diagnosis of nephrolithiasis depends upon the foregoing symptoms, the occurrence of renal colic, and the shadows cast by the Röntgen rays. This means of diagnosis is imperatively required in order to determine the presence and position of calculi, and whether they exist in one or both kidneys in connection with the considerations of surgical intervention.

The differential diagnosis between renal and vesical calculus may sometimes be in doubt. In the latter the pain is usually bilateral, more common at the neck of the bladder. The tenesmus is continuous, with frequent micturition, and the sound will detect the presence of the stone.

Prognosis.—Many cases are recurrent. There is the ultimate danger of calculous pyelitis. The more serious accidents of renal colic and actual obstruction are rare. Many lives have been saved by surgical procedures.

IX. HYDRONEPHROSIS.

Definition.—A collection of urinary fluid in the pelvis and calices of the kidney due to obstruction of the ureter, forming a cyst by the distention and atrophy of the organ.

Etiology.—Hydronephrosis may be congenital owing to developmental defects, and may be of sufficient size to interfere with parturition. Much more commonly it is acquired. The obstruction may be in the urethra or in the bladder. In suddenly developing polyuria the normal ureter may be incapable of carrying off the excess of urine, and an acute hydronephrosis result. The ureter may be occluded by calculus, stricture following ulcer, or torsion or kinking in movable kidney. It may be blocked by neoplasms, particularly tubercle or cancer. Compression from without, by cicatricial adhesions or bands, or by abdominal or pelvic tumors, is a more common cause. Bilateral hydronephrosis may result from the implication of both ureters by any of the lesions just named. When, however, the obstruction is in the bladder, the hydronephrosis is almost always double. The common conditions are cancer, which may involve only one ureteral orifice, habitual urinary distention of the bladder in prostatic hypertrophy or stricture, and the extreme thickening and contraction of the bladder wall which accompanies these conditions.

The fluid accumulates, causing distention of the ureter above the obstruction, but especially of the pelvis of the kidney. Pyelitis may occur, but usually the kidney substance undergoes a simple, progressive atrophy, forming in extreme cases a large cyst, upon the inner surface of which traces of renal tissue may be found. As the secretion of urine diminishes mucus and serum accumulate until the cyst contains a pale yellow or straw-colored fluid, holding in solution traces of the urinary solids and frequently a little albumin. There may be turbidity from the presence of pus. The cyst thus formed may attain an enormous size and simulate ascites. Cardiac hypertrophy is frequently associated with hydronephrosis. Exceptionally complete occlusion of the ureter is followed by atrophy of the kidney without dilatation.

Symptoms.—The urinary conditions are by no means constant. In one-sided hydronephrosis with complete occlusion, the urine from the vicariously acting opposite kidney may be normal in quantity and composition. When the condition is bilateral, oliguria and, ultimately, complete anuria result and death occurs with uræmic symptoms. Intermittent hydronephrosis arises when a valve-like obstruction yields to the pressure of the accumulated fluid, or the twisted or kinked ureter is restored to its natural condition. Under these circumstances there is a sudden large discharge of clear urinary fluid with the subsidence of the tumor. When the obstruction recurs the cystic tumor reappears, to vanish again when the obstruction is relieved—*flush-tank symptom.* Intermittent hydronephrosis may continue for years. It is unilateral, commonly associated with movable kidney, and usually occurs in women. In cases in which pyelitis exists the urine may be normal when the tumor is forming and turbid from the presence of mucus, pus, or blood as the tumor subsides.

Physical Signs.—When the tumor attains sufficient size it may be recognized upon palpation or inspection. If unilateral and of moderate size, it occupies the renal region; when large it may simulate ovarian or other cysts; an enormous hydronephrosis may be mistaken for peritoneal effusion. It may also simulate solid tumors of the kidney or enlarged retroperitoneal glands. The physical signs of hydronephrosis and a large renal abscess are the same. The latter is, however, usually attended by septic phenomena. They consist of dulness in the renal region with deep fluctuation. When the tumor presents anteriorly, the ascending colon on the right side, the descending colon on the left, usually yields an oblique band of tympany.

Diagnosis.—Direct.—Hydronephrosis can only be recognized when it gives rise to a fluctuating tumor. Even then it is readily confounded with other cysts. The greatest difficulties arise when the condition involves a displaced kidney; the least, in the intermittent form. The relation of the tympanitic colon to the tumor is important. The sac may be aspirated. The fluid obtained is of low specific gravity, commonly clear, and contains urea and urinary salts, and transitional epithelium. In very old cases with extreme atrophy of the kidney substance, the fluid may contain nothing characteristic.

DIFFERENTIAL.—*Ovarian Cysts.*—Large hydronephrosis is frequently mistaken for an ovarian tumor. The latter may be differentiated by its mobility, except in the case of hydronephrosis involving a floating kidney. Hydronephrosis has its starting-point in the renal region, whereas ovarian tumors spring from the pelvis, as can be determined by bimanual examination externally, or with the fingers of one hand in the vagina, later in the rectum, and the other hand upon the abdomen. By this mode of examination the relation of the uterine appendages and the presence or absence of a pedicle can be determined. In ovarian disease disturbances of function—for example, amenorrhœa—are not constant, just as in hydronephrosis the condition of the urine varies in different cases. *Renal Cysts.*—The differentiation is mostly impossible. In the new-born cystic kidneys and hydronephrosis may present the same signs. When the abdominal wall is congenitally defective the dilated bladder and ureters may be readily observed. Echinococcus cysts of the kidney may be suspected if daughter cysts or hooklets are found in the urine or in non-albuminous fluid obtained by exploratory puncture. But a positive diagnosis of echinococcus cyst of the kidney can only be made when it is possible to exclude such a condition perforating into the urinary passages. The hydatid thrill is by no means constant. *Mesenteric Cysts.*—These tumors vary in size from the closed fist to a cocoanut and are commonly situated in the lower right quadrant of the abdomen. They are freely movable, of oval contour, smooth surface, elastic, and fluctuating. Paroxysms of pain and vomiting are often associated with their presence. *Ascites.*—The differential diagnosis may be difficult in bilateral hydronephrosis. Movable dulness upon change of position, the absence of tympany in the flanks, and the character of the fluid in peritoneal effusion are important.

Prognosis.—Unilateral hydronephrosis when quiescent constitutes a benign tumor, but the prognosis must in all cases be guarded. The condition acquires importance by progressive increase in size, the danger of rupture into the peritoneum or lung, pyonephrosis, and the possibility of the blocking of the other ureter, with anuria followed by uræmia. The fluid may discharge by way of the ureter and never reaccumulate. Intermittent hydronephrosis may cause little inconvenience and finally undergo spontaneous cure. When double, the condition is far more serious, and the outlook depends upon the possibility of relieving the condition which causes the obstruction—urethral stricture, prostatic enlargement, tumor-pressure. The cases due to malignant disease are without hope.

X. CYSTS OF THE KIDNEY.

The multiple small cysts due to obstruction of uriniferous tubules in chronic nephritis, and larger solitary cysts sometimes seen post mortem in kidneys otherwise normal, do not cause symptoms or signs by which they can be recognized during life. Rare forms are combined cystic disease of the liver and kidneys, and paranephric cysts. Of greater importance clinically are: *Congenital Cystic Kidneys.*—The organs are enormously enlarged and consist of a mass of round or oblong cysts, varying in diameter from 0.5 to 3 centimetres, with kidney tissue sufficient to discharge the renal function distributed in the interspaces. The fluid contained in these cysts varies in color from clear to opaque reddish or black, and in consistence from limpid to colloid. It contains albumin and other proteids, cholesterin plates, crystals of hæmatoidin and ammoniomagnesium phosphate, and fat droplets.

Etiology.—Cystic kidneys occur in the fœtus and may be the cause of dystocia. They are regarded as developmental defects. They are sometimes associated with hydrocephalus, defects of the bladder, and malformations of the extremities. The cystic condition increases with age and is encountered in young adults, the tumors often attaining great size. The condition is usually bilateral, though unilateral cases have been observed. The condition has been noted in several members of a family.

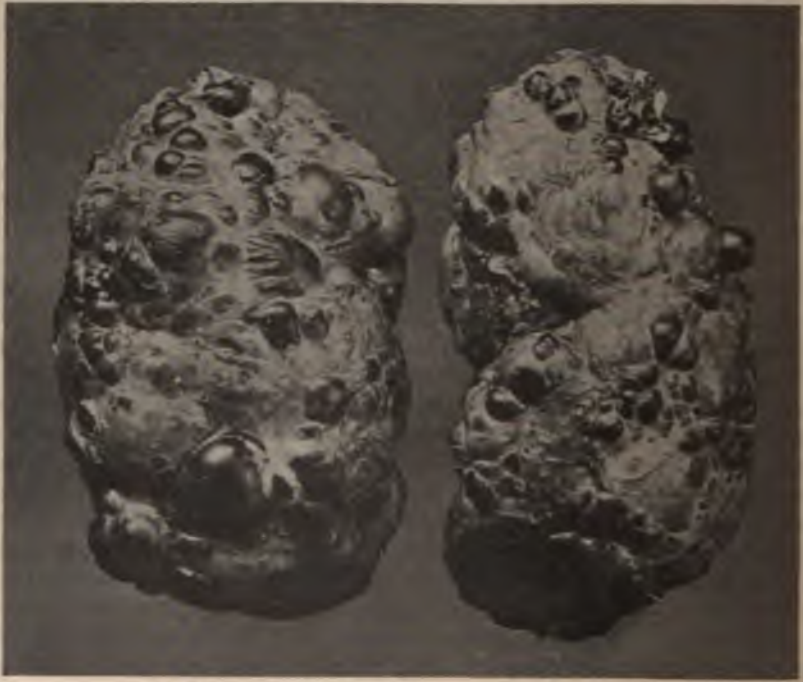

FIG. 326.—Congenital cystic kidneys.—German Hospital.

Symptoms.—The urine is abundant, of low specific gravity, and contains albumin in small amounts and hyaline and granular casts. Recurrent hæmaturia may be noted. There is pallor, a muddy skin, and not rarely diffuse cutaneous pigmentation.

Physical Signs.—The physical examination reveals the evidences of cardiovascular disease—enlargement of the heart, accentuated aortic second sound, hardening of the arteries—and the signs of double tumor in the renal region extending forward, elastic but non-fluctuating, and, when distinctly palpable through the belly walls, irregularly nodular. I have observed these signs in a unilateral case in which the diagnosis was confirmed by operation.

The symptoms, urinary conditions, and general physical signs are those of chronic nephritis; the local signs, those of renal tumors.

The **diagnosis** rests upon the association of the foregoing symptoms and signs. The recognition of the condition derives its importance from the fact that, as a rule, to which there are very few exceptions, surgical intervention is positively contraindicated.

Prognosis.—The outlook is not favorable. Death results in many cases before or directly after birth. If the patient survive infancy, death occurs before thirty from cardiovascular changes or uræmia.

XI. TUMORS OF THE KIDNEY.

Solid tumors of the kidney are benign and malignant. Benign tumors comprise fibroma, lipoma, myxoma, angioma, gumma, and adenoma. They do not give rise to symptoms referable to the kidneys, nor do they, as a rule, attain such a size as to respond to the methods of physical examination—inspection, palpation. They are, however, of anatomical rather than clinical interest.

Malignant neoplasms—carcinoma, sarcoma—are primary or secondary. They are accompanied by distinct symptoms, both general and urinary, and frequently grow to a large size.

RENAL CARCINOMA is a rare lesion and when small may not reveal its presence during life. Larger cancers cause conspicuous abdominal tumors, usually immovable upon deep respiration or palpation, occupying the upper quadrants of the abdomen in relation with the liver or spleen, obliquely traversed by the ascending or descending colon as the case may be, and of an irregular surface and consistence.

SARCOMA OF THE KIDNEY is much more common. It constitutes a frequent form of abdominal tumor among children, especially the primary variety. The growth reaches a large size and may greatly distend the abdomen. It is almost always unilateral and presents objective characters similar to those of carcinoma, save that it is commonly less nodular and softer, in some cases even fluctuating over extensive or limited areas.

Hypernephroma, which may develop from suprarenal tissue exceptionally upon the kidney, as a rule within its substance, is a very common form of renal tumor. This neoplasm may be found upon post-mortem examination as small tumors within the cortex, or it may form large tumors having the characters of malignant growths of the kidneys in general. Metastases occur.

Symptoms of Malignant Tumors of the Kidney.—Pain, usually of a dull, dragging character and referred to the flank upon the affected side, is common. It may radiate to the thigh or extend to the pleura. There are large growths in which pain does not occur. Emaciation is commonly progressive and rapid, and a high grade of cachexia is usual. There are cases, however, especially of sarcoma, in which the nutrition and strength are fairly well maintained. Pressure upon the abdominal veins may cause venous distention and œdema of one or both lower extremities, and ascites. Pressure upon nerve-trunks may cause pain or pressure neuritis in the distribution of the intercostals or the crural nerves. Metastatic growths occur in various organs, and, in particular, in the lungs. They may occasion special symptoms. If the spinal cord is involved paraplegia may result.

The urine contains blood at some time in the course of a majority of the cases. The hæmaturia is intermittent, the urine being practically normal during the intervals. If the growth involves the pelvis the urine may be turbid and albuminous. The blood is sometimes fluid, sometimes clotted. It is characteristic of the hæmaturia of malignant disease of the kidney that blood-casts of the ureter, sometimes of the pelvis of the kidney, are occasionally passed. The passage of these clots is attended with intense pain like that of renal colic. In rare instances cancer elements have been discovered in the urine.

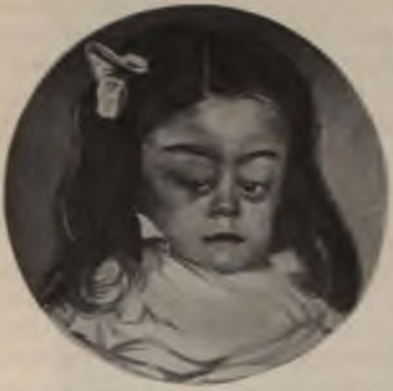

FIG. 327.—Suprarenal sarcoma; metastases in the skull.—Robert Hutchinson.

PHYSICAL SIGNS.—If the growth involves a floating kidney, the organ may remain for some time movable and be found in the iliac fossa. When the kidney is affected *in situ* the tumor is stationary, unilateral, and develops from the upper and posterior region of the abdomen. Other attributes have been mentioned. Bimanual palpation is important. The relation of the colon as indicated by the tympanitic percussion sign which it yields is of great value in the differential diagnosis.

Diagnosis.—DIRECT.—The diagnosis of malignant tumor of the kidney in well-developed cases may be readily made when all of the above symptoms and signs enter into the symptom-complex. In proportion as several of them are absent the diagnosis becomes uncertain. The nature of the neoplasm cannot in all cases be positively determined. Carcinoma is more common in adult life, attended with a greater tendency to wasting and cachexia, and to hæmaturia. Sarcoma is far more common under ten years of age; it frequently runs its course without hæmaturia, and may be attended with little disturbance of the general health. If primary or metastatic growths accessible to direct examination are present, the determination of their character settles any diagnostic uncertainty as to the nature of the renal tumor. The examination of tissue elements found in the urine, or obtained by exploratory puncture, may yield conclusive results. The recent investigations of Kelly and others render it probable that a large proportion of the cases described as primary carcinoma and sarcoma of the kidneys are hypernephromata.

DIFFERENTIAL.—*Tumors of the Pelvic Organs.*—Tumors of the kidney are frequently mistaken for ovarian tumors. The greater mobility of the latter, their development from the pelvis, their relation to the uterus and the presence of a pedicle as determined by vaginal examination, and derangement of sexual functions, as menstruation, are significant. The presence or absence of intermittent hæmaturia is important. Tumors of the uterus are less likely to present diagnostic difficulties. *Retroperitoneal sarcoma*—Lobstein's cancer—may give rise to diagnostic uncertainty, particularly in children. Both conditions form very large tumors. The disease of the lymphatic glands is more central and less movable. It may extend to the kidneys. In the advanced stages the diagnosis cannot always be made.

The careful application of the methods of physical diagnosis in connection with the facts relative to tumors of the kidneys renders their discrimination from tumors of the liver and spleen an easy matter.

Prognosis.—The outlook is unfavorable. The extirpation of a small growth has in a low percentage of the cases been followed by recovery.

XII.

THE DIAGNOSIS OF DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

DISEASES OF THE BLOOD.

I. ANÆMIA.

Definition.—A morbid condition of the blood characterized by a diminution of the erythrocytes or the hæmoglobin, or of both.

i. General Considerations.

PALLOR of the skin and mucous membranes is in a high degree suggestive of anæmia. For a positive diagnosis, however, a systematic microscopic examination is essential. By this method only can the existence of anæmia in every case be recognized and the nature of the anæmia positively determined.

PSEUDO-ANÆMIA.—There are individuals in whom marked pallor of the skin and mucous membranes suggests anæmia, but whose blood shows, upon microscopical examination, a normal number of erythrocytes and a normal percentage of hæmoglobin. Such persons usually owe their pallor to one or another of the following conditions: (1) hereditary peculiarities of the integuments, among which is an opaque but non-pigmented skin deficient in capillary network; (2) prolonged life in tropical regions, the so-called tropical anæmia; (3) chronic nephritis, arteriosclerosis, certain cases of cardiac disease, neurasthenia, and tuberculosis; (4) habitual indoor life, as in prisoners,—the so-called "prison pallor,"—workers in sweat-shops, miners, and others whose occupations and circumstances deprive them of sunlight and fresh air.

There are transitory conditions in which pallor of the skin and mucous surfaces is ischæmic rather than anæmic, as syncope, rigor, chilling of the surface, fatigue, pain, and sudden intense emotions, especially fear. The volume of the blood and its corpuscular values are unchanged, but it retires from the surface and accumulates in the viscera and deeper tissues of the body. The tidal blood flows and ebbs, not rhythmically, but under the influences of various physiological and pathological influences.

LOCAL ANÆMIA.—The distribution of the blood mass is controlled by the arteries, which contract or dilate under the influence of the central or peripheral vasomotor ganglia. Hence afflux and deflux, physiological within limits; pathological in excess. In one territory congestion; in another anæmia. Cerebral anæmia with faintness or syncope results from sudden dilatation of the mesenteric vessels, such as is caused by intense emotion, pain, the rapid removal of pressure, as in the abrupt change to the upright posture, or a copious stool in advanced aortic incompetence, or the evacuation of a large ascites. Chronic anæmia of the central nervous system may be the cause of many of the vague symptoms in cardiac disease and enteroptosis. Local anæmias due to spasm of peripheral vessels, such as is seen in Raynaud's disease, causing asphyxia of the extremities, may affect visceral vessels, causing functional derangements, or circumscribed areas of brain tissue, and give rise to transitory palsies, aphasia, or hemicrania.

GENERAL ANÆMIA.—The general anæmias are primary, essential or cytogenic, and secondary or symptomatic.

II. Primary Anæmia.

The blood-making organs are at fault. The etiology is obscure. The essential lesions involve the blood as a tissue. Clinical phenomena manifest in other structures are secondary to changes in the blood. This group comprises chlorosis, pernicious anæmia, and splenic anæmia.

a CHLOROSIS.

Definition.—Anæmia of undetermined cause, common in females at or shortly after the age of puberty, and characterized by a peculiar greenish-yellow pallor of the skin, constipation, breathlessness upon exertion, and marked relative diminution of the hæmoglobin.

Etiology.—PREDISPOSING INFLUENCES.—Sex is paramount. Chlorosis occurs only in females. The period of onset corresponds to that of puberty, and varies from about the [illegible] to the seventeenth year. Earlier [illegible] it is due to recurrences, which are [illegible] occur in successive generations, [illegible] suffered from chlorosis are often [illegible] often manifest the disease. [illegible] hypoplasia of the aorta and [illegible] walks of life, but is much more [illegible] young girls in factory towns and [illegible] domestic servants. Among [illegible] of fresh air and sunshine, [illegible] important predisposing influences. [illegible] and phlegmatic. Sometimes emotional and nervous. The disease has, upon insufficient grounds, been regarded as a neurosis, attributed to copræmia from constipation and to the wearing of the corset.

THE EXCITING CAUSE.—The actual cause of chlorosis is unknown. It is apparently due to a functional default of the blood-making organs—defect of hæmogenesis—incident to the functional maturity of the reproductive organs.

Symptoms.—Pallor is marked, and the skin has a faint yellowish-green tinge to which is due the designation chlorosis, or green sickness. The conjunctivæ are faintly bluish and the mucous membranes of the mouth and lips very pale. In some cases the normal pigmentation about the folds of the joints is slightly intensified. The subcutaneous fat is frequently increased and there is an increased turgor of the integumentary tissues. There may be actual œdema of the face and ankles. There are rare cases in which the cheeks have a reddish color, especially upon exertion or during excitement—*chlorosis florida, chlorosis rubra.* Gastro-intestinal symptoms are common and often prominent. The appetite is capricious. The school-girl's fondness for pickles and bits of chalk is well known. Hyperacidity is often present and associated with epigastric distress. Dilatation of the stomach, gastroptosis, and movable right kidney are frequently present. Constipation is a common and troublesome condition. Dyspnœa, palpitation, and vertigo occur upon exertion, as in the rapid ascent of a flight of stairs. The heart is frequently dilated and the apex displaced slightly to the left. A systolic murmur in the mitral area may be the sign of relative insufficiency. Much more common are systolic murmurs at the base, particularly in the pulmonary area. Distinct pulsation in the second left intercostal space is not uncommon. Diastolic murmurs are infrequent. A distinct, loud, continuous, venous hum may be heard over the jugular vein on the right side—*nun's murmur, bruit de diable, humming-top murmur.* Pulsation may sometimes be seen in the veins of the neck; less frequently in the peripheral veins. Thrombosis may occur in the cerebral sinuses or in the left femoral vein. In the latter event there is danger of pulmonary infarct. Slight enlargement of the thyroid body is not uncommon. It may be associated with Joffroy's sign. Amenorrhœa and dysmenorrhœa are common. Hysterical manifestations occur in a large proportion of the cases. Headache, coldness of the extremties, and mental depression are prominent symptoms. Moderate rises of emperature are occasionally observed.

THE BLOOD.—The droplet is pale and flows freely from the puncture. t is characterized by transparency and fluidity as contrasted with normal lood. The coagulation period is short and the specific gravity decreased. he alkalinity of the blood, according to most observers, remains normal. here is marked absolute decrease in the hæmoglobin; and a high degree of minution in hæmoglobin relative to the reduction in the number of ythrocytes, as manifested by low color indices, constitutes a charactertic phenomenon of chlorotic blood. Microscopically the erythrocytes are oderately decreased, usually to about 4,000,000 per cubic millimetre. ey may fall as low as 3,000,000 or even 2,000,000 in severe cases. There a slight general diminution in their average diameter. Nucleated forms

—*normoblasts*—are occasionally present. Poikilocytosis is common and may be marked in severe cases. Microcytosis also occurs in the graver cases. Polychromatophilia is rare. The leucocytes are usually normal or slightly increased. There is relative increase in the number of lymphocytes. Small percentages of myelocytes occur in severe cases. Eosinophiles are absent in the majority of the cases.

Pseudochlorosis.—This term has been applied to a rare condition in which the characteristic symptom-complex is present, but no marked change in the blood can be discovered upon ordinary clinical examination. It has been assumed that the actual condition of the blood as regards the relation between the hæmoglobin and erythrocytes is masked by a diminution in the volume of plasma.

Diagnosis.—The DIRECT DIAGNOSIS of chlorosis is unattended with difficulty. The pallor, the pearly or bluish conjunctivæ, the preservation of subcutaneous fat occurring in a girl at or shortly after puberty constitute a characteristic clinical picture. Headache, breathlessness upon exertion, and gastro-intestinal symptoms, especially constipation, are of diagnostic value, particularly in the absence of visceral disease attended by anæmia and dropsy, as disease of the heart, or forms of nephritis, or chronic infections, as tuberculosis, syphilis, or malaria. In the majority of cases the characters of the blood differentiate chlorosis from other forms of anæmia; but the fact is not to be overlooked that there are cases of secondary anæmia, particularly in the above-mentioned organic and infectious diseases, in which changes in the blood closely corresponding to those of chlorosis occur.

DIFFERENTIAL DIAGNOSIS.—*Cardiac Disease.*—The dyspnœa and palpitation upon exertion frequently suggest valvular disease of the heart. The anamnesis is important. A history of rheumatic or scarlet fever, followed by such symptoms becoming progressively more severe; distinct cardiac enlargement; murmurs having characteristic points of maximum intensity, definite lines of propagation, and constant relations to the revolution of the heart; the absence of a venous hum, are in favor of disease of the heart. The sex and age of the patient are highly important. *Renal Disease.*—General pallor and some degree of anasarca, as shown in puffiness of the face and œdema of the feet, may be suggestive of nephritis—a diagnosis at once negatived by the absence of albumin and casts. The possibility of transient albuminuria—*albuminuria of adolescence*—is to be borne in mind. The intermittent nature of this form of albuminuria and its tendency to disappear upon rest are significant. *Tuberculosis.*—The pallor of the early stages of pulmonary tuberculosis in a young girl may simulate chlorosis. Cough, wasting, positive physical signs, rapid pulse, and a slight rise of temperature recurring about the same time every day, increased by exercise and at the time of menstruation, point to phthisis. The examination of the blood is essential. The deficiency of the hæmoglobin in chlorosis may be apparent when a drop of blood is allowed to fall upon a piece of white blotting paper or a handkerchief, the blood of a healthy person being used for contrast. *Malaria.*—The character of the febrile paroxysm and the presence of malarial parasites in the blood are conclusive. *Syphilis.*—The rapid anæmia and the fever of secondary syphilis may give rise to doubts as to the diagnosis. The macular syphilide, adenopathy and mucous patches are diagnostic. When a chlorotic girl contracts syphilis the question of diagnosis becomes complicated.

Prognosis.—The prognosis is favorable. The majority of cases recover under treatment in the course of a few weeks. The tendency to repeated attacks persists in some cases for several years. The influence of pregnancy and lactation in arresting this tendency is usually positive. The administration of iron in proper doses appears in a majority of cases to exert a specific curative influence—a fact that might be of diagnostic importance in a doubtful case.

(b) PERNICIOUS ANÆMIA.

Idiopathic or Progressive Anœmia.

Definition.—Severe anæmia developing either idiopathically or in the absence of discoverable adequate cause, and characterized by a progressively unfavorable course, maintenance of body weight, and constant changes in the blood, namely, great reduction in the number of erythrocytes, megalocytosis, microcytosis, poikilocytosis, the presence of erythroblasts, and relative increase in hæmoglobin.

Etiology.—Clinically several different groups of cases may be recognized which present the symptoms of progressive pernicious anæmia, but are due to different causes. A majority of the cases correspond to the description of Addison, and arise in the absence of the usual causes of anæmia. Intense progressive anæmia, presenting all the clinical features of pernicious anæmia, is occasionally encountered, (a) in child-bearing women, beginning either during pregnancy or after parturition; (b) in gastro-intestinal diseases, particularly atrophy of the stomach; and (c) in certain forms of intestinal parasitism. especially uncinariasis and the presence of the Bothriocephalus latus. The blood picture seen in the anæmia which follows certain cases of nitrobenzol poisoning is similar to that of pernicious anæmia. Cases have been observed in every quarter of the globe. The onset is gradual and wholly independent of seasonal influences. The exacerbations which follow remissions under treatment sometimes occur in the spring of the year. The onset of the disease is most common in late middle life. It has been observed at all ages. Excluding the cases which begin during pregnancy and lactation, it would appear that males are much more frequently affected than females. Rare cases have been encountered in parents and children—a fact which does not warrant the assumption that the tendency to the disease is transmitted from the parent to the offspring. Severe nervous or mental shock and prolonged intense depressing emotions have in many instances been followed by the onset of the disease. The researches of William Hunter lend support to the conclusion that this form of anæmia is due to chronic septic infection associated with lesions of the gums or mouth, and gastric and intestinal sepsis, which lead to hæmolysis.

Symptoms.—The onset is insidious, and the symptoms are those of a profound and progressive anæmia. Pallor of the skin and visible mucous membranes, languor, breathlessness upon slight exertion or emotional excitement, flabbiness of the tissues associated with remarkable preservation of the subcutaneous fat and slight puffiness about the ankles, extreme debility, make up the clinical picture. In a majority of the cases there is irregular fever of moderate intensity. Cardiovascular symptoms consist of faintness, dyspnœa and palpitation upon exertion, a full, soft, and compressible pulse, visible pulsation of the superficially placed arteries, and hæmic murmurs. The pulse not rarely resembles the water-hammer pulse of aortic regurgitation,—*Corrigan's pulse*,—a resemblance frequently increased by the presence of capillary and a penetrating venous pulsation. There is a marked tendency to hemorrhage from the mucous membranes and into the skin. Retinal hemorrhages are common as in other grave anæmias. *Gastro-intestinal Symptoms.*—The appetite fails, and in advanced cases anorexia is complete. The mouth is dry, the tongue furred, often sore, the gums are ulcerated, and the breath is offensive. Nausea and vomiting are common. Attacks of diarrhœa occur without apparent cause. The urine is abundant and usually of low specific gravity; sometimes pale, sometimes of a deep sherry color, due to the presence of urobilin. The skin is blanched, smooth and waxy in appearance. It is commonly intensely pale, frequently of a faint lemon color, especially marked upon the hands, sometimes subicteroid, and less commonly pigmented as in Addison's disease. The pigment may be more or less uniformly distributed, or deposited in irregular patches. It is sometimes associated with patches of vitiligo. In some cases the cutaneous pigmentation follows the prolonged administration of arsenic.

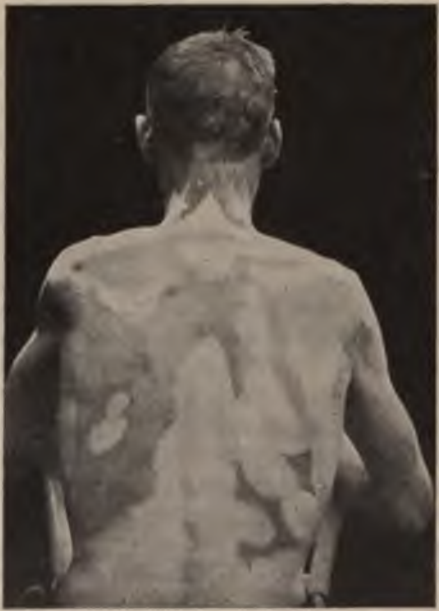

FIG. 328.—Pigmentation of the skin in a case of pernicious anæmia.—Packard.

Spinal symptoms may be encountered as the result of degeneration involving the posterior and lateral columns, and in some cases extending beyond these tracts to the anterior part of the cord. They may appear earlier than the blood changes, but more commonly not until the disease is well advanced. They consist of numbness and tingling in the legs and feet, weakness, and in some cases severe pain. The reflexes are increased. Later similar symptoms may involve the upper extremities. There may be marked ataxia, with disturbance of the functions of the bladder and rectum. After a time loss of sensation may occur, with flaccidity and abolition of the reflexes.

THE BLOOD.—There is absolute diminution of the hæmoglobin, which is, however, increased relatively to the number of red cells, so that the color index is high. The erythrocytes show a great numerical decrease, often to 1,000,000, or in terminal states to 500,000 per cubic millimetre. Erythroblasts are constant, the common form being megaloblastic. Poikilocytosis is constant and marked. Megalocytes and microcytes occur, the former being more common and more marked. Polychromatophilia is found in many of the erythrocytes, both non-nucleated and nucleated. The leucocytes are usually decreased, often markedly so. A relative increase in the lymphocytes is common. Myelocytes are usually present. The eosinophiles are almost always decreased in number and frequently absent altogether. The number of blood-plaques is variable. Blood crises (von Noorden) are characterized by the appearance in the blood of large numbers of nucleated red blood-corpuscles, very often in successive crops, and are usually of sudden onset and brief duration, sometimes lasting but a few hours. These crises are common in severe anæmia following hemorrhage and in chlorosis, and not rare in some forms of leukæmia and in pernicious anæmia, and are usually followed by periods of temporary improvement in the blood count. The blood crises of pernicious anæmia are more commonly of the megaloblastic type and are frequently followed by the death of the patient. When of the normoblastic type they may be followed by an actual increase in the erythrocytes.

Aplastic Anæmia.—This term has been applied to a limited group of cases presenting the symptoms of pernicious anæmia but characterized by atrophy of the erythroblastic tissue in the bone-marrow. Clinical differences between this variety and the ordinary form of pernicious anæmia are found in its earlier occurrence, a majority of the cases having occurred before the thirty-fifth year of life; its greater frequency among females than males; its rapid course, unattended by remissions and usually terminating within a period measured by months; a greater tendency to hemorrhage. Differences in the blood picture consist in a lower color index than in the ordinary form; a marked increase in the percentage of lymphocytes; the absence, as a rule, of erythroblasts; the absence or comparative infrequency of poikilocytosis, anisocytosis (differences in the size of the erythrocytes), polychromatophilia; and the great diminution of the blood-plates. Pathologically the most constant and striking change is manifest in the marrow of the long bones, from which the erythroblastic tissue has disappeared, leaving the medullary cavities filled with fat. That this change has occurred in all the bones has not as yet been demonstrated in any case. The femur is used as a standard, and if its marrow is yellow and homogeneous throughout, the form of anæmia may be regarded as aplastic. *Diagnosis.*—The direct diagnosis cannot be made *intra vitam.* It is rendered probable by the concurrence of the above clinical symptoms and hæmatological findings, but finally rests upon the post-mortem examination of the marrow of the bones.

Diagnosis.—The direct diagnosis of pernicious anæmia can only be made by a microscopical examination of the blood. In general practice the true nature of the disease is not often suspected in its earlier course and rarely recognized after it has made some progress. Even the blood changes are not at all times present in every case in the beginning. A severe anæmia insidiously arising in the absence of any obvious cause, pursuing an unevenly progressive course but little influenced by treatment; preservation of the subcutaneous fat and body weight to a remarkable degree; a blanched, smooth, and waxy appearance of the surface which has a faint lemon-yellow tint; extreme languor and breathlessness upon exertion; a tendency to hemorrhage into the skin or from mucous surfaces; retinal hemorrhage; gastric symptoms; and the occurrence of febrile paroxysms of moderate intensity constitute a symptom-complex which is highly characteristic. A blood picture showing the association of oligocythæmia of high grade, falling in many cases below 1,000,000 per cubic millimetre; erythroblasts mostly of the megaloblastic type; poikilocytosis; great variation in the size of the erythrocytes; a high color index; and leukopenia render the diagnosis positive.

Differential.—1. *Grave secondary anæmias*, such as occur after copious hemorrhages (especially a prolonged habitual blood loss), visceral cancer (especially carcinoma ventriculi), and in advanced syphilis, often present clinical symptoms precisely similar to those of pernicious anæmia. The anamnesis is important. As a rule, an obvious cause for the anæmia may be discovered. There are, however, cases in which malignant disease cannot be located, or the history of syphilis is obscure. The actual pathological condition then rests upon the examination of the blood. The following points are of diagnostic importance: In secondary anæmia, (a) the oligocythæmia is less marked, the count rarely falling below 1,000,000 per cubic millimetre; (b) the color index is lower; (c) leucocytosis is often present; (d) megalocytosis does not occur. The fault does not primarily involve the hæmatopoietic organs. The blood changes result from, and are secondary to, constitutional diseases, as certain of the acute and chronic infections and intoxications, diabetes, parasitism and nutritional disorders, local anatomical lesions which seriously interfere with the functions of important viscera,—as the heart, lungs, the organs of digestion, or the kidneys,—malignant disease, or to hemorrhage. The anæmia is due to deficient blood formation—hæmogenesis; excessive blood destruction—hæmolysis; or the association of these two processes. Severe secondary anæmia, when long continued, may exhaust the function of the blood-making organs in such a manner as to be converted into primary anæmia. 2. *Chlorosis*.—There are often clinical phenomena present which suggest pernicious anæmia, namely, preservation of subcutaneous fat, smooth, waxy, and faintly greenish-yellow tint of the skin, pallor of mucous surfaces and pearly tint of sclera, breathlessness, and languor; but the blood examination shows essential differences. The following points are to be considered: (a) In chlorosis we have to do with a hæmoglobin rather than a corpuscular anæmia; hence a low color index, the reverse of the condition in pernicious anæmia; (b) there is a general diminution in the diameter of the erythrocytes; (c) megaloblasts may occasionally be encountered, but are never present in great numbers—nucleated red corpuscles when seen are almost always normoblasts; (d) leukopenia of high grade is not common; (e) myelocytes may be observed in small numbers in both diseases, but are much less common in chlorosis. 3. *Bothriocephalus Anæmia*.—The therapeutic test yields positive results. Metabolic products of the intestinal parasite have been thought to possess hæmolytic properties. Its expulsion may be promptly followed by an improvement in the general health and the restoration of the blood to its normal condition, the megaloblasts and oligocythæmia disappearing and the color index falling to the usual range of health. 4. *Various Affections.*—In the absence of a blood examination the lemon-yellow tint of the skin may suggest jaundice, from which pernicious anæmia is to be differentiated by the pearly sclera, the fact that bile pigments are not present in the urine, and the absence of definite signs and symptoms indicating disease of the liver or bile passages; the anæmia, puffiness of the face, and swelling of the ankles may simulate nephritis, especially if, as is sometimes the case, albumin is found in the urine, a view not sustained by the results of close analysis of the urine and the history of the case; palpitation, dyspnœa, and the condition of the arteries point to cardiac disease, but the anamnesis and physical signs lend little support to such a diagnosis; finally, nervous symptoms, such as numbness of the legs and feet, less commonly of the hands, pain, sometimes very severe, impairment of station and gait, and loss of the deep reflexes arouse the suspicion that the affection is of spinal origin, a diagnosis always obscure but much influenced by the blood examination, since pernicious anæmia has not been found to arise as a secondary affection in spinal degenerations, while posterolateral sclerosis has been frequently observed in this form of anæmia.

Prognosis.—The course of the disease is not, as a rule, steadily progressive. There are periods of improvement followed by relapse. Many cases go on in this way for years. There are cases which run a very rapid course and end fatally within a few months. The average duration is about a year or fifteen months. Occasionally recoveries have been recorded. The following conditions are of bad omen: an oligocythæmia of less than 1,000,000 per cubic millimetre, a high percentage of megaloblasts and blood crises of megaloblastic type, the inability to take arsenic, tendency to hemorrhage, gastro-intestinal disturbances, and, as in almost all grave chronic diseases, the privations and disabilities incident to poverty.

(c) SPLENIC ANÆMIA.

This affection is considered under the heading Diagnosis of Diseases of the Spleen.

iii. Secondary or Symptomatic Anæmia.

Under this heading are included those forms of anæmia caused by acute and long repeated hemorrhages, certain intestinal parasites, unhygienic surroundings, insufficient food, prolonged lactation, the metal poisonings, acute and chronic infections, acute and chronic visceral diseases, especially nephritis and cardiac disease, and malignant growths.

In moderate cases the freshly drawn blood presents an appearance nearly normal, but in cases of intense secondary anæmia it may look like serum faintly tinged with crimson. In the latter case the tendency to rouleaux is slight. The coagulation period is diminished in proportion to the intensity of the anæmia. The further changes in the blood are as follows: hæmoglobin diminished to an extent proportionate to the ener of the cause; color index correspondingly decreased; erythrocytes dimi ished to a varying degree; nucleated forms in intense anæmia, the norm blastic type prevailing; departures from normal in the size and shape, an polychromatophilia in severe cases; leucocytosis commonly presen polynuclear neutrophiles relatively increased; lymphocytes decrease lymphocytosis may occur in severe and prolonged cases; myelocytes i small numbers; blood-plaques increased.

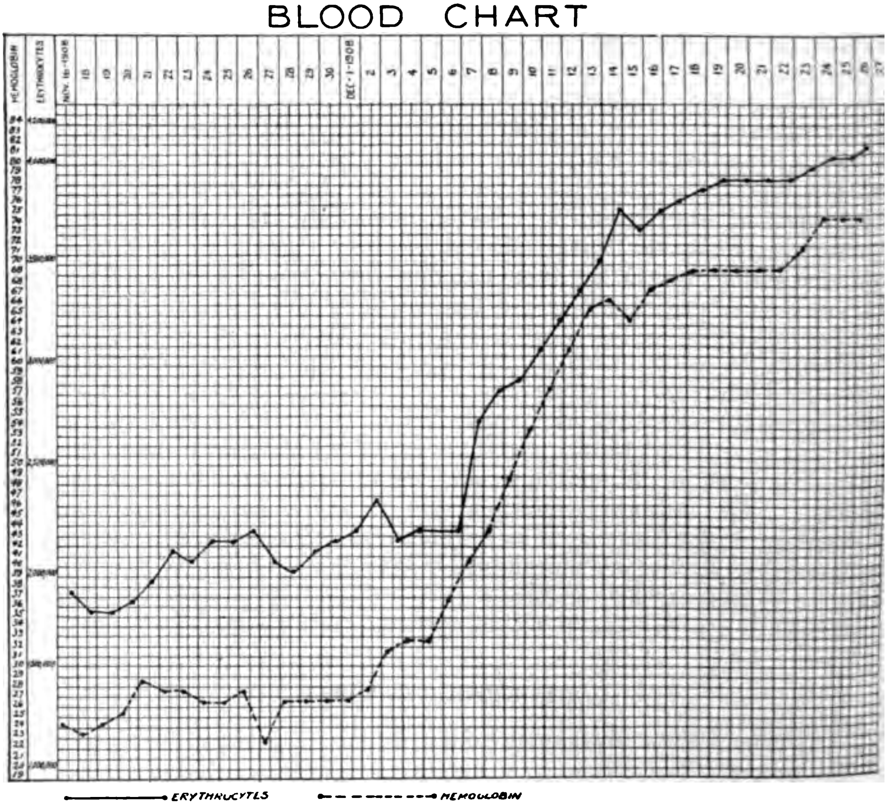

FIG. 329.—Chart showing variations in the number of erythrocytes and in the percentage of hæmoglobin in posthemorrhagic anæmia due to gastric ulcer.

POSTHEMORRHAGIC ANÆMIA.—An examination directly after the blood loss may fail to show diminution in the hæmoglobin or corpuscles by reason of the oligæmia. Fluid is, however, rapidly taken from the tissues into the capillaries so that a condition of hydræmia ensues and the decrease in hæmoglobin and erythrocytes becomes apparent, the minimum counts occurring some time within the course of a week. A more or less marked leucocytosis commonly occurs and persists for about a week, gradually declining. A gradual regeneration of the blood takes place in uncomplicated cases and is completed in from three to four weeks, the hæmoglobin reaching normal at a later period than the erythrocytes.

Secondary anæmia is very common in childhood. It may be congenital, as in syphilis and other infections, or acquired, as in (a) hemorrhages of various kinds, particularly from the navel, after circumcision, and in the purpura group, or (b) from general causes, as malnutrition, improper hygiene, syphilis, rickets, tuberculosis, the fevers, sepsis, gastro-intestinal and other visceral diseases, nephritis, acute disease of the heart, and malignant disease.

II. LEUKÆMIA.

Definition.—An affection of undetermined causation, characterized by an enormous but fluctuating increase in the leucocytes, decided decrease in the erythrocytes and hæmoglobin, and lesions of the spleen, lymphatic glands, or bone-marrow.

Varieties.—Two well-defined clinical varieties are recognized: (1) myelogenous or splenomedullary leukæmia, and (2) lymphatic leukæmia. The splenomedullary is much the more common type. Combined forms and variations are not infrequent.

Etiology.—PREDISPOSING INFLUENCES.—The disease has been encountered at all periods of life, from early infancy to the seventieth year. It is most common in the third, fourth, and fifth decades. Leukæmia is more common in males than females in the ratio of about two to one. Cases have been observed in pregnancy, and the disease not rarely develops at the grand climacteric. There are instances in which leukæmia has been observed in successive generations, and a leukæmic mother has borne leukæmic children. On the other hand, leukæmic mothers have borne non-leukæmic children, and a woman showing no signs of the disease has borne a leukæmic child. Leukæmia occurs in all parts of the world. It is not rare in the United States. Cases have been observed in almost all kinds of domestic animals.

EXCITING CAUSE.—The disease has upon inadequate grounds been attributed to malaria. It has followed blows and grave bodily injury. The tendencies to hemorrhage and to habitual nose-bleeding, which have been regarded as etiological, are much more probably early manifestations of an affection the true nature of which has been revealed by a study of the blood. In point of fact nothing is as yet known of the actual cause.

General Symptoms.—The onset is insidious and may be associated with persistent and intractable gastro-intestinal symptoms. In some instances the patients present the appearance of fairly good health until the occurrence of grave symptoms shortly before death. A lad of sixteen was supposed to be in his usual health until the occurrence of hemorrhage from the stomach, which proved fatal in the course of two days. Such cases are not very uncommon. Epistaxis is a frequent symptom. Pallor, palpitation, and dyspnœa upon exertion are very common. Distention of the abdomen due to splenic tumor and enlargement of the liver may first attract the attention of the patient. Tenderness over the sternum or the long bones occurs in many of the cases. Diffuse enlargement of the superficial lymph-nodes, which are generally of moderate consistency, not adherent among themselves or to the skin, and variable in size from time to time, is common in the lymphatic form. The groups usually involved are the cervical, axillary, and inguinal. These changes may be restricted to the mesenteric and other deep groups without demonstrable signs of enlargement during life, and there are cases in which the bone-marrow alone is involved, without enlargement of the spleen or lymphatic glands. There may be a high degree of emaciation and anasarca, or effusion into the serous sacs.

i. Myelogenous or Splenomedullary Leukæmia.

Symptoms.—Gradual enlargement of the spleen is usually the most conspicuous clinical phenomenon. The enlarged organ extends downward and to the right, and may reach the level of the pubic arch and pass beyond the median line. Its well-defined border and often the notch or notches may be distinctly felt. It is usually painless, but occasionally both pain and tenderness are present. Perisplenic friction may, in such cases, be recognized upon auscultation and palpation. Minor fluctuations in size may be observed: enlargement during digestion, and diminution during fasting and after diarrhœa or free hemorrhage. Fluctuations of greater amplitude frequently occur as spontaneous events in the natural history of the disease, or in consequence of the administration of arsenic or other therapeutic measures. Massive splenic enlargement causes pressure symptoms, among which the more common are distress after eating and constipation. Gastro-intestinal symptoms occur in the course of almost every case. Nausea and vomiting may be early and persistent. Diarrhœa is common. Hemorrhage from the bowel is not common. It may be due to chronic dysentery. Fatal obstruction of the bowel may be caused by pressure of the enlarged spleen upon the intestine. Jaundice is a rare symptom. Peritonitis and ascites may also be caused by the pressure of a massive spleen. Symptoms due to circulatory derangements and the changes in the blood are prominent. The cardiac impulse is displaced upward an interspace or more by the big spleen; hæmic murmurs may be heard. The pulse is usually of large volume and rapid, but soft and com-

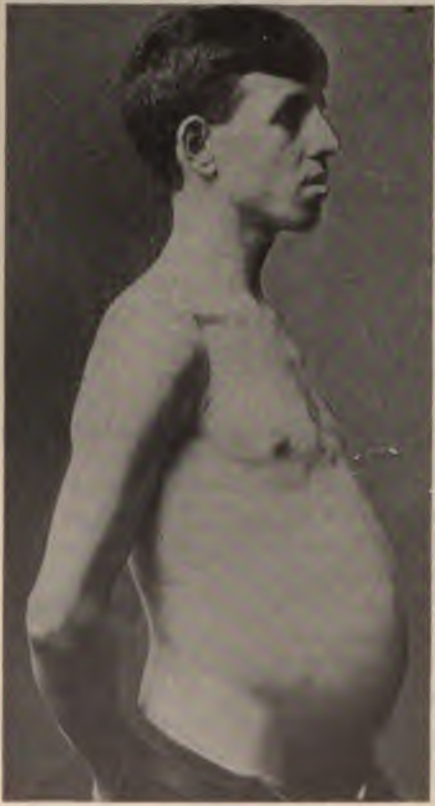

FIG. 330.—Splenomedullary leukæmia; distended abdomen due to massive enlargement of the spleen.—Jefferson Hospital.

pressible. The dyspnœa is anæmic. Hemorrhage is a very common symptom. Epistaxis, bleeding from the gums, hæmatemesis, purpura, retinal hemorrhage, and cerebral hemorrhage are frequent. Hæmoptysis and hæmaturia are much less common. Fluid exudates into the serous sacs are usually hemorrhagic. Œdema of the feet and general anasarca are terminal conditions. As in all the grave anæmias, headache, vertigo, and syncopal attacks are of frequent occurrence. Leukæmic retinitis may be due to hemorrhage or minute leukæmic deposits. Optic neuritis is rare. Deafness is common and the syndrome known as Ménière's disease has been observed. With the exception of a constant excess of uric acid the urine presents no characteristic changes. Priapism has been frequently noted. It may be an early and persistent symptom. Pneumonia or pulmonary œdema are common terminal events.

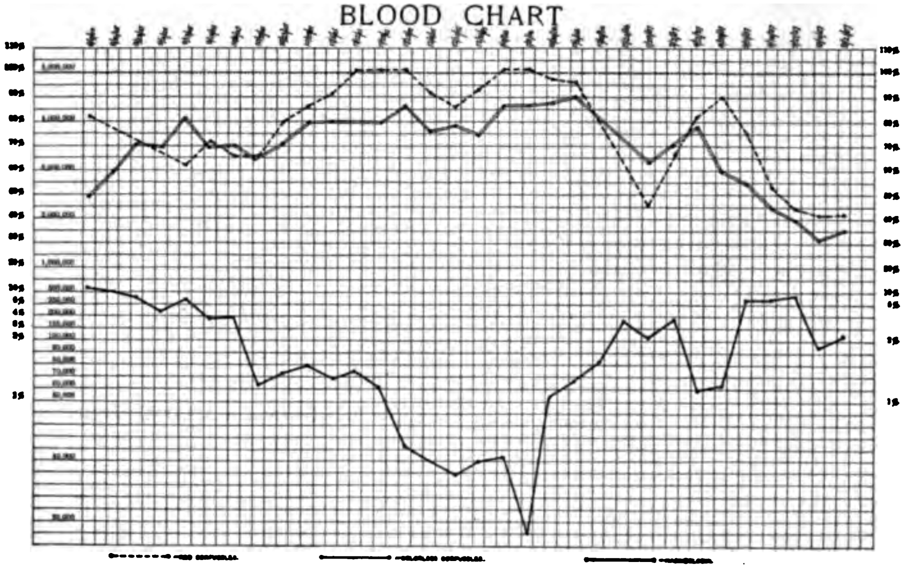

FIG. 331.—Chart showing variations in the number of leucocytes, erythrocytes, and in the hæmoglobin percentage in a case of splenomedullary leukæmia.

THE BLOOD.—The hæmoglobin is diminished not rarely as much as 50 per cent. The color index is correspondingly low. Exceptionally the color index is high.

Diminution in the number of erythrocytes is constant but not extreme. Counts falling below 2,000,000 are rare. Nucleated erythrocytes are very numerous, the normoblastic type being in excess. Megaloblasts are relatively less numerous and frequently present in atypical forms. Deformities in size and shape are present in varying degrees, dependent upon the extent of the hæmoglobin loss and cellular diminution, together with polychromatophilia affecting both the nucleated and non-nucleated erythrocytes.

The leucocytes are enormously increased. Counts of 300,000 per cubic millimetre are common, of 500,000 by no means rare, and of 1,000,000 occasional. When the oligocythæmia is marked the whites may nearly equal or even exceed the reds. The number fluctuates widely from time to time in the course of the disease, and frequently undergoes remarkable diminution under the free administration of arsenic. Wide diurnal fluctuations are observed in some cases, the lower counts being noted late in the day. Extreme changes occur in certain cases, both in the blood picture and in the general condition, so that periods arise in which there are neither symptoms nor signs of leukæmia present. These remarkable remissions are not, however, permanent. Such reversions to the normal blood conditions accompanied by involution of the splenic tumor have frequently followed the therapeutic use of the Röntgen rays. Myelocytes in great numbers are present and constitute at least 20 per cent. of all leucocytes, as determined by the differential count in the majority of the cases. The polynuclear neutrophiles are much increased, but their percentage is relatively low. Many of these cells are dwarfed and deformed. The relative percentage of lymphocytes is much below normal. Eosinophilia is almost invariably present. In the majority of cases mast-cells are present in considerable numbers and the blood-plaques are much increased.

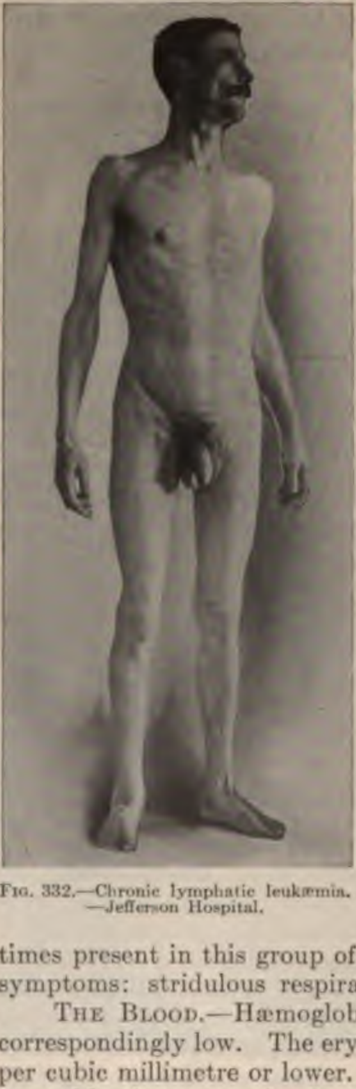
FIG. 332.—Chronic lymphatic leukæmia. —Jefferson Hospital.

ii. Lymphatic Leukæmia.

Symptoms.—The general symptoms are similar to those of the myelogenous form. There is enlargement of the lymph-nodes, which may involve the superficial or merely the deeper groups and is usually associated with more or less marked enlargement of the spleen. In the acute cases the lymph structures of the mouth, tongue, and pharynx may be enlarged. Enlargement of the thymus gland is sometimes present in this group of cases and may be the cause of characteristic symptoms: stridulous respiration with dyspnœa, usually paroxysmal.

THE BLOOD.—Hæmoglobin is much diminished and the color index correspondingly low. The erythrocytes are reduced in number to 3,000,000 per cubic millimetre or lower. Nucleated red cells are scanty, those of the normoblastic type predominating. Deformities in size and contour and the tinctorial reactions are proportionate to the degree of degeneration of the blood—anæmia. The leucocytes are much increased in number, but not to the extent often seen in myelogenous leukæmia, counts above 200,000 being comparatively unusual. The differential count shows this increase to be largely due to an excess of large and small lymphocytes, which comprise about 90 per cent. of all forms. There are numerous atypical forms. The relative proportion of polynuclear neutrophiles is much reduced, those cells not usually exceeding 5 or 10 per cent. of all forms. Myelocytes are present in almost all cases, but in very small numbers. Eosinophiles are also present in the majority of the cases in scanty numbers. The blood-plaques are increased.

Leukanæmia.—Leube has used this term to designate a symptom-complex consisting of intense anæmia with changes in the form of both the erythrocytes and leucocytes—conditions sometimes described as transitional or mixed forms of leukæmia and pernicious anæmia. He expressly reserves this term for forms of blood disease in which "both leucocytes and erythrocytes are uniformly and decidedly damaged in their development, and the case can neither be put in the category of leukæmia nor in that of pernicious anæmia." The cases are of sudden onset, with signs suggestive of an acute infectious process. In some of the cases there is severe tonsillar angina; in others stomatitis, extreme pallor, weakness, fever, hemorrhages, hyperplasia of the spleen, and moderate enlargement of the liver. Enlargement of the lymph-nodes is not constant. The course is unfavorable; the duration varies from a few days to several weeks.

Chloroma.—The blood changes in this rare affection are similar to those of lymphatic leukæmia. There is progressive diminution of the hæmoglobin and erythrocytes, associated with increase in the leucocytes. Deformities in size and shape in the erythrocytes, and nucleated forms, chiefly normoblasts, show themselves as the anæmia becomes more intense. There is marked lymphocytosis. The symptom-complex consists of progressive weakness, pallor, orbital pain, exophthalmus, deafness, and elastic swellings in the orbital and temporal regions. There is irregular hyperplasia of the bone-marrow with subperiosteal infiltrations and tumor-like metastases of greenish color—so-called "green cancer." The blood condition closely resembles acute lymphatic leukæmia, and chloroma has been regarded as a malignant form of leukæmia with greenish infiltrations and metastases.

Diagnosis.—The DIRECT DIAGNOSIS can only be made by a microscopical examination of the blood. The disease is always leukæmia when the proportion of leucocytes to erythrocytes is 1 to 15 or less; when the leucocyte count is more than thirty times greater than normal and when at the same time many of the cells are conspicuously immature—myelocytes, large lymphocytes, and nucleated red corpuscles.

Even in cases in which there is a temporary return to the normal ratio between the leucocytes and the erythrocytes, immature forms very rarely seen in normal blood—erythroblasts and myelocytes—are present in sufficient numbers to warrant a provisional diagnosis of leukæmia in the absence of a previous knowledge of the conditions of the blood.

DIFFERENTIAL.—The discrimination between myelogenous and lymphatic leukæmia can only be made by the blood examination. In the myelogenous form the type of the blood is myelocytic, that is to say, locytes are present in enormous numbers together with an increase sinophiles and mast-cells; oligocythæmia is moderate, erythroblasts are numerous, the normoblastic type predominating. In the lymphatic form the blood type is lymphocytic, namely, there is an excessive increase in the lymphocytes, myelocytes being absent, or present in very scanty numbers; there are very few eosinophiles or mast-cells; oligocythæmia is marked; and erythroblasts are few in number and proportionate to the general deterioration of the blood.

The clinical manifestations as regards the spleen and superficial lymph-nodes do not afford a basis for the differential diagnosis between the two forms of leukæmia; but this fact is without importance since an examination of the blood is essential to the general diagnosis.

Hodgkin's Disease.—The enlargements of the lymph-nodes and of the spleen in this affection often closely simulate leukæmia. The differential diagnosis rests upon the fact that the blood is normal or presents the changes which occur in the various forms of secondary anæmia. Associated inflammatory or infectious processes when present may cause an increase of leucocytes with the characters of a polynuclear neutrophile leucocytosis.

Chloroma may be recognized by the exophthalmus, orbital pains, and elastic tumor formations.

Splenic Anæmia.—The remarkable size of the splenic tumor, absence of enlargement of the superficial lymph-nodes, and a high grade of anæmia with leukopenia constitute positive differential criteria. Banti's disease, the terminal stage of splenic anæmia, is characterized by hypertrophic cirrhosis of the liver, jaundice, and ascites.

The diagnosis of leukæmia rests wholly upon the microscopical examination of the blood. Other conditions which, by reason of the presence of superficial lymphatic enlargements or splenic enlargement associated with more or less pronounced secondary anæmia, resemble leukæmia may be at once differentiated by the findings in the blood. It is only necessary in this connection to name the splenic tumor of chronic malarial infection; amyloid disease; malignant growths, cysts, and abscess; enlargements involving the left kidney, as hydronephrosis, cysts, perinephric cysts; abscess and malignant disease or cysts of the pancreas; and retroperitoneal sarcoma—all of which present resemblances to splenic tumor; and the hyperplasias of the lymphatic glands which occur in tuberculosis, syphilis, and malignant disease.

Hasty conclusions in a suspected case, in which the ratio of leucocytes to erythrocytes is normal, are unwarrantable because remarkable falls in the morbidly increased whites occur, (a) in the natural history of the disease; (b) during or immediately after acute intercurrent affections, as influenza, enteric fever, sepsis; (c) after the administration of drugs,—as arsenic and quinine,—the injection of nuclein, tuberculin, and antidiphtheritic serum, and the use of the X-rays.

Prognosis.—The outlook is in a high degree unfavorable. The number of permanent recoveries reported is limited. Remarkable and prolonged remissions occur. The influence of the X-rays upon the disease in some cases is to be considered. The acute lymphatic variety is peculiarly malignant and often runs a rapid course. The myelogenous form sometimes extends over eight or ten years, with remissions, even intermissions, and exacerbations. Progressive deterioration of the blood, hemorrhages, marked gastro-intestinal disturbances (especially intractable diarrhœa), fever, dropsy, and massive enlargement of the spleen are of unfavorable significance.

Mikulicz's Disease.—There is gradual, symmetrical, painless enlargement of the lachrymal glands, followed by similar changes in the salivary glands. Special predisposing influences are not recognized, though males suffer more frequently than females and in the majority of the cases the disease has occurred between the twentieth and fortieth years of life. The condition is generally regarded as an infection, but a special pathogenic agent has not been demonstrated. It has been attributed to tuberculous infection, syphilis, and hypothyroidism. Howard, whose recent studies of the subject are of great importance, regards the cases as constituting not a simple morbid entity but a clinical syndrome varying in its etiology, form, and course, and comprising isolated and symmetrical disease of the lachrymal and salivary glands due to simple lymphomata, pseudoleukæmia, leukæmia, tuberculosis, and syphilis. This observer arranges the cases in three groups: (a) the simple form, in which only the lachrymal and salivary glands are involved, neither the adjacent nor distant lymphatics being affected and the blood picture remaining normal for years. (b) Pseudoleukæmia. In this group of cases the clinical manifestations are similar to the simple form except that the lymphatic glands, either locally or generally, are involved. The enlargement is variable in degree. Softening and caseation do not occur. The spleen may be enlarged. The blood may be normal or there may be a moderate secondary anæmia. In other cases there is a relative or absolute increase in the small lymphocytes, and in some cases large lymphocytes are present. (c) Leukæmia. In this group, in addition to the enlargement of the lachrymal and salivary glands of both sides there are the characteristic general enlargements of the lymphatic glands and a blood picture of leukæmia of the lymphatic type with the usual clinical phenomena—progressive weakness, irregular fever, slight œdema, and tendency to hemorrhage.

III. HODGKIN'S DISEASE.

Pseudoleukæmia.

Definition.—An affection of undetermined causation characterized by successive enlargement of the lymph-nodes (those upon one side of the neck being first involved) and of the spleen; the deposition of metastatic nodules in the various viscera, especially the spleen, liver, and lungs; and marked secondary anæmia with cachexia.

Etiology.—Predisposing Influences.—The disease usually appears in childhood, adolescence, or early adult life. It is very rare after the fourth decade. It is more common in males than in females in about the proportion of six to one. The occasional occurrence of cases in a parent or child, or among the children of the same family, about the same time, suggests rather the action of a local cause or direct infection than the hereditary or family transmission of the disease. Malaria, syphilis, and tuberculosis have been regarded upon wholly insufficient grounds as predisposing influences.

EXCITING CAUSE.—The nearly constant presence of diphtheroid organisms in the glands and their occasional presence in the blood, together with the results of experiments upon monkeys (Bunting, Yates) and the results obtained by the use of autogenous vaccines (Billings, Rosenow), point strongly to the infectious nature of the disease. Among the clinical facts which support this hypothesis are the following: preliminary or inflammatory irritation of the mucous membrane of the upper air-passages; disease of the tonsils, early localization in the cervical lymph-nodes, gradual extension from one group of lymphatic glands to another with recurrence of fever and the acute course of some of the cases.

There are clinical resemblances to lymphosarcoma, which are not borne out by the results of histological studies of the affected glands in the two diseases, and to glandular tuberculosis, which are negatived by the following facts: (a) absence of tubercle bacilli and the failure of inoculation experiments in a majority of cases studied; (b) absence of reaction to the tuberculin test in well-characterized cases; and (c) specific histological characters in Hodgkin's disease. The view that Hodgkin's disease is a form of lymphatic tuberculosis has arisen from the fact that secondary tuberculous infection not infrequently occurs, especially in the terminal stages.

Morbid Anatomy.—The internal lymph-nodes are also generally enlarged—those of the thorax, the retroperitoneal, and the abdominal glands in the order named—and form large, firm masses, which give rise to the pressure symptoms which constitute striking clinical phenomena of the disease. The veins are especially liable to compression. The nerve-trunks and ureters do not always escape. The lymph-nodes even when greatly enlarged are not often adherent, nor is there a special tendency to capsular infiltration or invasion of contiguous structures. Caseation and necrosis do not occur in the absence of secondary infection. The spleen and liver are enlarged and the seat of scattered lymphoid masses.

Symptoms.—The most striking and usually the earliest clinical phenomenon is the enlargement of one or several lymph-nodes at the angle of the jaw. This enlargement is gradual and may attract little attention until several weeks have elapsed. The swollen glands are painless and may be recognized upon palpation as separate and distinct. They are not usually adherent to the skin. In some cases they are adherent among themselves. The peculiar adenopathy is progressive. The glands first involved gradually increase in size, adjacent groups in the lower part of the neck presently become implicated, then those in the axilla, the inguinal region, and sometimes those at the elbow and in the popliteal space. The lesions, at first unilateral, after a time appear upon the other side, but as corresponding groups are not usually enlarged to the same extent, they are irregularly symmetrical. The enlargement is, as a rule, most marked upon the side of the neck, where the masses often exceed the size of the fist and give rise to conspicuous deformities. It is not so great in the axillary and inguinal regions. The enlarged glands are at first of moderate consistence and may be moved under the skin; later in the course of the disease they become harder and more or less adherent to the subjacent tissues. Tonsillitis, sometimes chronic, frequently precedes the early glandular changes in the neck; but this is not always the case and there are instances in which the lymph-nodes in this region remain unaffected in the presence of every other feature of the malady. The evidences of enlargement of the internal lymph-nodes do not usually show themselves until after the changes in the superficial groups have made considerable progress. In fact there are rare cases of Hodgkin's disease in which the external glands are not at all or but slightly increased in size. Lymph-nodes of the digestive tract, namely, the tonsils, the adenoid masses at the base of the tongue and in the pharynx, and the solitary and agminate glands of the intestines, are frequently implicated without giving rise to marked symptoms. The spleen is usually enlarged, but not to the extent often seen in spleno-medullary leukæmia.

Anæmia does not show itself until the malady has made some progress. After a time the changes in the blood, characteristic of anæmia of secondary type, develop rapidly in the acute cases; more gradually in the chronic forms of the disease. At a period when the symptom-complex is fully established, the condition of the blood is as follows: hæmoglobin decreased to about 50 per cent., not commonly much below this; color index usually low; moderate erythrocyte reduction, rarely below 2,000,000 per cubic centimetre; nucleated forms rare, when present of the normoblastic type; leucocytes normal or slightly increased, the increase often transient; relative increase of polynuclear neutrophiles or lymphocytes; no increase of eosinophiles.

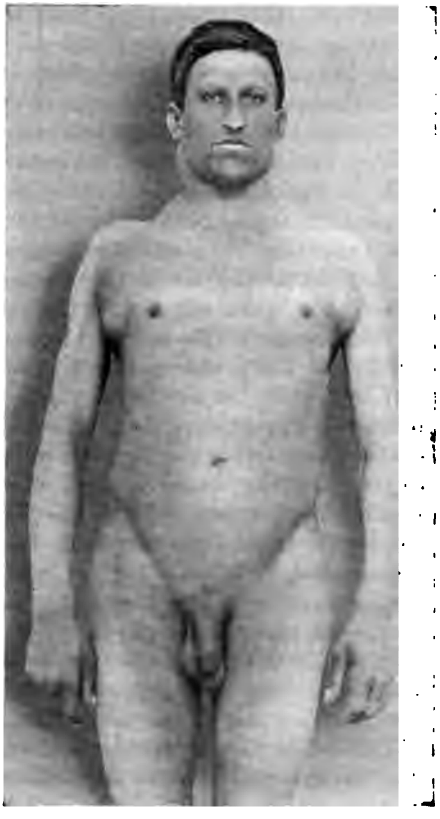
FIG. 333.—Hodgkin's disease.—Jefferson Hospital.

Three groups of symptoms may be recognized, namely, those due to the enlarged glands, those due to the anæmia, and the cachexia. But the symptom-complex is not well defined.

(a) SYMPTOMS DUE TO LYMPHATIC HYPERPLASIA. —*Superficial.*—External deformities and impairment of function, as of movement and the like. The prominence of these symptoms depends upon the location and degree of enlargement of the affected groups of glands. *Deep.*—Pressure symptoms, consisting of cough, dysphagia, dyspnœa, dilatation of superficial veins, cyanosis, local disturbances of circulation, various dropsies, œdema, and effusions into the serous sacs. Derangements of function due to pressure doubtless contribute to the cachexia. Neuralgias in various distributions are caused by pressure upon deep or superficial nerve-trunks.

(b) SYMPTOMS DUE TO THE ANÆMIA.—Pallor, headache, dyspnœa in some cases, asthenia, anasarca, hemorrhages, and fever. The last symptom may occur as a mild, irregular pyrexia early in the course of the disease, the attacks often being associated with sudden increase in the size of the affected glands; as an irregular ague-like paroxysmal fever when the disease has reached an advanced stage; and finally in the form of the "chronic relapsing fever" of Pel, or "recurring glandular fever" of Ebstein, in which febrile periods of from ten to fourteen days' duration, and either intermittent or remittent in type, alternate with irregular periods of apyrexia lasting several days. In view of the frequency with which intercurrent and terminal infections occur in Hodgkin's disease, the likelihood that these forms of fever are not primarily symptomatic of the disease under consideration must be considered.

(c) THE CACHEXIA.—The general disturbances of nutrition ultimately reach a high grade. Extreme pallor, emaciation, profound asthenia, bronzing of the skin in some of the cases, hemorrhages into the skin and from various mucous surfaces, together with the tumor masses in the inferior carotid triangle and the occipital region and elsewhere, constitute a striking and most suggestive clinical picture.

Diagnosis.—The DIRECT DIAGNOSIS rests upon the association of a widespread affection of the lymph-nodes involving progressively superficial and deep chains, anæmia of high grade and secondary type, and cachexia. The researches of Reed, Longcope, and others have settled the question as to the specific nature of the histological lesions in Hodgkin's disease and the uncertainties as to its relationship with tuberculosis, lymphosarcoma, and leukæmia. The association of the following changes is characteristic: proliferation of connective-tissue stroma resulting in fibrosis, and in a majority of the cases numbers of eosinophiles. These facts fully justify the excision of one or more superficial glands in a doubtful case for histological examination.

DIFFERENTIAL.—*Tuberculous adenitis* rarely develops after the twenty-fifth year. The enlargement frequently involves the glands of the neck upon one or both sides. Inflammatory adhesions between the individual glands and to the adjacent structures, and in particular to the overlying skin, are the rule. There is tenderness, and a tendency to softening, suppuration, and caseation with sinus formation. The process is sluggish and mostly limited to one or two groups of glands, rarely extending to distant regions. The signs of tuberculous lesions in the mouth (especially in the tonsils), in the skin, bones, lungs, or other viscera are suggestive. The presence of tubercle bacilli in the excised glandular tissue is suggestive of a tuberculous process, but the coexistence of the two diseases is by no means rare, while the definite histological lesions of Hodgkin's disease are distinctive. In afebrile cases the tuberculin tests may be employed.

Syphilitic Adenitis.—The glands in direct relation to the initial lesion, usually the inguinal, are commonly first affected, the glands of the neck being primarily involved only when there are buccal or labial lesions. The adenopathy is general, with an especial tendency to implication of the postcervical and epitrochlear glands. The enlargement is moderate, firm, painless, and not accompanied by a tendency to form adhesions to contiguous structures. A history of exposure, the presence or vestigia of an initial lesion, secondary manifestations, and the effects of mercurial treatment are of diagnostic importance.

Leukæmia (see p. 1148).

Lymphosarcoma.—The conditions are very often mistaken for each other. In lymphosarcoma the masses are of rapid growth and may attain great size; adhesions among individual glands and to adjacent structures, with inflammatory changes attended by redness, tenderness, and local œdema, are common. Softening and fluctuation suggestive of abscess formation are frequent. The cutaneous veins are often enlarged and tortuous, pain is sometimes a prominent symptom, and there are progressive infiltrations and metastases.

It is important in the differential diagnosis of Hodgkin's disease that this affection is characterized by a progressive implication of successive groups of glands, while in those affections, with the exception of syphilis, to which it may sometimes bear a superficial resemblance, the lesions are limited to single glands or, at most, to two or three groups which are anatomically related and become affected at or about the same time.

Prognosis.—The disease runs its course with exacerbations of varying intensity and remissions of irregular duration. Acute cases may end fatally in two or three months. Very chronic forms may last three or four years. The average duration is about one year. Urgent pressure symptoms, intense anæmia, hemorrhages, and early cachexia are of unfavorable prognostic significance. The outcome is lethal, but remarkable improvement in the blood and other clinical phenomena has followed the use of the X-rays, the early extirpation of the enlarged glands, and the administration by the mouth and subcutaneously of arsenic in the form of Fowler's solution, or of sodium cacodylate.

IV. DISEASES CHARACTERIZED BY HEMORRHAGE.

The Hemorrhagic Diathesis; Morbus Maculosus.

The essential symptom is hemorrhage, which may be into the skin, from the mucous surfaces, subserous, or into the viscera.

(a) Purpura.

Definition.—An affection which appears spontaneously, and is characterized by hemorrhages into the skin, mucous membranes, and internal organs. The following forms are recognized: (1) purpura simplex; (2) purpura hæmorrhagica or morbus maculosus Werlhofii; (3) purpura rheumatica or peliosis rheumatica; (4) purpura with visceral symptoms; and (5) symptomatic purpura. There are those, as Litten, who regard these as manifestations of the same disease, differing in intensity.

Purpura always occurs as a sporadic affection and never, like scurvy, [illegible] local outbreaks or as an endemic disease, or like the hemorrhagic cases [illegible]iola, varicella—during the course of epidemics. It is

Etiology.—PREDISPOSING INFLUENCES.—Females are somewhat more disposed than males. The greatest liability is in middle life, but neither infancy nor old age escapes. Purpura sometimes has developed during the convalescence from enteric fever, malaria, scarlet fever, and measles, and in the course of all forms of nephritis, especially the interstitial variety. Intense nervous shock, such as attends fright, fever, or pain, has been followed by the occurrence of purpura.

EXCITING CAUSE.—No immediate cause can usually be discovered. The hypothesis that purpura is an infectious disease has not yet received demonstrable support. Unfavorable conditions of life, damp and insalubrious dwellings, and chilling of the body have been regarded upon inadequate grounds as etiological factors.

1. **Purpura Simplex.**—**Symptoms.**—Isolated hemorrhagic spots upon the skin may constitute the only manifestation of the disease. They vary from a mere point in diameter to patches of considerable size. The latter are not common in simple purpura. Purpuric spots are more commonly and more abundantly distributed on the lower extremities than elsewhere. They are at first bright red in color, but rapidly become darker, and fade, leaving patches of brownish pigmentation which gradually undergo resorption. These spots do not disappear upon pressure or when the skin is made tense. In some cases the spots appear abruptly and wholly without prodromes, more commonly they are preceded by symptoms such as attend the initial stage of a mild infection, lassitude, loss of appetite, nausea, and fever. The disease sometimes lasts but a day or so and is marked by a single crop of hemorrhages into the skin; in other cases its course may extend over a week or two and be marked by successive crops of petechiæ. The spots are scattered over the arms, abdomen, and legs. They are rarely seen upon the face. The course of this variety of purpura is favorable. In a few days, or at most a fortnight, the spots have disappeared and the patient has regained his usual health. A slight degree of anæmia and transient albuminuria may occur during convalescence. It is much more common in children than adults, and is often associated with diarrhœa and slight rheumatoid pains and swelling of the joints.

Diagnosis.—The DIRECT DIAGNOSIS of purpura simplex may be made when, in the absence of a primary antecedent affection, the purpuric eruption appears after trifling derangements of health and disappears in the course of a few days or at most a week or two. The DIFFERENTIAL DIAGNOSIS from other forms of purpura depends upon the mildness of the attack, its short duration, and the absence of special characteristic manifestations, such as copious hemorrhages into the skin, hemorrhages from mucous surfaces, fever, joint affection, urgent gastro-intestinal symptoms, tendency to recur, and so forth.

2. **Purpura Hæmorrhagica; Morbus Maculosus; Werlhof's Disease.**—The cutaneous hemorrhages are abundant and extensive, and there is bleeding from mucous surfaces. This form of purpura sometimes attacks individuals in apparent health, but is far more common in delicate children or adolescents, especially girls. It may also occur without prodromes and run an afebrile course. When fever is present it is of moderate intensity—101°–102° F. (38.5°–39° C.). Gastric disturbances, a mild rheuma-

toid affection of the joints, and albuminuria occur in many of the cases. The cutaneous hemorrhages are profuse and extensive, and in some instances extensive areas or even the greater part of the surface of the body are involved, so that the discolored space in some portions of the body greatly exceeds that of the normal skin. Frequently, but not always, there is bleeding from mucous surfaces, usually first in the form of epistaxis, which is soon followed by hemorrhage from the buccal and gingival mucous surfaces, hæmaturia, hæmoptysis, and bleeding from the bowel. Under these circumstances profound anæmia may rapidly develop. Death may result from the loss of blood. PURPURA FULMINANS.—A malignant variety which occasionally occurs in children may cause death in the course of twenty-four or forty-eight hours from extensive cutaneous hemorrhage with or without bleeding from mucous surfaces. The course of the disease is often protracted, with frequent relapses, which are apt to occur when the patient attempts to leave his bed, and leads to great debility and anæmia, with vertigo, cardiac palpitation, and syncopal attacks. The DIRECT DIAGNOSIS of Werlhof's disease depends upon the severity of the affection, the prominence and extent of the cutaneous hemorrhage and bleeding from mucous surfaces.

It is to be differentiated from purpura simplex on the one hand by the above criteria, and from purpura rheumatica upon the other by the infrequency and mildness of the arthritis, the absence of urticaria and erythema, the tendency to hemorrhage from mucous surfaces. From scurvy it is to be distinguished by the conditions under which the disease develops, its more or less abrupt occurrence in an individual of fair previous health, and the absence of swelling of the gums; from the malignant or hemorrhagic forms of the infectious diseases by the symptoms of the onset in the latter, the gravity of the attack, the first appearance of petechiæ upon the forehead and wrists, their association with abortive eruptions or extensive suggillations.

3. **Purpura Rheumatica; Peliosis Rheumatica; Schönlein's Disease.**—This hemorrhagic affection is characterized by an eruption in which the spots "never coalesce," a multiple arthritis, and protracted course. It occurs in young adults, chiefly in males and usually in individuals who have a delicate, highly vascular skin and have previously suffered from rheumatism. It frequently begins with angina tonsillaris, fever of moderate intensity,—102° F. (39° C.),—and rheumatoid pains in the joints, especially the ankles, knees, the joints of the hands, and the shoulders. The affected joints are painful to the touch and upon movement and the seat of endo- and periarticular exudate. The eruption appears first upon the lower extremities or over the affected joints. It is frequently merely purpuric, but may show urticarious wheals, erythematous patches, or infiltrated areas suggestive of erythema nodosum. Vesication may occur. Local œdema occasionally appears, especially about the face—*febrile purpuric œdema*. The eruption tends to recur in crops and may continue to reappear for several weeks. The patients are sensitive to cold, and fresh outbreaks occur upon rising from bed or any chilling of the sur-
tacks at the same season in successive years have been noted.
special characters. It sometimes contains albumin. The

DIRECT DIAGNOSIS of Schönlein's disease rests upon the association of a well-defined joint affection with purpura, which tends to recur in crops, erythema, and local œdema. The joint affection is usually mild and shows neither the evanescence characteristic of rheumatic fever nor the persistence of gonorrhœal arthritis. DIFFERENTIAL.—The existence of Schönlein's disease as a nosological entity has been much questioned. It has been regarded as true rheumatism, from which it is easily distinguished by the eruption, which tends to recur, and the almost constant absence of cardiac lesions; as a form of gonorrhœal arthritis, a view that cannot be entertained, since gonorrhœa, though sometimes present, is mostly absent; as an intense form of purpura with rheumatoid symptoms, an opinion which finds some support in the fact that mild multiple arthritis is not uncommon in the hemorrhagic diseases.

4. **Purpura and Visceral Symptoms; Purpura Abdominalis; Henoch's Purpura.**—The cutaneous hemorrhages are accompanied by abdominal symptoms, vomiting, diarrhœa, often hemorrhagic, and colic. These symptoms occur in paroxysms separated by intervals of several weeks or months. The disease is most common in childhood and adolescence, but may occur in early adult life. It is much more common in males than females. It frequently attacks individuals living in poverty and want, but is by no means unknown among the affluent. There is a varying period of impaired health with headache, weakness, and loss of appetite, followed by subacute or acute arthritis affecting one or more joints, and moderate fever. Purpura now appears, often but not invariably upon the legs and feet, but spreading to the trunks and elsewhere, and being particularly abundant in the neighborhood of the affected joints. The hemorrhagic eruption is associated in varying degree with erythema, patches of œdema, and urticaria. Coincidently with the eruption the above mentioned abdominal symptoms appear. Epistaxis and hemorrhage from other mucous tracts occur. The colic, most intense about the umbilicus, is often especially severe and protracted; the abdomen is retracted and tender; there is complete loss of appetite; retching is frequent and distressing; the spleen is enlarged; the pulse small and frequent, and in many instances there is alarming exhaustion. In fact it is to this variety of purpura that the rapidly fatal *purpura fulminans* is to be referred. Nephritis is of frequent occurrence and may persist. These symptoms in the ordinary cases undergo gradual amelioration, and the infant enters upon an apparent convalescence, only to suffer from similar paroxysms at varying intervals. The duration is variable. The attack may last for several days, and recurrences may extend over a period of months or even years. The prognosis in children is favorable—mortality less than 5 per cent.; in adults less so—mortality about 23 per cent.

The DIRECT DIAGNOSIS of this form of purpura rests upon the association of purpura with erythema and urticaria, visceral symptoms, especially the abdominal crises, and nephritis, the joint affection and fever, and the paroxysmal nature of the recurrent attacks. This affection, concerning the cause of which we have no definite knowledge, presents a well-defined symptom-complex. The differential diagnosis requires no special consideration.

The blood changes in the various forms of purpura are similar and without diagnostic significance. They are those of secondary anæmia of varying intensity, with leucocytosis. The coagulation time is much protracted, in some cases reaching ten or even fifteen minutes.

5. **Symptomatic Purpura.**—Purpura is a symptom, not a disease, yet in the foregoing affections it is a symptom of such prominence as to justify the older use of the term to designate a group of diseases the etiology of which is as yet unknown, and of which cutaneous hemorrhage is the only common and constant phenomenon. But purpura is also a very common symptom in many conditions and well-defined diseases. This form is known as symptomatic purpura. TRAUMATIC.—The ecchymosis which follows a blow or contusion does not differ in appearance or course from similar cutaneous hemorrhages occurring in disease. Mechanical purpura may result from severe transient venous stasis, as in whooping-cough or epilepsy, or more prolonged interference with the circulation, as the pressure of a splint or bandage. TOXIC.—The venom of snakes causes rapid and extensive blood extravasations. Jaundice, especially when prolonged, is frequently associated with petechiæ. Among the drug exanthems purpura occupies an important place and may follow the administration of copaiba, ergot, quinine, belladonna, mercury, and the iodides. There is usually an idiosyncrasy. The question as to whether the cutaneous hemorrhage is due to the drug or the disease for which the drug has been given is always to be considered. There is no question as to the significance of a petechial rash in ergotism or the specific effect of the iodides in causing this symptom in certain cases. In the case of the iodides, coryza, angina tonsillaris, erythema, and fever may accompany the purpura. These symptoms may quickly follow small doses of the drug. INFECTIOUS.—The ordinary rashes of typhus and cerebrospinal fever are purpuric; similar lesions of the skin occur in sepsis and especially in malignant endocarditis; petechiæ and ecchymoses characterize the malignant forms of measles, scarlet fever, and smallpox. In that form of variola known as purpura variolosa there are petechiæ and most extensive suggillations of blood. CACHECTIC.—The terminal dyscrasia of cancer, chronic nephritis, Hodgkin's disease, tuberculosis, and other incurable wasting diseases is frequently accompanied by petechial eruptions, usually confined to the lower extremities. A similar form occurs in old age and prolonged want of food, and extensive purpura is characteristic of scurvy. NEUROTIC.—Myelopathic purpura occasionally occurs in spinal diseases, particularly forms of myelitis, especially transverse myelitis. The bleeding may be associated with trophic disturbances, erythema, and localized sweating. It occurs also in rare instances in tabes in the course of the lightning pains, and in association with herpes, œdema, and local sweating. Purpura may occur in the distribution of the affected nerve in severe neuralgia. The "stigmata" or bleeding points of hysteria, when not artificially produced or purposes of deception, must be referred to this category.

(b) Hæmophilia.

Bleeders' Disease.

Definition. — A constitutional anomaly mostly hereditary, — *vitium primæ formationis,*—very rarely acquired, which manifests itself by the occurrence of uncontrollable bleeding, either spontaneous or from traumatism which may be slight, and occasionally by hæmarthrosis sometimes followed by permanent deformity.

Hæmophilia is a hereditary and congenital condition; hemorrhage, which shows neither tendency to stop nor yields to treatment, the sign by which it makes itself known. In the absence of a history and of bleeding, there are no indications of the constitutional fault.

Etiology. — Predisposing Influences. — Grandidier has described hæmophilia as the most hereditary of all hereditary diseases. The occasional occurrence of fatal hemorrhage from trifling wounds has long been known, but the transmission of the tendency from generation to generation has been especially studied during the last century. In the well-known Appleton-Swain family there have been bleeders for nearly two hundred years. Grandidier gives the history of 200 bleeder families. In some instances the transmission is direct from the parents to the children. Usually, however, the transmission follows a peculiar law of heredity, namely, that the females transmit while the males acquire the condition. Exceptionally females also acquire it. From this it follows that hæmophilia is much more common in males than in females. The actual ratio is about 13 to 1. It is the rule that in a bleeder family a woman not a bleeder transmits the condition to her children without having acquired it herself, a generation having thus escaped. Men who are bleeders, but whose wives are not descendants of bleeder families, do not always beget bleeder children, and men who are members of bleeder families, but themselves not bleeders and whose wives do not belong to bleeder families, rarely beget bleeder children. Certain lines in bleeder families thus tend to become normal. It has occasionally happened that children born to parents neither of whom belong to bleeder families have been bleeders—*congenital hæmophilia, spontaneous hæmophilia.* Hæmophilia is more common in Germany, England, and the United States than elsewhere. The condition is usually discovered in infancy or early childhood. Social conditions are apparently without influence. In some bleeder families a neuropathic constitution has been recognized. As a rule the stock is fine, the families large, the members healthy and robust looking, with good skins and delicate complexions.

The actual cause of hæmophilia remains unknown

Pathogenesis. — Various theories have been advanced. Among them are habitual disproportion between the volume of blood and the vessels; hydræmic plethora; abnormal composition of the blood; an increase in the red corpuscles—erythrocythæmia; fragility of the vessels; pathogenic infection; and deficiency in the fibrin ferment. None of these have met the requirements of the condition.

Symptoms.—The existence of hæmophilia is usually discovered in consequence of trauma. The hemorrhage cannot be controlled, or is arrested with difficulty and only after prolonged effort. There are various grades of severity, from the mildest to the most severe which terminates fatally. The hemorrhage may be spontaneous or traumatic. The traumatism is often so slight as to escape attention, as the contusion resulting from a slight fall or blow, or chastisement. Spontaneous hemorrhages are sometimes preceded by fulness in the head, vertigo, tinnitus, palpitation, and nausea. They may be superficial, as from mucous surfaces, namely, that of the nose, mouth, female genitalia, urinary passages; or from the lungs, stomach, or intestines, or finally from cicatrices or ulcers upon the skin. Interstitial spontaneous hemorrhages are usually superficial. They chiefly occur in the scalp, face, scrotum; less frequently upon the extremities, and rarely upon the trunk. It is probable that the majority of such cases are in point of fact the result of slight traumatism. They consist of petechiæ, ecchymoses, and subcutaneous hæmatomata. The ordinary forms of trauma by which hæmophilia is manifest in external hemorrhage comprise abrasions, scratches, cuts, wounds, and surgical incisions. The bleedings named in the order of frequency are from the nose, mouth, bowels, urethra, vulva, stomach, lungs. Less frequently continuous bleeding takes place from areas of skin, especially upon the head and the scrotum, the tongue, eyelids, conjunctiva, finger-tips, lobe of the ear, and vulva. Bleeding from the head is far more common and usually more severe than from the extremities or trunk. Trifling operations, as lancing the gums, the extraction of a tooth, circumcision, or venesection, have been followed by fatal hemorrhage. Lethal hemorrhage has followed the rupture of the hymen in coitus. The bleeding usually is of the type described as parenchymatous; there is capillary oozing, more or less abundant, from every point of the exposed surface. It is not common to find flowing vessels of any size. It is continuous and may last for hours or for many days. After a time syncope may occur and the bleeding cease. Prolonged bleeding is often followed by death. Epistaxis may prove fatal in twenty-four or thirty-six hours. The coagulation time of the blood is much retarded. With Wright's instrument it has varied from twenty to forty-five minutes as compared with three to six minutes with normal blood. The arthropathies of hæmophilia are rheumatoid in character. They occur both spontaneously and after contusions. The knees and elbows are most commonly affected. The onset is acute, with swelling, pain, redness, and slight fever. Less commonly there is hæmarthrosis without fever. Repeated hemorrhage into the joints and muscles may occur in the absence of external or subcutaneous bleeding, and give rise to a false diagnosis of chronic rheumatism or tuberculosis. The resulting deformities sometimes suggest arthritis deformans.

Diagnosis.—Direct.—The recognition of hæmophilia depends upon the family history and the occurrence of persistent or uncontrollable hemorrhage. Neither the family history alone, since there are members of bleeder families who are not bleeders, nor a single unmanageable bleeding from a trifling cause, since difficult local hemorrhages are common enough in those who are not hæmophilic, justifies a positive diagnosis. The association of an hereditary tendency to repeated stubborn bleeding from slight injury with arthritis is highly suggestive. Even heredity is absent in the spontaneous or congenital cases. DIFFERENTIAL.—Habitual epistaxis and hæmaturia are not attended with the tendency to hemorrhages from slight wounds or cuts. In the hereditary local bleedings from the nose or mouth, associated with telangiectasis of mucous membranes, and nævi, the blood losses arise from definite lesions, and arthropathies are absent. Purpura rheumatica presents points of resemblance to hæmophilia, especially in the prominence of the arthritis. There may be more than one case in a family, but the peculiar form of heredity seen in bleeder families does not occur, and there are multiple spontaneous hemorrhages rather than excessive bleedings from limited surfaces.

Prognosis.—The outlook is unfavorable; many of the cases die in early infancy, a majority before puberty. The hæmophilic tendency becomes less marked as life advances, but the subjects rarely reach seventy. The prognosis is less favorable in boys than girls. Death does not often occur in a first bleeding, but it occasionally results from uncontrollable bleeding after ritual circumcision. Females who are hæmophilic are apt to menstruate early and freely, but neither this function nor that of parturition is attended in bleeder families with an especial tendency to dangerous blood loss. Any form of hemorrhage may, however, prove fatal; that which is most frequently so being epistaxis.

(c) The Hemorrhagic Diseases of the New-born.

Acute Fatty Degeneration of the New-born; Buhl's Disease.—This rare affection is characterized by fatty degeneration of the heart, liver, and kidneys, and hemorrhages in the various organs. The chief symptoms are inanition and external hemorrhages, of which the more common are omphalorrhagia, melæna, and hæmatemesis. Bleeding may also take place from the mouth, nose, eye, and ear. The infant is soon in a condition of asphyxia, from which resuscitation is only partial, and dies at once or in the course of a week or ten days. The skin is cyanotic and icteric. An anatomical diagnosis cannot be made in the absence of a microscopical diagnosis. It is therefore probable that the condition is very often overlooked. The differentiation from phosphorus and arsenic poisoning, in which similar parenchymatous changes in the viscera occur, may be apparent from a study of the circumstances; from sepsis, with interstitial hemorrhages and fatty degeneration, it may be difficult. Infection by way of the cord must be excluded. The prognosis is lethal.

Infectious Hæmoglobinuria of the New-born; Epidemic Hæmoglobinuria; Winckel's Disease.—This obscure affection arises as an endemic or epidemic disease in lying-in hospitals. It begins about the fourth day of life and is characterized by marked cyanosis with icterus, hæmoglobinuria, somnolence, and collapse without fever. Vomiting and diarrhœa are common. The urine contains small amounts of albumin and methæmoglobin. Epithelial granular and blood-casts are also present. It may attack strong, well-developed children. It runs a rapid course and is extremely fatal. Death may be preceded by convulsions. The etiology is unknown. The post-mortem findings are in some cases similar to those of Buhl's disease. The spleen is enlarged. The diagnosis rests upon the occurrence of associated cyanosis and icterus, the sudden onset upon the fourth day, the character of the urine, and the endemic or epidemic prevalence of the affection in an institution. The disease is to be distinguished from Buhl's disease by its onset some days after birth and the urinary conditions, and from icterus neonatorum by the severity of the process.

"Hemorrhagic Disease of the New-born"; Morbus Maculosus Neonatorum. — Townsend has made a thorough study of a condition of not infrequent occurrence and uniform symptomatology, which has been described under the above terms. This affection is self-limited, attended with moderate fever, and occurs almost always within the first week of life. Hemorrhage arises from the mouth, nose, bowels, and navel. Petechiæ and ecchymoses are common. Visceral hemorrhages and bloody collections in the serous sacs are found upon post-mortem examination. The children are very anæmic in appearance, but the peripheral blood may show an increase in hæmoglobin and erythrocytes. In a case of Townsend's the hæmoglobin was 125 per cent. and the erythrocytes 6,245,600. The affection is of brief duration. The mortality is about 60 per cent. When recovery takes place it is usually complete and permanent. The diagnosis may be based upon the general character of the disease, its manifestly infectious nature, its self-limited course, and its prevalence in institutions. It is to be distinguished from other forms of disease of the new-born, characterized by hemorrhage, which have been described, and especially from hæmophilia. A general rather than a local tendency to hemorrhage is of diagnostic significance. It is probable that many of the cases described as melæna neonatorum belong to this category.

DISEASES OF THE DUCTLESS GLANDS.

I. DISEASES OF THE SPLEEN.

i. Anatomical Anomalies.

Complete absence of the spleen occasionally occurs in association with other developmental faults. Much more commonly the organ is rudimentary. Very frequently there are accessory spleens — *splenunculi* — the supernumerary spleen or spleens lying within the folds of the gastrosplenic omentum and other processes of the peritoneum passing to the spleen. Abnormal lobulation and departures from the usual shape are common. These abnormalities are of no clinical interest. Entire absence of the spleen may be unattended by functional disturbances.

In complete *transpositio viscerum* the spleen occupies a position upon the right side corresponding to its normal position on the left. Under these circumstances it is sometimes represented by a number of lienculi which may be arranged in a cluster or loosely separated. Exceptionally the transposition may involve only the liver and spleen. Displacement may occur in the new-born downward as the result of abdominal deformities or umbilical hernia, upward in consequence congenital diaphragmatic hernia. These displacements are of minor rest.

ii. Movable Spleen.

Lien Mobilis; Wandering Spleen.

This condition is sometimes the result of congenital elongation of the gastrolienal ligament; sometimes of a similar elongation acquired through mechanical influences, such as pressure, blows, the succussion of violent, continuous coughing, the traction of peritoneal adhesions, and the weight of the enlarged organ itself. Wandering spleen is most commonly encountered in women suffering from enteroptosis.

Symptoms.—The dislocation is downward and forward, and the organ may reach a position below the level of the umbilicus or even pass into the pelvis, or it may form part of the contents of a large inguinal hernial sac. It is, as a rule, more or less enlarged. Subjective symptoms may be wholly absent, and the condition may be accidentally discovered. More commonly there are sensations of weight and dragging, with diffuse dull pain in the left flank. Colic, constipation, dysuria, and neuralgia may result from derangement of the various structures upon which the displaced and enlarged spleen exerts traction or pressure. The obstruction to the circulation sometimes causes great distention of the splenic vein. Torsion of the pedicle may lead to strangulation with great pain, tenderness, and local swelling, followed by necrosis with local or general peritonitis.

Physical Signs.—The upper end of the organ may sometimes be felt below the edge of the ribs, an important diagnostic point in the differentiation between an enlarged and a dislocated spleen. Palpation detects the indented median, the outer rounded, and the sharp lower border. The respiratory movements do not affect the dislocated spleen as they do the normal or merely enlarged organ. The normal dulness in the left hypochondrium is replaced by tympany.

Diagnosis.—The diagnosis is usually unattended by difficulty. The size, shape, and position of the organ, its free mobility, and the absence of dulness in the normal position of the spleen serve to distinguish it from the various abdominal or pelvic tumors, which its presence in an abnormal position might suggest.

iii. Acute Splenic Tumor.

The spleen undergoes enlargement in the acute febrile infections. The degree of enlargement varies in different diseases and in different cases of the same disease. The enlargement is almost constant in malaria, and so common in enteric fever as to constitute a phenomenon of diagnostic importance. It is also very frequent in typhus and relapsing fever, and occurs in pneumonia, smallpox and the other exanthemata, anthrax and septic conditions. Moderate splenic enlargement is frequently observed in acute miliary tuberculosis, secondary syphilis, and cerebrospinal fever. It occurs also, but is less common and less marked, in various acute catarrhal inflammatory conditions of the respiratory system, as coryza, [illegible]itis, and bronchitis.

Symptoms are usually wholly absent. In exceptional cases there are sensations of weight and tension in the left flank, and some discomfort is experienced when pressure is made over the splenic region.

Physical Signs.—INSPECTION.—In rare instances in persons with little subcutaneous fat the side may slightly bulge and the outline of the lower border of the enlarged viscus may be seen below the margin of the ribs, especially upon deep inspiration. PALPATION.—By this method moderate degrees of splenic enlargement may be recognized. The patient should be partly upon the right side, with his knees and thighs moderately flexed, and his head and shoulders supported upon a pillow. The physician standing to the left of the patient performs bimanual palpation, the palms of his right hand exerting pressure over the splenic area in the posterolateral aspect of the chest, while the fingers of his left hand are passed firmly upward beneath the margin of the ribs in front, the patient at the same time being directed to breathe slowly and deeply with an open mouth. The spleen descends with each deep inspiration and if enlarged may be readily felt by the fingers of the left hand. When the enlargement is considerable, the notches in the anterior border may be palpated, and in very rare instances pulsation has been recognized. PERCUSSION.—This method of examination yields unsatisfactory results in moderate degrees of enlargement and cannot be depended upon. Errors occur in gaseous distention of the stomach, meteorism, fecal accumulations in the colon, and enlargement of the left kidney. When the spleen is considerably enlarged this method is more satisfactory. Very light direct percussion with the dorsal surface of the finger-tip yields satisfactory results, as does auscultatory percussion. *Auscultation.*—Intermittent and continuous soft murmurs have been heard in the splenic area in the malarial paroxysm and in relapsing fever.

iv. Chronic Splenic Tumor.

Hypertrophy of the Spleen; Congestive Hypertrophy.

Chronic enlargement may follow acute splenic tumor and be due to the action of infectious principles. It occurs also in chronic malaria, leukæmia, cirrhosis of the liver, and cardiac affections. The organ may be increased to twenty-five or thirty times its normal size. Its surface is commonly smooth and there is thickening of the capsule.

Symptoms.—When the enlargement is of moderate size there are often no subjective symptoms. When it is considerable, weight, dragging, and a dull pain in the left side are experienced. Interference with the respiratory play of the diaphragm may cause dyspnœa, especially when the patient lies upon his left side. Traction upon the stomach may lead to loss of appetite, indigestion, nausea, and vomiting. Cardiac palpitation, œdema of the ankles, and colic occur. Anæmia is often marked. Hemorrhages and especially hæmoptysis are occasionally present.

Physical Signs.—The left side of the abdomen and the left hypochondrium may be distended. The visible and palpable tumor is popularly known in malarial districts as "ague cake." The lower and anterior margins are often distinctly palpable with their characteristic features, and may, in extreme cases, reach to the brim of the pelvis and to the right of the median line respectively. Upon auscultation friction sounds may sometimes be heard. The bruits occasionally heard in acute splenic tumor are not present. In cases of moderate enlargement in which there are no adhesions, the spleen may be so freely movable as to constitute one of the forms of movable spleen.

Diagnosis.—DIRECT.—The physical signs elicited upon inspection, palpation, and percussion, the presence of dulness in the normal splenic area, together with its uninterrupted extension to abnormal limits downward and forward and the well-defined borders notched in the anterior and rounded in the inferior lines, and more or less distinct participation of the tumor mass in deep respiratory movements of the diaphragm justify a diagnosis of hypertrophy of the spleen.

DIFFERENTIAL.—The nature of the enlargement may be obscure. In leukæmia the condition of the blood is diagnostic. In Hodgkin's disease the history is important, and the presence of enlarged superficial lymph-nodes, especially when they form groups or masses in the cervical, axillary, or inguinal regions, is suggestive. In congenital syphilis and rickets the associated phenomena are significant. In splenic abscess, fluctuation and fever, together with other septic phenomena, point to the presence of pus. Echinococcus cysts of the spleen are rare and may give rise to uncertainty. Rupture into the intestine or externally may afford clinical evidences by which the true condition may be recognized during life, and rupture into the peritoneum may cause fulminant peritonitis, but these accidents are among the most infrequent of clinical occurrences. Malignant disease of the spleen is attended with grave disturbances of the general health, cachexia, and metastasis.

Tumors of the kidney usually occupy a lower position and are less movable. They are crossed diagonally by the colon and often associated with urinary symptoms of importance. Nephromata are common in early life, and hydronephrosis and pyonephrosis at all ages. A diagnosis of splenic hypertrophy or abscess should never be made until after every form of renal tumor has been excluded. A tumor of the fundus of the stomach, of the colon, or the omentum may, as a rule, be differentiated without difficulty from enlargement of the spleen.

Moderate enlargement is usually present in movable spleen. The latter condition is characterized by the free movement of the organ, its contour, and by resonance in the region of normal splenic dulness.

Prognosis.—The outlook depends upon the cause of the enlargement. Congestive and malarial spleens often undergo remarkable diminution in size. The leukæmic spleen shows wide oscillations in volume in the course of the disease and under treatment. Enlarged spleens may become smaller during pregnancy. The enlargement may exist for years without detriment to health.

v. Splenic Tumor with Anæmia.

Primitive Splenomegaly; Splenic Anœmia; Banti's Disease.

Anæmia is characteristic of many conditions in which the spleen is enlarged, especially the primary anæmias, as leukæmia and pernicious anæmia. Secondary anæmia accompanies splenic enlargement in Hodgkin's disease, chronic malaria, and various forms of hepatic cirrhosis. Idiopathic splenomegaly with secondary anæmia occurs in a group of cases of which the following types are the most important:

(a) PRIMITIVE SPLENOMEGALY.—Marked and persistent enlargement may occur without associated disease and with but slight blood changes, and give rise to no symptoms other than those caused by the pressure and weight of the enlarged organ.

(b) SPLENIC ANÆMIA.—The spleen is very large. There is marked anæmia of secondary type. Hemorrhages, particularly hæmatemesis, are common. Purpura, melanoderma, and œdema of the lower extremities occur. The disease runs a very chronic course.

(c) BANTI'S DISEASE.—The advanced stages of splenic anæmia are characterized in a sub-group of cases by secondary cirrhosis of the liver with subicteroid discoloration of the skin, or actual jaundice and ascites.

Diagnosis.—The DIRECT DIAGNOSIS of splenic anæmia rests upon the association of primitive splenomegaly with secondary anæmia, and the absence of enlargement of the lymph-nodes.

The DIFFERENTIAL DIAGNOSIS between splenic anæmia and pernicious anæmia depends upon the morphological characters of the blood, the relatively high hæmoglobin percentage, and small size of the spleen in the latter affection. Leukæmia may be at once differentiated by the blood picture, and Hodgkin's disease by the enlargement of the lymph-glands and their peculiar massing in the cervical, axillary, and inguinal regions. Banti's disease and hepatic cirrhosis present similar pictures, but in the former the splenic enlargement is primary and of long duration before the changes in the liver and the resulting jaundice and ascites make their appearance. A history of alcoholism is of diagnostic importance.

vi. Splenic Tumor with Polycythæmia and Cyanosis.

Osler and others have recently described a condition characterized by cyanosis, an increase in the number of the red blood-corpuscles to 9,000,000 or even 13,000,000 to the cmm., and enlargement of the spleen. Headache, giddiness, and constipation are common symptoms. The cause of the disease is wholly unknown. It occurs in adults. The cyanosis is more marked in cold than in warm weather. Urinary changes, as low specific gravity, a trace of albumin, and hyaline and granular casts, are common. The condition is a persistent one. Such causes of cyanosis as emphysema, congenital and acquired heart disease, pulmonary sclerosis, and acetanilid poisoning must be excluded in making the diagnosis. The polycythæmia observed in those who have resided at high altitudes must also be borne in mind.

vii. Splenic Capsulitis—Perisplenic Peritonitis.

Inflammation of the capsule of the spleen occurs in arteriosclerotic atrophy of the organ, and in particular in the senile form, in acute splenitis, infarction, and abscess. It is met with also in local and general peritonitis and in chronic proliferative peritonitis. In many cases the capsular perisplenitis results from the extension of inflammation from neighboring organs, as the fundus of the stomach, the pancreas, and loops of intestine, and adhesions between these structures and the spleen are found.

Symptoms.—The clinical manifestations are often subordinate to those of the associated disease, as in gastric ulcer, pancreatic disease, or general peritonitis. In other cases circumscribed pain, tenderness, and swelling are associated with fever and much general disturbance of health.

The prognosis depends upon the nature of the primary disease. Capsular thickening and adhesions are frequently found upon post-mortem examination in cases in which no history of acute symptoms has been obtained.

viii. Infarct of the Spleen.

Embolism of the terminal branches of the splenic artery may occur in endocarditis, thrombosis of the left heart, and atheroma of the thoracic aorta. The infarcts thus caused may be single or multiple. They vary greatly in size. They are pyramidal in shape, the apex presenting toward the helium and the base toward the periphery of the organ, the corresponding capsule being very frequently the seat of a circumscribed plastic inflammation. The infarct, hemorrhagic at first, may undergo softening or a gradual cicatricial change which results in a contracting and pigmented scar. Infected emboli undergo softening with abscess formation.

Symptoms.—A chill, sudden severe pain and tenderness in the region of the spleen, which at the same time becomes enlarged, sometimes vomiting and collapse symptoms, constitute the symptom-complex. When these symptoms occur in endocarditis or in cases in which there are signs pointing to atheroma, the diagnosis of splenic infarct becomes highly probable. If there are coincident indications of renal infarct, such as severe pain referred to the loins, and hemorrhagic and albuminous urine, the diagnosis is positive. A friction sound may be heard. In many cases in which the anatomical diagnosis is made neither symptoms nor the signs of enlargement of the spleen were present during life.

ix. Suppurative Splenitis—Abscess of the Spleen.

This condition results from direct infection by pyogenic micro-organisms. There are two forms: simple and embolic. The former may result from traumatism or be secondary to infective processes in adjacent structures or infection by way of the blood. The latter occurs in septic conditions, the infected emboli being derived from local suppurative foci in distant parts. The simple abscess is usually single. It may vary in size from a cherry to the dimensions of the enlarged and overdistended spleen, the substance of which it replaces. Embolic abscesses are small and numerous, and begin as infarcts which rapidly undergo softening and are converted into collections of pus.

Symptoms.—The symptoms are those of infarction, namely, localized pain and tenderness, splenic enlargement, and irregular fever. They develop rapidly in embolic, more gradually in simple, abscess. There may be cough and dyspnœa due to interference with the movement of the diaphragm; gastric derangement caused by sepsis; œdema of the lower extremities arising from compression of the abdominal veins.

Physical Signs.—The lower end of the spleen may be palpated below the margin of the ribs: Fluctuation may be elicited. The rupture of the abscess may be followed by sudden pain and collapse symptoms, and the discharge of a considerable quantity of pus by vomiting or by the bowel, or in rare instances by way of the bronchi or the urinary passages.

Prognosis.—A great majority of the cases die. Recovery may take place by the resorption of the fluid contents of a small cavity and inspissation, or after the evacuation of the pus spontaneously or by a surgical operation.

x. Rupture of the Spleen.

Spontaneous rupture must be extremely rare. It is said to occur in connection with the acute enlargement of the organ in enteric and malarial fevers. Undue force in palpation or some similar traumatism may be suspected. Rupture of the spleen has been noted in childbirth. Severe blows, contusions, or penetrating wounds have caused rupture of the normal spleen. The rupture may occur at the site of an infarct, or an abscess may give way. The symptoms denote sudden internal hemorrhage associated with intense pain in the splenic region. There may be extended dulness in the splenic region or in the flanks due to collections of blood. The abdomen may be swollen and the seat of general distress.

The **diagnosis** becomes probable when, in the presence of acute or chronic enlargement of the spleen, or in the case of direct violence to the left side of the trunk, sudden intense local pain, collapse symptoms, and pallor arise, together with signs of increasing enlargement of the spleen·or accumulation of fluid in the flanks. An exploratory laparotomy should be performed without delay.

Prognosis.—In a few instances spontaneous recovery has resulted, as shown by the cicatrix when death has occurred at a remote period after the accident. Most of the cases are promptly fatal. Immediate operation has been the means of saving life. Localized tumors of the spleen, the so-called splenic adenomata, which consist of localized hyperplasias of splenic tissue within the spleen itself, fibromata, gummata, primary and secondary sarcomata, hydatid and other cysts are rare conditions of pathological rather than clinical interest.

II. DISEASES OF THE THYMUS GLAND.

The functions of this transitional organ are unknown. It has been accredited with a hypothetical internal secretion capable of antagonizing infections, a hypothesis which accounts for the comparative immunity of young infants against many of the acute febrile infections. The weight of the thymus gland at birth is variously estimated at from 8 to 13 grammes. It gradually increases in size till the end of the second year, from which period it undergoes progressive atrophy, until at puberty it is a shrivelled mass containing only traces of its original structure.

I. **Persistence of the Thymus.**—This occurs under varied conditions which may be causal or accidental, but are not understood. It is usually a post-mortem finding, but may be suspected when localized dulness along the sternal border on the left side from the second to the fourth rib may be made out. Persistent thymus has been frequently observed in exophthalmic goitre.

II. **Hypertrophy.**—Enlargement of the thymus has been regarded as the cause of thymic asthma or laryngismus stridulus—a condition attributed to the pressure of the enlarged organ. Dyspnœa, laryngeal cough, and bronchial râles in young infants have been caused by hypertrophy of the thymus and relieved by partial excision, but that it is the cause of laryngismus stridulus is by no means established. Sudden death may occur in lymphatism in infants with hypertrophy of the thymus. The children are found dead in bed or die in a short time with symptoms of asphyxia. In certain cases of sudden death in adults the thymus has been found greatly enlarged with signs of status lymphaticus. Hypertrophy of the thymus has been occasionally present in epilepsy.

III. **Atrophy.**—Primary atrophy of the thymus in infants may be attended with general atrophy in the absence of other symptoms. Secondary atrophy attends tuberculous and other wasting diseases.

IV. **Hemorrhages.**—Extravasations of blood in the substance of the gland have been frequently found in children dead of asphyxia.

V. **Tumors** of the thymus are sarcoma, lymphosarcoma, dermoid and other cysts, and gumma. Miliary tuberculosis and gumma are rare pathological findings. Mediastinal tumors frequently have their origin in the thymus.

The majority of the diseases of the thymus gland cannot be recognized during life, and are of pathological rather than clinical interest.

III. STATUS LYMPHATICUS; LYMPHATISM; CONSTITUTIO LYMPHATICA.

Definition.—A constitutional condition characterized by hyperplasia of the lymph-nodes and lymphatic tissues generally, the spleen, the thymus, and the bone-marrow, and the liability to sudden death.

Morbid Anatomy.—The lymph-nodes of the pharynx, thorax, and abdomen are chiefly affected. The superficial glands of the cervical, axillary, and inguinal regions may be involved. As a rule, the enlargement is moderate in degree and symmetrical, and differs in this respect from that of Hodgkin's disease. The enlargement of the spleen is moderate, the thymus is persistent and usually hypertrophied, and the yellow marrow of the long bones may be replaced by red marrow. The thyroid body is enlarged, and hypoplasia of the heart and aorta is present. Many of the subjects are rhachitic.

Etiology.—The condition has been variously ascribed to heredity and to over-irritability of the lymphatic tissue. It occurs principally in children and young persons. The association with rickets is probably accidental.

Clinical Phenomena.—The patients are fat and flabby, and have pallid, opaque skins. Hypertrophy of the tonsils and overgrowth of the pharyngeal adenoid tissue, swelling of the thyroid, overgrowth of the thymus (as indicated by dulness over the manubrium sterni, especially at its left border), enlargement of the superficial lymph-nodes, and especially signs of enlargement of the mesenteric glands, are present. Moderate enlargement of the spleen is suggestive.

Diagnosis.—When the foregoing signs are well marked the diagnosis may be made with some confidence, but a positive diagnosis cannot often be affirmed. The number of cases in which the lesions of lymphatism have been found after unlooked-for death, under circumstances in which such an event has appeared unaccountable—as the injection of diphtheria antitoxin serum for prophylactic purposes, chloroform or ether anæsthesia for trifling operations in young children, during the bathing of young children wholly without apparent cause, and occasionally during convalescence from the acute febrile infections—have given the condition a sinister significance. This fatal occurrence has been attributed to the pressure of the hypertrophied thymus upon the trachea, or to a perverted or excessive internal thymus secretion—lymphotoxæmia.

The DIFFERENTIAL DIAGNOSIS from Hodgkin's disease rests upon the more marked and asymmetrical enlargement of the superficial glands, and the absence of hypertrophy of the pharyngeal lymph structures in the latter affection; from glandular fever, upon the acute and self-limited course of the latter disease, the presence of fever, and the subsidence of the enlargement of the lymph-nodes after defervescence.

Prognosis.—The subjects of lymphatism have feeble powers of resistance, and in consequence of the hypoplasia of the heart and aorta are especially liable to sudden death.

IV. DISEASES OF THE THYROID GLAND.

i. Acute Thyroiditis.

Acute inflammation of the thyroid gland is rare. It may follow traumatism. Much more commonly it occurs in association with an acute infectious disease, as enteric fever, scarlet fever, diphtheria, rheumatic fever, pneumonia, or mumps. In a case recently under my observation it developed in the course of an attack of influenza. In very rare instances acute thyroiditis has been noted as a primary affection.

Symptoms.—The whole gland may be affected, or only one lobe. The attack begins with a chill or chilliness followed by high fever. Swelling, pain, and tenderness rapidly develop. Externally there may be redness with engorgement of the superficial veins, and cyanosis. Internal pressure upon the blood-vessels, œsophagus, and trachea causes headache, dysphagia, dyspnœa, and stridor. As a rule, resolution takes place in the course of several days or a week or two. Occasionally suppuration occurs. Destruction of the entire gland has been followed by myxœdema. Œdema of the glottis may occur. Myxœdema has been observed after an attack of acute thyroiditis.

ii. Goitre—Bronchocele.

Definition.—Hypertrophy of the thyroid gland. This term includes all enlargements of the thyroid gland not caused by inflammation, new growths, tuberculosis, syphilis, Graves's disease, and animal parasites. The anatomical varieties of goitre are, (a) parenchymatous, (b) vascular, and (c) cystic.

Goitre occurs as a sporadic and as an endemic disease.

1. Sporadic Goitre.—The temporary congestions which give rise to enlargement of the thyroid body in girls at puberty, in many women during menstruation, and in some during pregnancy cannot be regarded as goitres. Nor do the transient swellings caused by the pressure of a tight collar or excessive use of the voice constitute goitre. Enlargement of the gland due to parenchymatous, vascular, or cystic lesions, and more or less persistent, is not uncommon and occurs almost without exception in the female sex. Age is a predisposing factor of some importance. There are rare cases of congenital goitre. The disease is uncommon in childhood. It frequently first appears after puberty or in early adult life, but may not appear until fifty or later.

2. Endemic Goitre.—This affection is prevalent in circumscribed regions in many parts of the world. These regions are frequently but not exclusively mountainous, or deep valleys surrounded by mountains. Sometimes they are plains, especially in lake countries. Parts of Switzerland, the southern slopes of the Italian Alps, the Himalayas, the hill country of China, and some regions in Siberia are seats of endemic goitre, either alone or in association with cretinism. The disease is rare in North America. In occurs in some parts of Pennsylvania, the parts about the eastern end of Lake Ontario, and in the Province of Quebec.

The exciting cause of endemic goitre is supposed to be contained in the drinking water of goitrous districts. This opinion is supported by the following facts, which are generally accepted:

i. A healthy family coming to reside in a goitrous district presently may develop goitre among its members.

ii. Drinking water from a new and distant source has been followed by subsidence of goitre.

iii. An outbreak has followed the introduction of water from a goitrous region.

iv. Certain wells in Europe have had the reputation of causing goitre in those habitually drinking their waters.

Symptoms.—The enlargement may involve the entire gland, or only one lobe. Moderate-sized goitres cause no annoyance beyond that due to the deformity which they produce. Large tumors may give rise to dyspnœa by pressure upon the trachea; small tumors extending beneath the sternum may compress the veins. In extreme cases the goitre may compress the œsophagus and give rise to difficulty in deglutition. Sudden death has occurred in large goitres from pressure upon the vagi, or hemorrhage into the substance of the gland or the adjacent tissues.

ACCESSORY THYROIDS; PARATHYROIDS.—These may lie in the thyroid or near it. Their number varies, rarely, however, exceeding four. In some instances thyroid tissue is situated at the root of the tongue, in the mediastinum, or even in the pleural cavity. A lingual thyroid may exist in the substance of the tongue or attached to the hyoid bone.

Diagnosis.—DIRECT.—The disease may be readily recognized. It is an important characteristic of all tumors of the thyroid gland that they move upward in deglutition.

DIFFERENTIAL.—Goitre is to be discriminated from, (a) adenomata, simple or malignant; (b) malignant neoplasmata, both carcinomatous and sarcomatous. The important diagnostic criteria are the smooth parenchymatous enlargement involving one lobe or the entire gland; the uniform vascular enlargement with distinct varix arrangement, pulsation, and murmur; or recognizable agglomerate cyst formation.

Prognosis.--Treatment is not usually satisfactory. Iodine and the iodides, ergot, and counterirritants, much recommended, are not always successful. Thyroid extract is of questionable value. Large goitres may be removed.

iii. Exophthalmic Goitre.

Hyperthyrea; Graves's Disease; Basedow's or Parry's Disease.

Definition.—A disease caused by derangement of the internal secretion of the thyroid body, and characterized by exophthalmus, enlargement of the thyroid, tachycardia, and tremor.

Pathology.—Various views in regard to the essential nature of this affection have been from time to time entertained, but that which now has the most satisfactory basis of support is that it is a primary disease of the thyroid gland, resulting in an increased or deranged internal secretion. In defense of this view the following facts are adduced:

i. The active proliferation in glandular substance during the progress of the disease.

ii. The production of symptoms resembling those of exophthalmic goitre by the administration of thyroid extract.

iii. The fact that thyroid extract usually aggravates the symptoms of the disease.

Etiology.—PREDISPOSING INFLUENCES.—*Sex.*—The disease is common in women; comparatively rare in men. *Age.*—The onset usually occurs in early adult life, somewhere between the eighteenth and fortieth years. Rare cases have been observed in infancy. *Heredity.*—It sometimes occurs in several members of the same family, and has been observed in three successive generations.

[illegible] EXCITING CAUSE.—Acute infections, especially tonsillitis, rheumatic [illegible] and influenza, have been noted in many instances shortly before the [illegible] exophthalmic goitre. More significant are severe depressing [illegible]ry, anxiety, fright, and overfatigue as antecedent conditions and possible causal factors. There is a close resemblance between the immediate effects of sudden fright or terror and the symptoms of exophthalmic goitre, namely, exophthalmus, tachycardia, and tremor.

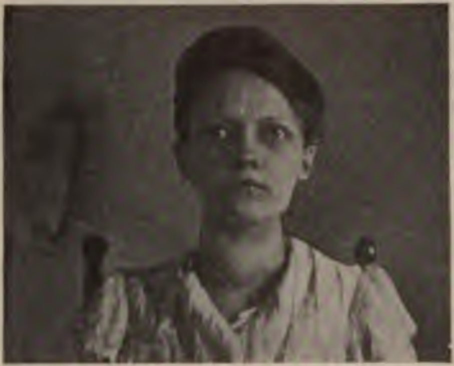

FIG. 334.—Exophthalmic goitre.—German Hospital

Symptoms.—The cardinal symptoms have been mentioned in the definition, namely, exophthalmus, goitre, overaction of the heart, and tremor. With these are usually associated anæmia, emaciation, sweating, diarrhœa, and irregular or suppressed menstruation. The cardinal clinical phenomena vary in degree and in the order of their development.

1. EXOPHTHALMUS.—In some instances the protrusion is so great as to prevent closure of the eyelids. In others it is so slight as to be scarcely noticeable. It is frequently different in degree upon the two sides and sometimes distinctly unilateral. Commonly a rim of white is seen above and below the cornea. In rare instances the eyes may be dislocated from their sockets.

Graefe's Sign.—When the eyeball is moved downward the upper lid does not follow it as in normal conditions. *Dalrymple's Sign.*—The palpebral fissure is wider than normal, owing to spasmodic retraction of the upper lid. *Stellwag's Sign.*—Infrequent, irregular, and incomplete winking. *Moebius's Sign.*—Insufficient power of convergence for near objects. The foregoing signs are commonly associated. *Joffroy's Sign.*—When the head is bowed forward and the patient asked to look up without changing his posture, the forehead is not wrinkled as occurs in health.

Pupillary changes and retinal lesions are rare. Defects of vision are uncommon. Subjective ocular symptoms, as sensations of pressure and phosphenes, occur. Ulceration of the cornea may take place, and in extreme cases destruction of the eyeball. Pulsation of the retinal arteries is frequent.

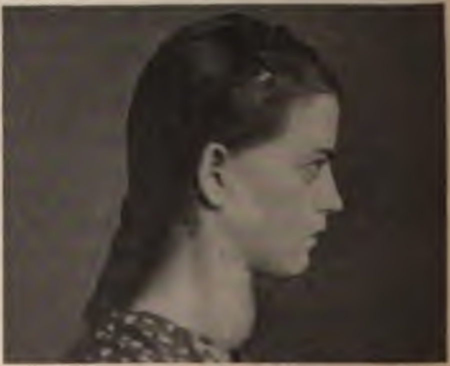

FIG. 335.—Exophthalmic goitre.—German Hospital.

2. ENLARGEMENT OF THE THYROID BODY.—The enlargement is usually moderate. Usually both lobes are affected, as a rule unsymmetrically, and more commonly the right to a greater extent than the left. The swollen gland is generally soft, but may be dense and hard, especially when goitre has preceded the disease. Distinct pulsation, thrill, and a systolic or continuous murmur are common phenomena. Sometimes the murmur is double. The thyroid enlargement undergoes remarkable

fluctuations in volume, increasing for a time and then subsiding, or undergoing repeated changes in the course of a short time. The attention of the patient is often first called to this symptom by the tightness of the neckband or collar.

3. CIRCULATORY DERANGEMENTS.—This group of symptoms constitutes the most constant and striking features of the disease. Tachycardia is always present. The pulse-frequency varies from 90 to 100 beats in the minute in the early course of the disease, to 100 to 130 and even 140 to 160 in severe and advanced cases. The rate is usually increased upon effort and under the influence of emotion. Forcible pulsations of the vessels at the root of the neck are associated with increased cardiac action and a greatly extended visible impulse. As a rule, the patients complain of palpitation and sometimes feel the sensation widely over the body. In some cases the overaction of the heart causes little discomfort. The heart is almost always dilated and sometimes hypertrophied. Its action is, as a rule, regular, but in grave cases arrhythmia is often marked. Systolic murmurs are common at the apex and across the base of the heart. The heart sounds are often intense, and may, in some instances, be heard at some distance from the body of the patient. Acute dilatation of the heart may occur. There may be visible pulsation of the peripheral arteries and the pulse-beat may be felt in the palms and finger-tips. A capillary pulse is common and the venous pulsation in the back of the hand may often be seen. Flushing of the neck and face is often pronounced.

4. TREMOR.—This cardinal symptom is a variable one. It may be forcible and annoying, involving not only the extremities but also the head, or so slight as to be discovered only upon careful examination. It is entirely involuntary, of limited extent, and about eight or nine to the second. It is usually symmetrical, but may be more marked upon one side, or confined to, or more distinct in, a single limb.

Anæmia, emaciation, and loss of strength are common. Fever is rare, but subjective sensations of heat, and copious perspirations, are common symptoms. Vomiting and diarrhœa occur in the absence of obvious cause. The electrical resistance is diminished, a fact attributed to the moisture of the skin due to vasomotor dilatation. Pigmentation is not uncommon. The parts chiefly affected are the face, neck, trunk, nipples, and flexures of the arms and thighs. In rare instances there is a general bronzing like that of Addison's disease; in other cases an irregular patchy discoloration. Patches of leucoderma may appear. Transient œdema is common and myxœdema is occasionally seen. The nutrition of the hair suffers and complete alopecia may occur. The teeth sometimes undergo rapid decay. Albuminuria, glycosuria, and true diabetes are occasionally encountered in the course of the disease. Menstrual derangements are common. When pregnancy occurs the condition of the patient often improves and the fœtus is born at term.

Nervous and mental symptoms are very common. Tremors, cramps of the hands and feet, a sensation of giving way at the knees, and increased tendon reflexes are observed. Irritability, altered disposition, and mental depression occur in most of the cases. The acute mania which sometimes ecedes death has been attributed to sudden, intense thyroid intoxication.

Course and Duration.—Incomplete forms are common and often overlooked. They may be characterized by tachycardia, nervous irritability, slight tremor, and little enlargement of the thyroid or protrusion of the eyes. There are acute and chronic forms. The latter are more common. Acute cases usually occur in childhood. They may last a few days or several weeks and get well. Relapses may occur. As a rule, when in adults the disease is well marked, recovery is infrequent. Death occurs from intercurrent affections.

Diagnosis.—When the four cardinal symptoms are associated a positive diagnosis can be made. Difficulty may arise in the rudimentary forms. Absence of exophthalmus or of thyroid enlargement is not often complete. These symptoms when slight are, in association with tremor and tachycardia, most significant. Anæmia, pigmentation, emaciation, and mental changes aid the diagnosis.

Prognosis.—The disease is essentially chronic. Acute cases are exceptional. A guarded prognosis should be given. In individual cases the more urgent the symptoms the less favorable the outlook. When the symptoms are mild the prospect of recovery is proportionately better. Cases in which the onset is sudden and severe, after fright, sometimes recover in a short time.

iv. Myxœdema.

Athyrea; Gull's Disease.

Definition.—A constitutional disease caused by the impairment or loss of function of the thyroid gland, and characterized anatomically by absence, atrophy, or goitrous degeneration of the thyroid, and clinically by profound nutritional changes, a firm, inelastic swelling of the skin and subcutaneous tissues, and nervous and mental symptoms.

Varieties.—Three forms are recognized: the infantile, or cretinism; the adult, or myxœdema proper; and postoperative myxœdema, or cachexia strumipriva.

Etiology.—The sporadic form of cretinism is due to the absence of the thyroid or suppression of its function. The endemic form occurs under local conditions associated with goitre, and is encountered in parts of France, Switzerland, and Northern Italy. The myxœdema of adults may develop at any period of life from puberty to seventy. More than half the cases begin between thirty and fifty. The disease is more common in females than in males in the ratio of about 6 to 1. In certain families myxœdema has been observed in two generations and there are numerous reports of its occurrence in several members of the same family. With reference to its geographical distribution, it is comparatively common in cold climates and very rare in the tropics. More cases have been observed in Great Britain and Europe than elsewhere. Many cases have been recognized in America. It is uncommon in Philadelphia. The colored races are said not to suffer from the affection. No walk of life is exempt, though it appears to be more common among the poor, a fact probably explained by the relatively larger numbers constituting this class. It is more frequent among married women and especially among those who have borne children than in others and has been thought to have some relation to the menopause, since many of the cases begin about the age at which the child-bearing function ceases. Occasionally it has followed symptoms of exophthalmic goitre. Postoperative myxœdema follows the total extirpation of the thyroid and the accessory thyroids. The cases of thyroidectomy to which cachexia has not supervened are thought to have been incomplete, an unobserved portion of the gland or accessory thyroids having remained.

The symptoms of cretinism differ from those of the adult form, but those of the latter and of the postoperative form are identical.

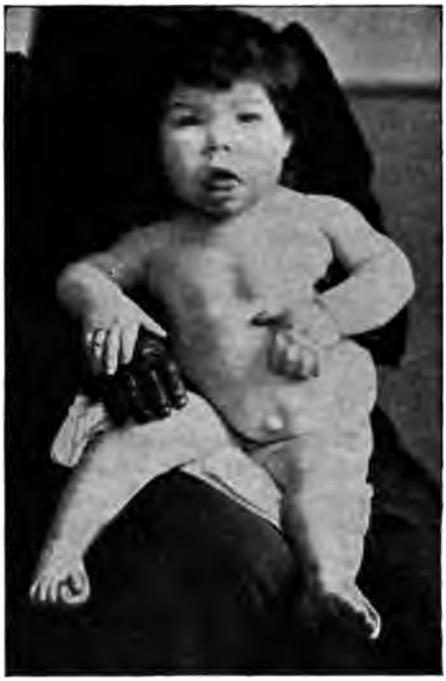

FIG. 336.—Cretinism; female, 9 years old.—Rotch.

1. Cretinism.—This affection occurs as a sporadic and as an endemic disease. There is retardation of physical and mental development. The condition is not usually recognized until toward the end of the first year, but becomes completely developed in the course of the second year. At this time the clinical picture is characteristic. The face is large, round, and bloated; the eyelids are puffy and congested; the nose is flattened and the alæ are thickened and coarse; the lips are full and swollen; the tongue is large and protrudes from the mouth. There is constant drooling. The eruption of the teeth is delayed, and they soon become carious. The complexion is pasty and sallow; the expression dull and fatuous. The fontanelles remain open. The belly is protuberant, the hands and feet are clumsy and ill-formed, the legs short, the muscles weak and flabby, and the child is unable to stand or walk. The hair is thin and brittle and the skin dry and harsh. The rectal temperature is commonly subnormal and there is great sensitiveness to cold. The mental condition remains undeveloped. Speech is acquired late and is rudimentary. There are various degrees of idiocy. Older cretins are dull and amiable, not often vicious, and present many of the characteristics of infancy at the age of ten or fifteen years. The fatty tumors seen in the myxœdema of adults are very common.

Diagnosis.—The DIRECT DIAGNOSIS is unattended with difficulty. The facies, the retardation of physical and mental development, the fat pads, and the subnormal temperature are distinctive.

DIFFERENTIAL DIAGNOSIS.—The early cases are sometimes mistaken for rickets. In the latter affection sweating of the head, craniotabes, restlessness at night in the early stages, and special deformities, as the rosary, enlargement of the epiphyses at the wrist and ankles, and bow-legs in the later stages, are characteristic. *Juvenile Cretinism.*—The forms that begin at the age of five or later in children previously well nourished and healthy, in consequence of atrophic changes in the thyroid following an acute febrile infection, are comparatively rare. Occasional cases of transient mild cretinism are seen in children in the second or third year and may be ascribed to functional derangements of the thyroid.

2. Myxœdema of Adults; Gull's Disease. — As a rule, the disease develops insidiously; exceptionally in the case of young adults it may be recognizable in the course of a few weeks. Languor, subjective sensations of cold, tardiness of movement, change of expression due to myxœdematous infiltration of the subcutaneous tissues of the face, and increase in the size and weight of the body are early symptoms.

The following phenomena are characteristic of the fully developed affection: (a) Dense, inelastic swelling of the skin and subcutaneous tissues, which does not pit upon pressure. This swelling is general but is most marked in parts where the subcutaneous tissues are loose. It is frequently first noticed in the face, sometimes in the lower extremities and the backs of the hands. (b) A change in the facies due to the swelling, which obliterates the lines of expression. The eyelids are swollen; the upper eyelid tends to droop; the eyebrows are habitually elevated; the forehead is corrugated by deep transverse wrinkles; the nose thickened and enlarged; the cheeks are full, large, sometimes pendulous, and often the seat of a circumscribed, pinkish flush. The lips are thickened and coarse and the mouth appears to be enlarged. Similar changes are seen in the ears and the parts about the angles of the jaw. (c) The hands and fingers are swollen and lose their expressiveness, assuming a thick, flat shape which has been described as "spade-like." Similar changes take place in the feet. (d) A general increase in the size and weight of the body, which may be mistaken for obesity, but which differs from that condition in the distribution of the swelling and the texture of the tissues.

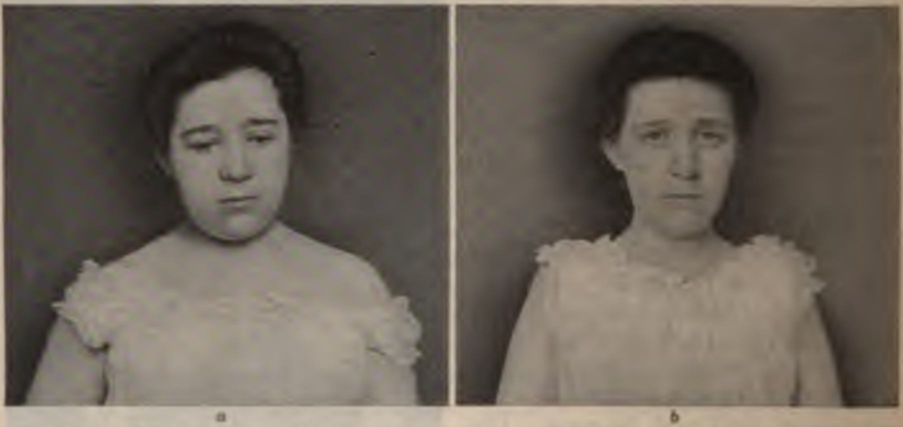

FIG. 337.—*a*, adult type of myxœdema. *b*, six weeks later; a reduction in weight of 20 pounds under thyroid therapy.—Jefferson Hospital.

(e) Local swellings of the skin and subcutaneous tissues, especially in the supraclavicular regions and in the posterior aspect of the neck. The occasional occurrence of fibrofatty pads in the retroclavicular spaces in healthy persons is to be borne in mind. (f) Changes in the thyroid gland, which cannot be felt at all or is of uncertain size in many of the cases, distinctly atrophic in others, and normal or increased in size in a very small proportion. (g) Dryness and roughness of the skin, thinness and brittleness of the hair, and alopecia which affects not only the scalp but the brows and axillary and pudendal regions. Similar atrophic changes affect the nails, which become cracked and discolored, while the teeth undergo rapid caries and become loose. (h) Subnormal temperature. The range is often continuously a degree or more of Fahrenheit's scale below normal and frequently several degrees. Temperatures of 95°-93° F. (35°-34° C.) or even lower have often been observed. Remarkable falls to 77° F. and 66° F. (25°-19° C.) have occurred before death. (i) Muscular weakness and slowness of voluntary movements. (j) Mental changes, especially slowness of apprehension and response, impairment of memory, sensitiveness, and irritability. The speech is tardy and drawling. Hallucinations are common. Fixed delusions may develop, and insanity terminating in dementia occurs. Albuminuria and glycosuria occasionally occur.

3. Postoperative Myxœdema; Cachexia Strumipriva.—The symptoms are those of the common form in adults.

Diagnosis.—The diagnosis is unattended with difficulty. From renal or cardiorenal dropsy myxœdema is to be differentiated by the character of the œdema, its failure to pit upon pressure, the absence of renal and cardiac lesions, the small thyroid, low temperature, the mental symptoms, and the promptly remedial effect of thyroid extract. From ordinary obesity the diagnosis is readily made.

Prognosis.—Formerly the outlook was practically hopeless. The patients improved somewhat in warm weather, but became worse as the cooler season approached. The course of the disease was chronic and progressive, sometimes extending over a period of ten or fifteen years. Death was due to intercurrent disease, very often to tuberculosis. At present in a majority of instances the prognosis is favorable. Treatment by thyroid extract causes the symptoms to disappear in a few months, and the continued administration of this remedial principle maintains the improvement.

4. Hypoparathyreosis; Status Parathyreoprivus.—These terms have been suggested by Halsted to designate degrees of the cachexia caused by the removal of some or all of the parathyroid bodies. It may also be caused by the arrest of the blood supply of those glandules in ligation of the thryoid arteries in partial thyroidectomy. Cachexia thyreopriva in many of the cases has been a complex condition made up of thyroid and parathyroid privation.

Among the symptoms of cachexia parathyreopriva tetany occupies the first place. This varies in degree from a subtetanic condition to the most violent manifestations of postoperative tetany, often terminating in death. Bleeding, with infusion of salt solution into the veins, the subcutaneous or intravenous injection of an extract or emulsion of the parathyroid glands, and finally the injection of a nucleoproteid prepared by Beebe from an emulsion of parathyroids, are followed by temporary relief of the tetany in parathyroidectomized animals, and favorable results in human beings have been reported by several observers abroad and by Halsted in one case in this country. Better and lasting results may be confidently expected from the transplantation or implantation of the living parathyroid gland. These glandules appear to play an important part in calcium metabolism, since their removal is followed by an increased excretion of calcium and diminution of the calcium content of the tissues. In dogs suffering from the most violent postoperative tetany, with muscular rigidity, clonic spasm, extremely rapid respiration and pulse, all the symptoms can be instantly dispelled by the injection of a calcium salt, the acetate or lactate, in 0.5 gramme doses, into the jugular vein (MacCallum).

V. DISEASES OF THE ADRENAL BODIES.

General Considerations.—The symptomatology of disease of the suprarenal bodies is obscure. Marked lesions of these organs have been found at autopsy in cases in which, during life, no symptoms suggestive of any disease involving them have been observed. In another group of cases tumor, pressure symptoms, and lumbar and sacral pain have suggested disease of these bodies. Again metastatic growths in various organs have been ascribed to malignant disease in the suprarenals. Finally, a characteristic symptom-complex—Addison's disease—has been found to be associated in a large proportion of the cases, but not in all, with definite lesions of these organs.

Addison's Disease.

Definition.—A constitutional disease, due to modification or cessation of the internal secretion of the adrenal glands in consequence of destructive lesions, usually tuberculous, and characterized by asthenia, gastrointestinal irritability, and pigmentation of the skin.

Etiology.—PREDISPOSING INFLUENCES.—Addison's disease is a rare affection. The most common and important predisposing influence is tuberculosis. It is somewhat more common in males than in females. It may occur at any period of life, but less frequently before twenty and after sixty than in the intervening stages. Malaria, alcoholism, depressing emotions, exposure, and traumatism have been regarded as causes of this as of many other diseases. In so far as they predispose to tuberculous infection they may act in this way.

Morbid Anatomy.—Very common are tuberculous deposits with caseous changes. Comparatively rare are atrophy,—either simple or resulting from chronic interstitial changes,—malignant disease, and interstitial hemorrhage. In a small group of cases the organs have been found normal, but inflammatory or pressure changes have been present in the semilunar ganglia. Cicatricial tissue implicating the semilunar ganglia and adrenals, together with sclerotic and pigmentary changes in the nerves, is not uncommon. The thyroid gland [illegible] absence of cancerous infiltration is usually small; the thymus sometimes persistent; the spleen enlarged or the seat of amyloid change.

Pathology.—Two principal hypotheses have been advanced: 1. That Addison's disease is an affection of the abdominal sympathetic system caused by disease involving the suprarenal bodies, or the solar plexus or semilunar ganglia. 2. That it is the result of loss of the function of the suprarenals. The theory of an internal secretion essential to normal metabolism is now fully established.

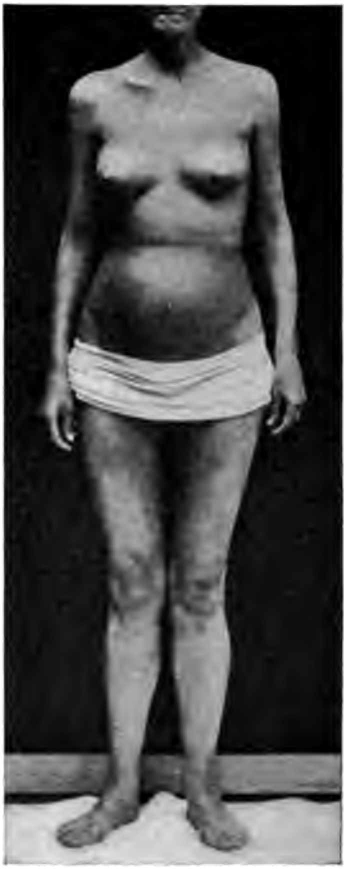

FIG. 338.—Addison's disease; showing distribution of bronzing.

Symptoms.—The onset is usually insidious. Little has been added to the description of Addison,—"Anæmia, general languor or debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of color in the skin." This description may be somewhat amplified.

1. ASTHENIA.—The first symptom is commonly a sense of fatigue in the performance of every-day accustomed duties. Fatigue symptoms, at first intermittent, soon become constant. Weakness is both muscular and circulatory. This may be marked, while the muscles still feel firm and the general nutrition and weight are preserved. The cardiac asthenia may be paroxysmal and lead to attacks of vertigo or syncope. Headache is common and pain in the loins may be an early and suggestive symptom.

The examination of the blood has yielded variable results. Anæmia is by no means a constant phenomenon.

Mental dulness is frequently observed.

2. DISTURBANCES OF THE DIGESTIVE ORGANS.—These appear gradually. Anorexia, epigastric distress, nausea and vomiting, and attacks of diarrhœa without obvious cause occur with varying prominence in a majority of the cases throughout the course of the disease. In a small proportion of the cases they are absent. Epigastric and abdominal pain are common toward the end. Gastro-intestinal symptoms may occur early.

3. PIGMENTATION OF THE SKIN.—Sooner or later a dark pigmentation of the skin appears. In many cases this is the first symptom to attract attention. The pigment accumulation is gradual. The affected portions of the skin are at first yellowish- or grayish-brown and later become brown or even blackish. The discoloration in well-marked cases is diffuse but never uniform. It usually begins in the parts exposed to the light and in those normally the seat of pigment deposits, and is deepest in those areas

and in regions subjected to the habitual pressure of the clothing. The face, backs of the hands, nipples and their areolæ, the genitalia, axillary folds, and parts pressed by the waistband, garters, and collar are especially pigmented. There may be diffuse patches of deep discoloration with indistinct borders, or small dark pigment areas with well-defined borders upon a less deeply pigmented surface. Patches of leucoderma are occasionally seen. The mucous membranes of the lips and mouth are frequently the seat of an irregular patchy pigmentation. Less commonly a similar discoloration affects the conjunctivæ or the vaginal mucosa. The course of the disease is essentially chronic, and marked by normal or subnormal temperature, subjective sensations of cold, suppression of menstruation, and the gradual development of cachexia. Urinary changes are inconstant. Increased pigments have been observed. Dropsy is rare. Death may occur early in the disease from sudden syncope. More commonly it is the result of progressive asthenia or advancing tuberculous lesions. It is sometimes preceded by acute toxæmic phenomena, urgent vomiting and diarrhœa, delirium with motor excitement, and convulsions followed by coma.

Diagnosis.—DIRECT.—A positive diagnosis may be made from the association of the following symptoms: general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach and irregular diarrhœa, a peculiar pigmentation of the skin and mucous membranes. In the early stages the diagnosis may be impossible. It is to be borne in mind that Addison's disease may occur without pigmentation.

The mere presence of pigmentation does not, however, warrant a diagnosis of Addison's disease. It may occur in the following conditions: physiological peculiarities due to racial and climatic influence; accidental pigmentation of the skin and mucous membranes in persons otherwise healthy; the mild cutaneous pigmentation of aged persons; chronic malaria; various cachectic states, especially those due to cancer and tuberculosis; pellagra; leukæmia; as the result of scratching in various chronic skin diseases attended by itching—prurigo, eczema, phthiriasis; diffuse melanosarcoma of the skin; rare cases of exophthalmic goitre; scleroderma; pregnancy and uterine disease; hæmochromatosis such as may occur in rare cases in association with hypertrophic cirrhosis of the liver and diabetes mellitus—*diabète bronzé;* certain cases of pancreatic disease; chronic arsenical poisoning; and argyria. Chronic jaundice has been confounded with the pigmentation of Addison's disease, an error that ought not to occur. Much difficulty attends the recognition of the actual condition when jaundice develops in the course of the disease and is associated with abnormal pigmentation.

DIFFERENTIAL.—In a doubtful case the foregoing conditions must be successively excluded as the cause of pigmentation by the systematic application of the rules of diagnosis, and the presence of asthenia, cardiac weakness, and gastro-intestinal symptoms determined before a positive diagnosis is made. In view of the tuberculous nature of the suprarenal disease in the majority of the cases the tuberculin test may be employed.

Prognosis.—The disease is probably always fatal. The rare reported recoveries have been ascribed to errors in diagnosis, or the mistaking of a prolonged remission such as sometimes occurs in the very chronic cases for an actual recovery from the disease. Cases unattended by bronzing of the skin are often rapidly fatal. The average duration is about two years. There are exceptional cases of very long duration—ten to thirteen years.

VI.

DISEASES OF THE PITUITARY BODY: DYSPITUITARISM.

Our knowledge of this subject is largely due to the labors of Cushing and his associates.[1] This author as the result of experimental and clinical studies has arranged the cases into five groups as follows:

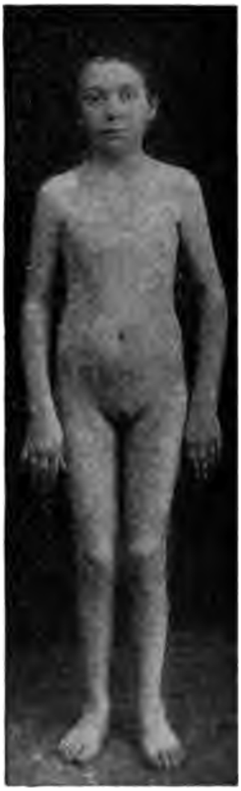

FIG. 339.—A typical case of infantilism with an enlarged 2 cm. sella. Patient aged 20 years 6 months: height 4 ft. 4 in. (133 cm.) (Ettore Levi) [*]

GROUP I.—Cases of dyspituitarism in which not only the signs indicating distortion of neighboring structures but also the symptoms betraying the effects of altered glandular activity are outspoken.

GROUP II.—Cases in which the neighborhood manifestations are pronounced but the glandular symptoms are absent or inconspicuous.

GROUP III.—Cases in which neighborhood manifestations are absent or inconspicuous though glandular symptoms are pronounced and unmistakable.

GROUP IV.—Cases in which obvious distant cerebral lesions are accompanied by symptomatic indications of secondary pituitary involvement.

GROUP V.—Cases with a polyglandular syndrome in which the functional disturbances on the part of the hypophysis are merely one, and not a predominant, feature of a general involvement of the ductless glands.

Neighborhood symptoms comprise such local pressure disturbances as primary optic atrophy, constriction of the visual fields, oculomotor palsies, anosmia, sometimes signs of implication of the crura cerebri and the uncinate region, epistaxis and pharyngeal protrusions.

Glandular manifestations consist of modifications of skeletal development, as overgrowth—acromegaly, gigantism—undergrowth—infantilism, dwarfism; cutaneous and subcutaneous changes, as hypertrophy of papillæ, hypertrichosis, thickening of subcutaneous tissues with slight bogginess or œdema in hyperpituitarism, and in hypopituitarism smoothness, transparency, freedom from moisture, with small, thin nails and scanty pubic and axillary hair and more or less irregular pigmentation, adiposity, high sugar tolerance, subnormal temperature, slow pulse, drowsiness, asthenia and arterial hypertension.

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[*] Figures 339, 340, 341, 342, 343, are inserted by courtesy of Dr. Harvey Cushing.

[1] The Pituitary Body and Its Disorders. J. B. Lippincott Company, 1912.

Psychic disturbances may be neighborhood symptoms due to pressure distortion of the temporal or frontal lobes by a growth, and consist of changes in disposition, loss of memory, disorientation, untidiness, and lack of appreciation of the existing condition; or they may be primarily glandular symptoms, as wakefulness, lack of decision, irritability and distrust. In hyper-

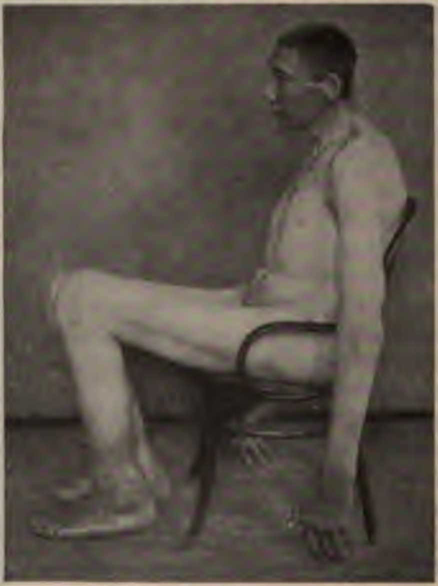

Fig. 340.—Preadolescent hyperpituitarism with giant overgrowth. Note extreme length of arms and thighs in relation to usual 18-inch chair.

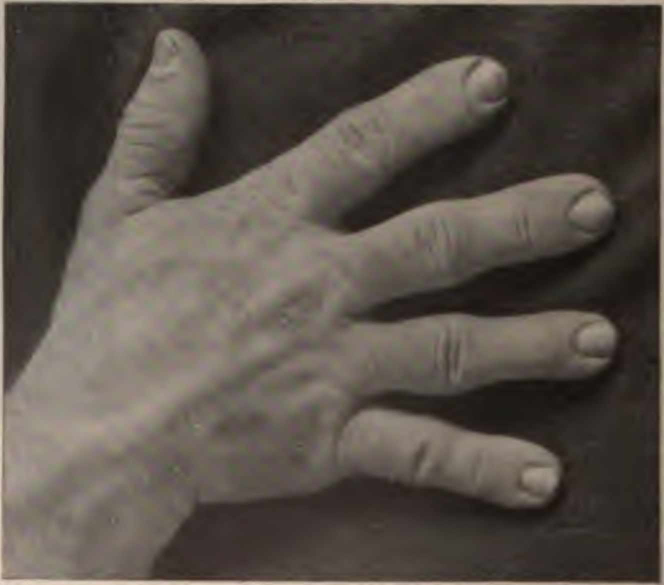

Fig. 341.—Characteristic square hand. Note heaping up of tissues about nails. "Type en large" of Marie.

tuitarism or in hypopituitarism any grade of mental disturbance from a ld psychosis to grave mental derangements with epilepsy may occur.

Retarded sexual development is on the one hand very common when the unifestations of hypophyseal lesions antedate puberty, while upon the other nd amenorrhœa or impotence with retrogressive sexual changes usually curs when dyspituitarism develops after adolescence.

The excessive subcutaneous fat observed in a large proportion of the cases s led to the employment of such descriptive terms as adiposis dolorosa, iposis universalis, dystrophy, dystrophia adiposo-genitalis, adiposis cerealis, to designate the affection.

Examination by X-rays may show distortion or enlargement of the sella.

ACROMEGALY.

This clinical manifestation is so com-)n and well characterized that its separate nsideration is important. The hypothesis at the skeletal changes are due to a func- nal hyperplasia of the pars anterior of e hypophysis with the production of an ered or excessive secretion has much pport in the collected facts.

Definition.—A trophic disease char- terized by symmetrical overgrowth in e soft parts and bones of the face and tremities, with deformities of the spinal umn and thorax.

Etiology.—PREDISPOSING INFLU- CES.—It is a disease of early adult life, st of the cases having first shown them- ves in the third decade. No case has en noted at an earlier age than twenty, d in only a few has the onset occurred er thirty-five. Males and females are ually liable, with the exception that in ses beginning at a late period more men suffer. The disease is not uncommon, and its geographical distribu- n is wide. Many of those who suffer from acromegaly were persons of eviously large growth.

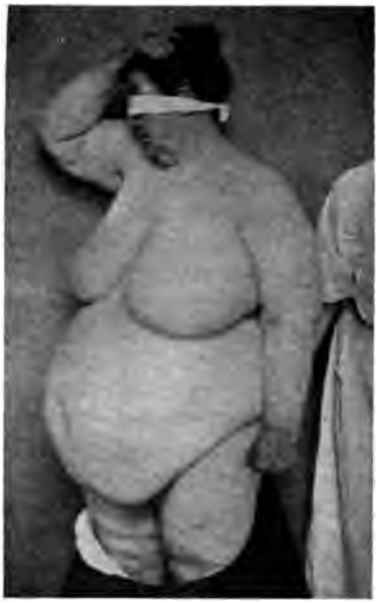

FIG. 342.—Extreme obesity—302 pounds ("adiposis dolorosa"), attributable to hypopituitarism.

Symptoms.—The disease develops gradually, the early symptoms con- ting of lassitude and vague pains, and abnormal sensations—*parœsthesiæ* in the head and extremities. Amenorrhœa and impotence are very nmon. The characteristic anatomical changes are first noticed in the e. The lines of expression are altered and the countenance is distorted. e skull is enlarged; the superciliary ridges are very marked and promi- nt; the zygomatic arches protrude, and there is remarkable hypertrophy the upper and lower jaw bones, the latter projecting in a conspicuous anner. The alveolar processes are similarly enlarged and the teeth e separated. The soft parts undergo corresponding and even more arked changes. The eyebrows are bushy and thick and almost meet in the median line; the eyelids are greatly thickened; the nose is conspicuously enlarged; the ears are enormously hypertrophied; and the lips swollen. Some degree of exophthalmus is often present and may vary in extent from time to time. Enlargement of the tongue is a very common lesion. This organ attains in some cases such dimensions that it is impossible to close the mouth. Similar and progressively increasing deformities involve the hands and feet, and are rendered especially noticeable by the relatively small size of the arms and legs, which do not share to any marked extent in the enlargement of the extremities. The thickening affects alike the bones and soft parts, and while not greatly interfering with the functions of the hands, causes the remarkable appearance described as spade-like.

A

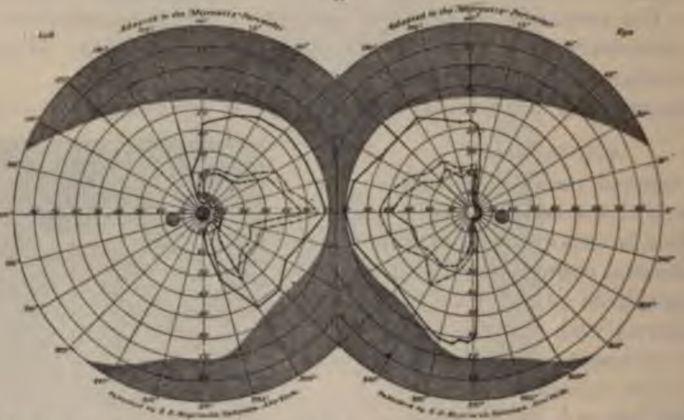

Fig. 343*A*.—Case X of Cushing's Series. Fields on admission. Bilateral primary optic atrophy. Bitemporal hemianopsia including the macula on left and sparing it on right. Acuity: Left 5/50; right 15/30. Marked photophobia. No exophthalmos; no diplopia.

B

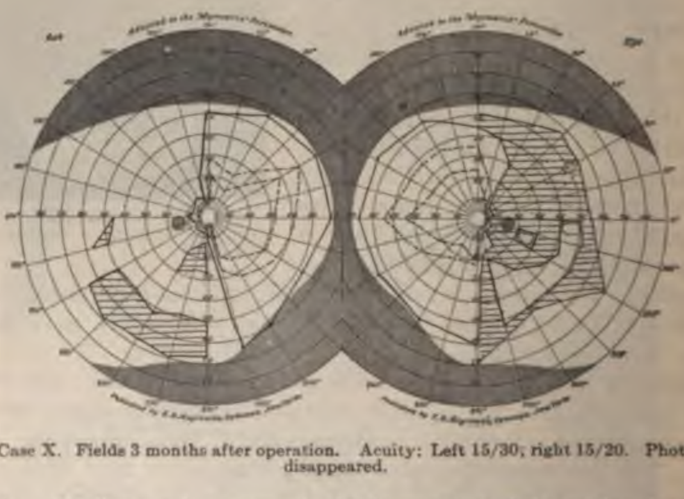

Fig. 343*B*.—Case X. Fields 3 months after operation. Acuity: Left 15/30; right 15/20. Photophobia has disappeared.

The nails are broadened but not incurvated and the drum-stick bulbous enlargement of pulmonary osteo-arthropathy is not seen. The feet are generally enlarged, the great toe being especially increased in size. The skin of the affected parts commonly preserves its natural appearance. In some instances, however, it becomes coarse and pigmented. As the disease advances the spine becomes affected. There are hyperostoses and exostoses of the vertebral processes, ankylosis of the vertebræ, and kyphosis. The clavicles are enlarged and there is a gradual enlargement of the ribs. The thyroid body may be atrophied or hypertrophied, but such changes are not constant. There are cases in which the thymus is enlarged. In a very large proportion of autopsies—73 in 77—the hypophysis cerebri has been found to be affected. Very often it has been increased in size, sometimes to that of an egg. Sometimes the increase is in an upward, sometimes in a downward direction. The histological changes are not constant. Glandular hyperplasia, softening, cystic degeneration, and fibrosis are described. Many of the cases have been regarded as malignant. Symptoms suggestive of cerebral tumor, namely, headache, vertigo, somnolence, are not infrequent. Ocular symptoms, bitemporal hemianopia, optic atrophy, and oculomotor palsies occur. The great frequency with which lesions of the hypophysis have been found lends support to the hypothesis that this organ is the source of an internal secretion by which the growth of the body is regulated, and that acromegaly is the result of some vitiation or defect in that secretion.

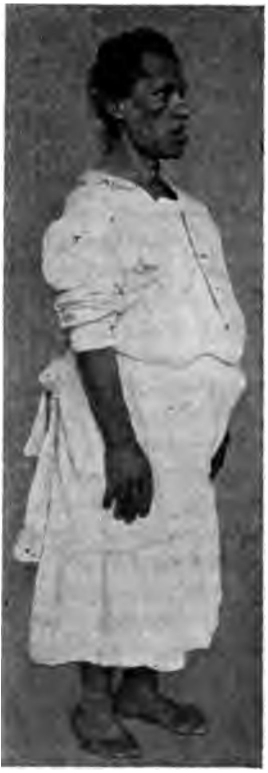

Fig. 344.—Acromegaly; diabetes mellitus; in a woman aged 46.—Jefferson Hospital.

Diagnosis.—The direct diagnosis of acromegaly is unattended with difficulty. In no other affection do similar anatomical changes in the bones and soft parts occur. The differential diagnosis from gigantism depends upon the specific nature of the changes in acromegaly, and the fact that they are for a long time chiefly confined to the face and extremities, the vertebral column and thorax being later involved. It is nevertheless true that acromegaly frequently develops in persons of large frame. From the rare cases of progressive overgrowth of one member or a part of it, or of one side of the body, acromegaly is to be distinguished by the fact that the hypertrophies are symmetrical and chiefly confined for a long period to the face and extremities.

Prognosis.—The disease may run a chronic, progressive course of a score or more of years. There are cases which terminate in death in four or five years. Restoration to normal conditions does not occur. A majority of the cases die of some intercurrent affection, as diabetes, cancer, or croupous pneumonia.

Polyglandular Syndrome.—Derangement of function in respect of increased or diminished activity in the thyroid and parathyroids, adrenals, thymus, pancreatic islets, pituitary, testis and ovary is associated with reciprocal functional and anatomical changes in other and frequently all of the correlated organs. To the resulting condition the term "polyglandular syndrome" is applied. It is not always clear which of the glandular organs is primarily at fault.

XIII.

THE DIAGNOSIS OF DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE HEART.

I. ABNORMAL POSITIONS OF THE HEART.

Congenital displacement is rare. It occurs in general transversus viscerum. The apex lies to the right; the right ventricle toward the anterior chest wall; the left ventricle behind it; while the pulmonary artery lies at the right border and the aorta at the left border of the sternum. Transposition of the heart alone—*dextrocardia*—is usually associated with complicated developmental anomalies affecting both the arteries and chambers. The heart is displaced to the left in congenital arrest of development of the left lung. Children with complete defect of the anterior chest wall and abdomen with protrusion of the heart—*ectopia cordis*—are usually stillborn or die shortly after birth. When there is merely a fissure of the sternum caused by defective development of the manubrium and body, the heart remains in its normal position and may be studied through the overlying soft tissues.

Acquired displacements are far more common and of greater diagnostic significance. Sudden death which sometimes occurs in massive pleural effusions has been ascribed to abrupt, angular bending or twisting of the inferior vena cava. The upward displacement of the heart, which occurs in the retraction of the lungs in patients long bed-ridden, or in meteorism, ascites, pregnancy, and large abdominal tumors, may lead to an erroneous diagnosis of dilatation of the heart. The high position of the apex and of the inferior border of the lung are significant. Large subphrenic abscesses displace the heart toward the opposite side and thus increase the resemblance to empyema, while a massive spleen, as in leukæmia, displaces the apex upward and outward.

The displacements of the heart toward the sound side in pleural effusions and pneumothorax, and toward the affected side in the contraction of the lung after resorption of a large pleural exudate, or in fibroid phthisis, are too familiar to lead to diagnostic errors. The fact that the displaced heart, in consequence of adhesions, occasionally remains upon the sound side is less generally understood. In pulmonary emphysema the dislocation of the heart is downward. Aneurisms of the ascending and transverse portions of the arch of the aorta displace the heart downward and toward the left.

II. DISEASES OF THE MYOCARDIUM.

i. Acute Myocarditis.

Definition.—Acute inflammation of the wall of the heart. Pathologically parenchymatous and interstitial forms are recognized. There is cell infiltration of the interstitial tissue, associated with granular, fatty, and hyaline degeneration of the muscle fibres, and loss of their contractile elements. There are three varieties: 1. PRIMARY ACUTE MYOCARDITIS.—In the absence of any recognized cause, or after a wetting, the symptoms of an acute interstitial inflammation of the myocardium develop with irregular fever and great weakness, and after a course of several days or a week or two terminate in death. Primary acute myocarditis is an exceedingly rare affection, the occurrence of which as a separate disease has been questioned. 2. SECONDARY ACUTE MYOCARDITIS.—This variety occurs in the course of or during the convalescence from many of the specific infectious diseases. 3. ACUTE SEPTIC MYOCARDITIS.—In a majority of the cases infection of the myocardium takes place by way of the coronary arteries. There are commonly multiple miliary abscesses. In other instances abscess formation follows the direct invasion of the heart muscles from the valvular or mural lesions of a septic endocarditis.

Etiology.—PREDISPOSING INFLUENCES.—These are wholly unknown. No plausible explanation of the fact that the myocardium suffers in some cases of infectious disease and sepsis, and escapes in others, has yet been adduced. There is nothing in age, sex, or previous health to account for this difference. Acute secondary myocarditis occurs in diphtheria, enteric and typhus fevers, scarlet fever, rheumatic fever, variola, and gonorrhœal infection. It is especially frequent in association with diphtheria. The septic form is less common.

EXCITING CAUSE.—The primary form has followed cold and exposure and traumatism to the left chest. The local action of specific toxins, the arrest of minute infected emboli in the branches of the coronary arteries, and direct bacterial invasion from the endocardium are the immediate causes of the myocardial inflammation.

Symptoms.—The clinical manifestations are usually obscured by those of the primary affection. Profound cardiac asthenia manifests itself by feeble heart sounds, a scarcely perceptible radial pulse, arrhythmia, and acute dilatation. Sudden increase of pallor, apathy, somnolence, and precordial distress occur. Vomiting is common and sometimes urgent. In some cases there is restlessness and agitation; in others subjective sensations are absent and the gravity of the complication reveals itself only by the objective manifestations.

Diagnosis.—DIRECT.—The diagnosis must, in a majority of the cases, be a provisional one. Only in the light of later events, often at a remote period in cases that have recovered, can the cause of the grave cardiac symptoms during the acute attack be recognized. The symptoms of chronic myocardial changes in an individual who has passed through a grave attack of one of the febrile infections are significant.

DIFFERENTIAL.—The intensity of the process varies. The average cases are to be differentiated from the acute cardiac asthenia of influenza by the rapidity with which the latter develops and the peculiar psychic manifestations with which it is so frequently associated; from acute cardiac exhaustion from overwork or athletic excesses by the history of the case; and from all of these by their occurrence in the absence of especial exposure to wet and cold, or of an acute infection, or of sepsis.

quality. Other murmurs are not common in the milder cases of myocarditis. In the graver cases dilatation to the left and upward, and usually toward the right, is almost constant. The transverse dulness is increased and the apex beat displaced to the left and slightly upward. The enlargement of the heart frequently increases and diminishes in accordance with the varying intensity of the symptoms, but a return of the heart to its normal size is no longer to be hoped for. Upon auscultation the increased muscular insufficiency is manifest in a well-characterized mitral systolic murmur, which after a time is associated with a tricuspid systolic murmur. Very rarely a faint aortic diastolic murmur may be recognized. These murmurs in many cases undergo rapid changes, corresponding to the degree of dilatation with the gain or loss in the power of the heart, and sometimes wholly disappear. In other cases they are persistent, and, when associated with hypertrophy, as in chronic nephritis, they cannot always be differentiated from the murmurs of organic valvular disease.

The pulse is usually weak and small. Sometimes, especially in the form of chronic myocarditis associated with interstitial nephritis, it shows increased tension. Its frequency is much increased by moderate exertion. In a majority of the milder cases arrhythmia does not occur; exceptionally it is an early sign. In a small proportion of the cases the pulse is slow. As the insufficiency becomes more marked the pulse-frequency augments and arrhythmia appears. The latter is often of high grade, both as to the time of the beats and as to their force. The inequality of the ventricular contractions is such that many of them fail to transmit the pulse-wave to the radials, so that the pulse counted at the wrist is less frequent than the impulse counted at the apex.

The following forms demand separate consideration: THE INFLAMMATORY FORM.—The symptoms are those already described. The affection occurs as a later stage of acute myocarditis, showing itself in some instances during the convalescence from the primary disease; in others not until a remote period. There are cases in which recovery takes place. In this connection it is to be noted that fibroid degeneration is sometimes the reparative process by which destructive lesions are corrected. THE FORM DUE TO DISEASES OF THE CORONARY ARTERIES.—*Sclerosis of the Coronary Arteries.*—The lesion consists chiefly of a thickening of the intima. It is either diffuse or circumscribed, and leads to narrowing of the lumen of the affected vascular twigs. The left coronary artery and its branches are involved much more commonly and to a greater extent than the right. When the narrowing of the lumen is of high grade, or there is complete occlusion of the affected vessels, infarction of the heart muscle—*myomalacia cordis* (Ziegler)—occurs. The necrotic focus is invaded by connective-tissue elements and converted into a fibroid cicatrix. There may be a diffuse increase of the interstitial tissue. This variety of chronic myocarditis presents symptoms of muscular insufficiency, but is especially characterized by a tendency to angina pectoris and so-called cardiac asthma, a special liability to precordial pain, and the signs of narrowing of the aortic orifice. In fact, arteriosclerosis of the coronary arteries is very commonly associated with similar pathological changes in the aortic valves and the beginning of the vessel itself. A systolic aortic murmur, usually not so loud and coarse nor beginning so promptly with the systole as in pure valvular stenosis, and followed by a well-marked, even accentuated, second sound, is usually heard. Dulness to the right of the sternum in the second and third interspaces and jugular pulsation are associated signs. *Embolism and Thrombosis of the Coronary Arteries.*—These lesions cause myocardial infarct. Thrombosis may result from sclerosis; embolism from various forms of infection, or very rarely from fragments of atheromatous plates. Death directly follows the closure of the coronary artery at its origin. Elsewhere the occlusion of the artery is followed by infarction and death after several days or, in rare cases in which the extent of the softening is limited, by recovery. The local necrosis may lead to rupture of the heart. The changes in the heart muscle depend upon the extent and degree of the sclerosis and the rapidity with which the blood supply to the muscle is arrested.

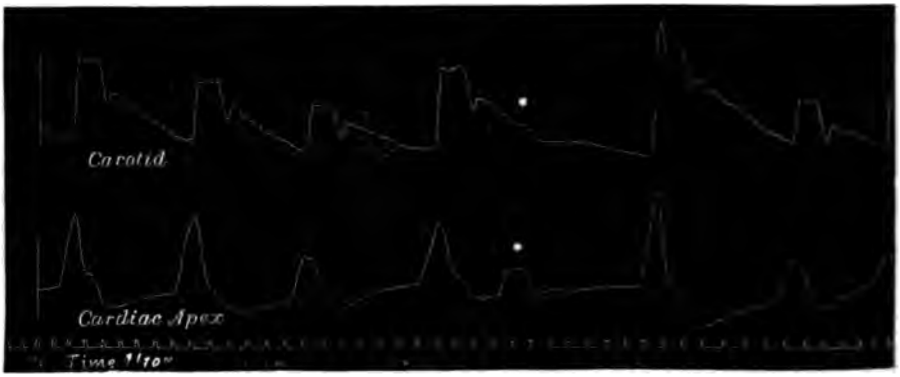

FIG. 345.—Tracing of the carotid artery and cardiac apex in chronic myocarditis showing irregularity in time and in force. * Ventricular contraction not of sufficient strength to produce a distinct carotid wave.

GENERAL ARTERIOSCLEROSIS IN CHRONIC MYOCARDITIS.—Habitual increase in the arterial blood-pressure is an important cause of chronic myocarditis. Prolonged muscular exertion, toxic substances, and arteriosclerosis alone or associated with chronic disease of the kidneys are potent factors. The changes in the myocardium develop gradually, and the symptoms and course of the disease are not different from those of the ordinary form. Middle-aged men who have lived well suffer from this form and the early symptoms in such cases are usually abdominal—fulness, weight, flatulence, and constipation.

SYPHILIS OF THE HEART.—Single or multiple gummata are very rare. Diffuse interstitial myocarditis is more common. Syphilis of the heart may be congenital or acquired. In the acquired form it is usually a tertiary manifestation. The condition is very often latent, but when well developed it corresponds clinically to the ordinary form of chronic myocarditis.

NUTRITIONAL DISORDERS AND CHRONIC MYOCARDITIS.—The myocardium suffers in the same way as the skeletal muscles. Previously existing myocarditis constitutes an important predisposing influence. The cardiac insufficiency may show itself in the morning when the duties of the day are undertaken after an insufficient breakfast. Free purgation may cause symptoms. A too strict antidiabetic diet and the injudicious treatment of obesity are to be considered. This form of cardiac inadequacy is common in the gouty, partly because of anomalous metabolism, partly because of insufficient food, and often as the result of an unwise use of colchicum or other drugs. To this category must also be referred the feeble heart of anæmia and chlorosis, together with the atrophy of the heart in the acute febrile infections, tuberculosis, and starvation. Long recumbency, as in illness or those who from other causes are bed-ridden, leads to gradual heart starvation. The symptoms are those of cardiac dilatation, dyspnœa upon exertion, gastrohepatic distress, œdema of the lower extremities, and general asthenia.

Diagnosis.—DIRECT.—Objective phenomena dependent upon changes in the heart upon which to base a diagnosis are usually absent. The anamnesis and the cardinal condition of weakness of the heart, as manifest by rational symptoms, justify a provisional diagnosis. A positive diagnosis may be reached by the method of exclusion. In a considerable proportion it cannot be reached at all.

In the anamnesis the age of the patient, his mode of life, the history of antecedent disease are important. The present condition of the superficial arteries, the blood-pressure, the ophthalmoscopic evidences of changes in the walls of the retinal arteries, the specific gravity of the urine and the presence or absence of albumin, tophaceous deposits in the ears or around the small joints, and the occurrence of asthma-like seizures, precordial pain, and angina pectoris are all to be carefully investigated. Dyspnœa, precordial and abdominal distress, diminution in the urine, and œdema are most significant symptoms; feebleness of the heart sounds, a weak or undiscoverable impulse, a faint or inconstant mitral systolic murmur are signs of importance. A marked increase in the pulse-frequency upon slight exertion is very suggestive.

The diagnosis of sclerosis of the coronary arteries may be made when, in a person of middle age presenting the signs of sclerosis of the aortic valves or atheroma of the ascending aorta, attacks of paroxysmal dyspnœa or angina pectoris occur in connection with the manifestations of cardiac inadequacy. The diagnosis of coronary embolism and thrombosis is usually impossible. These conditions frequently arise in advanced cases without any clinical manifestation whatever. The evidences of general arteriosclerosis in a case characterized by the signs of great and persistent cardiac weakness are of diagnostic importance. The symptoms of chronic nephritis, and, in particular, of contracted kidney, are also significant. Finally, transient attacks of increased cardiac asthenia in persons improperly nourished, overworked, much torn by the stress of life, or suffering from acute or chronic disease, are very often the manifestations of heart starvation.

DIFFERENTIAL.—*The Cardiac Neuroses.*—The differential diagnosis is rendered uncertain, especially in the early cases of chronic myocarditis, by the fact that the murmurs and especially neurasthenic symptoms are very common in myocarditis. A history of accidental or surgical trauma, overwork, anxiety, grief, onanism, sexual excess, hysteria, speak for a cardiac neurosis. The age of the patient is of some value in diagnosis. Under forty the neuroses are more common; after forty myocarditis.

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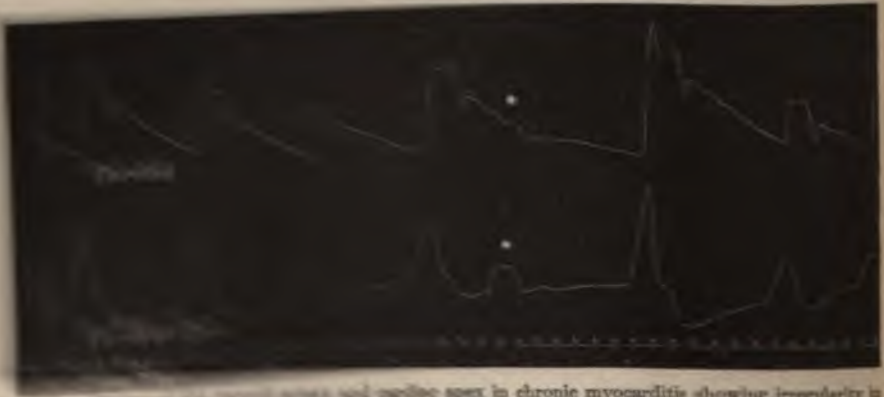

[illegible] and cardiac apex in chronic myocarditis showing irregularity in [illegible] contraction not of sufficient strength to produce a distinct carotid wave. [illegible]

[illegible] Diagnosis in Chronic Myocarditis.—Habitual [illegible] blood-pressure is an important cause of chronic [illegible] muscular exertion, toxic substances, and arterio[illegible] with chronic disease of the kidneys are potent [illegible] the myocardium develop gradually, and the symp[illegible] disease are not different from those of the ordinary [illegible] who have lived well suffer from this form and the [illegible] cases are usually abdominal—fulness, weight, [illegible].

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The various conditions which primarily or chiefly cause hypertrophy of the left ventricle ultimately cause also hypertrophy of the right ventricle. Hypertrophy of the right ventricle arises also in other conditions which increase the resistance in the pulmonary vessels, such as emphysema, fibrosis of the lungs, and deformities of the chest. The left auricle undergoes dilatation with hypertrophy in mitral disease, especially stenosis; the right in conditions characterized by increased blood-pressure in the pulmonary circuit, both of valvular and of pulmonary origin.

Enormous enlargement of the heart—*cor bovinum*—is usually due to aortic insufficiency, chronic mediastinitis, or chronic interstitial nephritis.

Hypertrophy of the heart is essentially a conservative process. Its development is gradual and for a time keeps pace with the advance of the lesions with which it is associated. So long as the equilibrium of the circulation is maintained, the hypertrophy is *compensatory;* when the hypertrophy begins to fail, the compensation is *impaired;* when dilatation is marked and cardiac insufficiency is extreme, the compensation is said to be *ruptured* or *broken.*

Moderate hypertrophy is unattended by marked direct symptoms. In fact it prevents to a great extent the manifestations of the underlying disease. Sensations of fulness, aggravated when lying upon the left side, easily induced overaction, and the consciousness of precordial pulsation or throbbing in the neck or head sometimes occur in advanced cases.

The physical signs depend upon the degree of hypertrophy and its preponderance over the accompanying dilatation, and the extent to which the heart is covered by the overlying borders of the lungs. When the hypertrophy is advanced and dilatation yet subordinated to it, the diagnosis may be readily made. Upon inspection the impulse is strong and extended to the left and downward. In marked enlargement it is heaving, and the whole precordial area may pulsate. There is pulsation at the root of the neck. A visible pulsation in the epigastrium occurs in hypertrophy of the right ventricle. Palpation confirms the signs obtained by inspection, and in women with large mammæ may enable the examiner to ascertain the position of the apex when inspection and percussion are ineffectual. The radial pulse is full, strong, and rather slow. Upon percussion the areas of relative and absolute dulness are usually both increased; in large-lunged persons and cases of emphysema, the relative dulness only. Extension of deep dulness to the left and upward is a sign of hypertrophy of the left ventricle; to the right and downward a sign of hypertrophy of the right ventricle. Upon auscultation the first sound is loud, often booming and prolonged; the second aortic sound accentuated in left ventricle hypertrophy, the second pulmonary accentuated when the right ventricle is involved. Accentuation of the second aortic occurs, however, in those conditions of high tension of the systemic arteries which cause left ventricle hypertrophy, and accentuation of the second pulmonary in those which interfere with the normal flow of blood through the pulmonary vessels.

(b) DILATATION OF THE HEART.

Definition.—Dilatation of the heart is an anatomical condition, characterized by an increase in the size of its chambers due to stretching of its walls. Dilatation may affect one or more chambers of the heart. It may be acute or chronic. Acute dilatation is usually primary; chronic dilatation secondary to cardiac insufficiency or valvular disease. A previously diseased heart is more liable to acute dilatation than a normal heart.

The capacity of the chambers of the heart varies considerably within normal limits. Pathological enlargement—*dilatation*—exists when the affected chamber is unable to empty itself of blood in systole, and is permanent. The myocardium, like other muscles, increases within certain limits with use, especially if the increase in work is gradual and nutrition is maintained.

Etiology.—Dilatation results from a disproportion between the power of the heart muscle and the work which it has to do. In other words, the pressure within the chambers may be increased or the muscular wall may be weakened. These factors may act singly or in combination. Increased pressure arises when there is an abnormal quantity of blood to be propelled or an abnormal resistance to be overcome. Under certain conditions, as in the gradually developing sclerotic form of aortic stenosis, and in the cardiovascular changes of chronic nephritis, the left ventricle may not undergo dilatation but hypertrophy.

Severe and prolonged muscular effort is a common cause of acute dilatation, as in mountain climbing or prolonged tests of endurance. The condition is known as heart strain. The symptoms are breathlessness upon exertion, cyanosis, lassitude, mental depression, and swelling of the ankles; the signs feebleness of the cardiac impulse, small, rapid, irregular and intermittent pulse, faint heart sounds, and upon percussion the evidences of enlargement of both the superficial and deep areas of dulness, increase in the area of liver dulness, and hypostatic congestion of the lungs. Relative insufficiency is shown by the development of a mitral systolic murmur, and the safety valve function of the tricuspid by a systolic murmur at the lower border of the sternum to the right. Under appropriate treatment, into which rest in the recumbent posture largely enters, gradual recovery takes place with disappearance of the signs of dilatation. In many cases they recur upon further unusual effort, and in some the damage to the wall of the heart is permanent. Acute dilatation occurs also in consequence of undue effort after acute illness, in Graves's disease, exophthalmic goitre, and paroxysmal tachycardia. It may follow any unusual effort in a case of chronic myocarditis.

The ultimate tendency of chronic myocarditis is to dilatation. In many forms the increase in the size of the chambers and stretching of the walls begin early; in others not until after an initial hypertrophy. The symptoms are those of cardiac insufficiency; the signs those of enlargement of the heart, displacement of the apex to the left and downward, feeble, undulating impulse extending over several intercostal spaces, and faint cardiac sounds reverting to the fetal type. Dropsy and the evidences of visceral congestion are present in well-marked cases.

Valvular disease is a constant cause of dilatation of the heart. In stenosis a portion of the normal quantity of blood that should pass the affected orifice is held back in systole; in incompetency a portion of the quantity that has passed the orifice flows back in diastole. In one case the blood entering the chamber meets blood that should have passed on; in the other blood enters the chamber in diastole from two opposite directions, one physiological, the other pathological. The result is dilatation of the affected chamber and transference of the increased blood-pressure backward from the site of the valvular lesion, from ventricle to auricle on the left side, through the pulmonary circuit, to ventricle and then to auricle on the right.

Hypertrophy at first, then dilatation of the left ventricle thus results from aortic stenosis and from aortic insufficiency; hypertrophy and dilatation of the left auricle from mitral stenosis and mitral insufficiency; pulmonary hyperæmia from either stenosis or insufficiency of the aortic or mitral valve systems; hypertrophy and dilatation of the right ventricle from pulmonary hyperæmia due to valvular disease of the left side of the heart, or to intrapulmonary conditions which increase the resistance in the pulmonary circuit; hypertrophy and dilatation of the right auricle from overdistention of the right ventricle.

Whenever dilatation is in excess of hypertrophy there is a tendency to the transference of blood-pressure from the arterial to the venous side of the circulation.

When the myocardium is weakened by, (a) the toxins of the acute infections, (b) the extension of the inflammation in endocarditis or pericarditis, (c) the malnutrition of starvation in any form, anæmia, or chlorosis, dilatation may result in the absence of increase in the blood-pressure.

iv. Fatty Heart.

Fatty Overgrowth; Fatty Infiltration; Cardiac Inadequacy of the Obese; Cor Adiposum.

Definition. — A condition common in fat persons, characterized anatomically by excess of fat beneath the epicardium and among the strands of muscular fibres, and clinically by cardiac insufficiency.

It has been customary to include, under the designation fatty heart, fatty degeneration of the heart muscle and fat overgrowth or infiltration.

lunged persons and cases of emphysema, the relative dulness only. Extension of deep dulness to the left and upward is a sign of hypertrophy of the left ventricle; to the right and downward a sign of hypertrophy of the right ventricle. Upon auscultation the first sound is loud, often booming and prolonged; the second aortic sound accentuated in left ventricle hypertrophy, the second pulmonary accentuated when the right ventricle is involved. Accentuation of the second aortic occurs, however, in those conditions of high tension of the systemic arteries which cause left ventricle hypertrophy, and accentuation of the second pulmonary in those which interfere with the normal flow of blood through the pulmonary vessels.

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[illegible] of the heart varies considerably within [illegible]—exists when the [illegible] of blood in systole and is per[illegible] increases within certain [illegible] work is gradual and nutrition [illegible]

Etiology. [illegible] proportion between the power [illegible] In other words, the [illegible] wall may [illegible] nutrition. Increased [illegible] quantity of blood to be propelled [illegible] Under certain conditions, as [illegible] stenosis, and in the [illegible] the left ventricle may not [illegible]

[illegible] is a common cause of acute [illegible] tests of endurance. The [illegible] symptoms are breathlessness [illegible] and swelling of the [illegible] small, rapid, irregular [illegible] the evi[illegible] areas of dulness. [illegible] Under appropriate treat[illegible] largely [illegible] gradual [illegible] signs of [illegible] In [illegible] the damage [illegible] Acute dilatation occurs also in consequence of undue effort after acute illness, in Graves's disease, exophthalmic goitre, and paroxysmal tachycardia. It may follow any unusual effort in a case of chronic myocarditis.

The ultimate tendency of chronic myocarditis is to dilatation. In many forms the increase in the size of the chambers and stretching of the walls begin early; in others not until after an initial hypertrophy. The symptoms are those of cardiac insufficiency; the signs those of enlargement of the heart, displacement of the apex to the left and downward, feeble, undulating impulse extending over several intercostal spaces, and faint cardiac sounds reverting to the fetal type. Dropsy and the evidences of visceral congestion are present in well-marked cases.

Valvular disease is a constant cause of dilatation of the heart. In stenosis a portion of the normal quantity of blood that should pass the affected orifice is held back in systole; in incompetency a portion of the quantity that has passed the orifice flows back in diastole. In one case the blood entering the chamber meets blood that should have passed on; in the other blood enters the chamber in diastole from two opposite directions, one physiological, the other pathological. The result is dilatation of the affected chamber and transference of the increased blood-pressure backward from the site of the valvular lesion, from ventricle to auricle on the left side, through the pulmonary circuit, to ventricle and then to auricle on the right.

Hypertrophy at first, then dilatation of the left ventricle thus results from aortic stenosis and from aortic insufficiency; hypertrophy and dilatation of the left auricle from mitral stenosis and mitral insufficiency; pulmonary hyperæmia from either stenosis or insufficiency of the aortic or mitral valve systems; hypertrophy and dilatation of the right ventricle from pulmonary hyperæmia due to valvular disease of the left side of the heart, or to intrapulmonary conditions which increase the resistance in the pulmonary circuit; hypertrophy and dilatation of the right auricle from overdistention of the right ventricle.

Whenever dilatation is in excess of hypertrophy there is a tendency to the transference of blood-pressure from the arterial to the venous side of the circulation.

When the myocardium is weakened by, (a) the toxins of the acute infections, (b) the extension of the inflammation in endocarditis or pericarditis, (c) the malnutrition of starvation in any form, anæmia, or chlorosis, dilatation may result in the absence of increase in the blood-pressure.

iv. Fatty Heart.

Fatty Overgrowth; Fatty Infiltration; Cardiac Inadequacy of the Obese; Cor Adiposum.

Definition. — A condition common in fat persons, characterized anatomically by excess of fat beneath the epicardium and among the strands of muscular fibres, and clinically by cardiac insufficiency.

It has been customary to include, under the designation fatty heart, fatty degeneration of the heart muscle and fat overgrowth or infiltration.

The present tendency, however, is to restrict the term to the peculiar changes that occur in corpulent persons, since fatty degeneration is a common form of parenchymatous degeneration in myocarditis due to many different causes, and wholly without distinctive etiological or clinical features.

Fatty degeneration of the heart is common in the infectious fevers, wasting diseases, and the cachexias. It is very marked in acute yellow atrophy of the liver, phosphorus poisoning, and pernicious anæmia. It constitutes one of the most important changes of old age. The degeneration may also under all these conditions affect the various viscera. The wall of the heart may be locally or generally involved. The heart muscle is flabby, relaxed, and friable. The color is that of the "faded leaf." The heart when thrown upon the table sinks into a shapeless mass. Microscopically the fibres are filled with minute fat globules.

The condition under consideration is entirely different. It is essentially an affection of the corpulent. There is an excess of the subpericardial fat, so great in some instances as to wholly envelop the muscle in a casing of fat. It is usually more abundant in the intraventricular grooves, along the course of the coronary arteries, and upon the wall of the right ventricle. It penetrates the muscles, separating the fibres, and may extend to the endocardium. The heart is dilated and its wall flabby and relaxed. Upon microscopical examination the muscular fibres are found to be atrophied and in some instances to have undergone fatty degeneration. There is a disproportion between the size of the heart and the requirements of the body. In many fat persons with well-developed muscles cardiac insufficiency does not occur. Cardiac symptoms are marked in that type of obesity characterized by anæmia, flabby muscles, and indolence.

Etiology.—The causes of fatty infiltration of the heart are those of the obesity of which it constitutes such an important part. The middle periods of life, the male sex, heredity, addiction to the pleasures of the table, much fluid, malt liquors and alcohol in general, luxurious habits, and indolence are potent factors in the production of corpulence and the fatty heart.

Symptoms.—The clinical manifestations are those of cardiac inadequacy, dyspnœa, a feeble pulse, much accelerated upon effort and commonly intermittent and irregular. Poor appetite, much thirst, and constipation are common. Such patients are often drowsy by day and sleepless by night. Dropsy of the lower extremities is sometimes marked.

Physical Signs.—The methods of physical examination usually yield unsatisfactory results by reason of the excess of subcutaneous fat. This is particularly true of inspection, palpation, and percussion. The results of auscultation are sometimes more satisfactory. The heart sounds are usually feeble and distant. When, however, they are well defined and the aortic sound distinct, they may be regarded as indicating fairly well-maintained myocardial nutrition. When, on the other hand, the first sound is extremely faint or replaced by a systolic murmur, the aortic sound feeble, and the pulmonic sound accentuated though feeble, the integrity of the heart muscle is greatly impaired.

Diagnosis.—The recognition of fatty heart depends upon the association of cardiac insufficiency with obesity.

The DIFFERENTIAL DIAGNOSIS between this condition and the primary anæmias, especially chlorosis and pernicious anæmia, may be made by a proper blood examination. The blood of corpulent persons of the pallid type who suffer from fatty heart may show the characters of a more or less marked secondary anæmia, but not, in the absence of specific lesions, the characters of the primary anæmias.

Prognosis.—The outlook in general is unfavorable and is rendered more so by unwise attempts to reduce the weight of the body by insufficient food, unduly increased exercise, exhausting baths, or depressing drugs. Thickened arteries, paroxysmal dyspnœa, and angina pectoris are of ominous significance.

v. Various Degenerations, New Growths, and Parasites of the Heart.

Degenerations of the heart muscle not already considered are amy- oid degeneration, the hyaline transformation of Zenker, and calcareous nfiltration. None of these is recognizable during life nor of clinical nterest.

Tumors of the heart are commonly carcinoma and sarcoma. They e usually secondary. Fibroma, lipoma, myoma, gumma, and leukæmic filtrations are extremely rare. Malignant tumors very commonly lead pericarditis, which may be plastic or purulent.

Of the parasites which affect the myocardium, the echinococcus is the ost common. It selects the right ventricle twice as often as the left. long as it remains within the myocardium it does not occasion symptoms. hen it finds its way into the interior of the heart it gives rise to embolism, ecially in the lungs. The cysticercus and trichinella find access to the rt muscle, but do not occasion symptoms.

vi. Wounds and Foreign Bodies.

External injuries, as stabs and gunshot wounds, are very common. ir diagnosis is obvious. The subject belongs to surgery, and has ac- ed great importance in consequence of the recent success which, in wounds, has attended the prompt exposure of the heart and suturing e wound. Internal injuries are extremely rare. They are caused by gn bodies—a bone or artificial denture ulcerating its way from the hagus, or in the case of insane or hysterical persons by pins or needles have been swallowed. In the former instance the nature of the lesion d be recognized by the history of the case and sudden fatal hæma- sis, in the latter pericarditis would occur; but a positive etiological osis *intra vitam* cannot be made.

vii. Rupture of the Heart.

his accident may occur as the result of the arrest of the blood supply affected area in consequence of sclerosis or embolism of a branch oronary artery, inducing acute softening—*myomalacia cordis*. The heart wall may undergo similar circumscribed impairment from suppurative myocarditis or a softening gumma. Local fatty degeneration is the most common cause. The rupture occurs most frequently on the anterior wall of the left ventricle near the septum. The softened area gradually yields, and upon some effort which causes heightened intraventricular pressure, as ascending a staircase or straining at stool, it suddenly gives way and the escape of blood into the pericardial sac—*hæmopericardium*—is followed by death. Rupture in the posterior wall of the left ventricle is much less common, and rupture of the wall of the right ventricle or the auricles very rare.

Traumatic rupture of the heart may result from violent blows or contusions of the thorax, such as occur in falls or railroad accidents. This variety of heart rupture is more apt to involve the right ventricle or an auricle. In some cases the borders of the rent maintain their position. Pericardial adhesions may occur and death may be postponed for several hours or days.

Diagnosis.—In the majority of the cases death results at once and the diagnosis is impossible. When the opening is small, signs of internal hemorrhage—feeble pulse, oppression, air hunger, ghastly pallor, and orthopnœa—are suggestive. When pericardial adhesions exist the blood outflow is hindered and life may be correspondingly prolonged.

Prognosis.—The outlook, however, is without hope.

viii. Aneurism of the Heart.

Aneurism of a valve may result from malignant endocarditis. The condition is not common. The aortic valves are affected with greater frequency than the mitral. The cusp shows a bulging in the direction of the ventricle, which presently ruptures, causing acute insufficiency. The signs are not characteristic and are obscured by the primary changes. A positive diagnosis cannot be made.

Aneurism of the wall is also a rare condition. Its most common position is the left ventricle in the region of the apex, which is the portion of the wall of the heart most commonly affected in the fibroid degeneration of chronic myocarditis.

Etiology.—This condition mostly follows chronic myocarditis, but has been observed in acute mural endocarditis. Wounds of the heart and gumma are also etiological factors. The dilatation is usually single, but may be multiple.

Diagnosis.—DIRECT.—The symptoms are not characteristic. The associated myocarditis causes cardiac inadequacy, manifest by the usual clinical phenomena. When the tumor attains considerable size it is usually lined by laminated clots and may give rise to irregular enlargement of the diameters of deep cardiac dulness. In other cases there may be bulging in the region of the apex and perforation of the wall of the chest.

The DIFFERENTIAL DIAGNOSIS from mediastinal or pleural tumor is to be considered. The feeble pulse of cardiac inadequacy may be in marked contrast to the cardiac impulse. The X-rays may be of service in the differential diagnosis.

ix. Atrophy of the Heart.

Definition.—A diminution of the heart in weight and size. A single amber or the entire heart may be atrophied.

The term hypoplasia of the heart is used to designate congenital dersize.

The myocardium is of a dark, reddish-brown color, and abnormally sistant. The surface is often marked or puckered. The muscle fibres e diminished in size, their transverse striæ indistinct and presenting llections of yellowish-brown pigment near the nuclei.

Etiology.—The small size of the left ventricle in extreme mitral steno- may be looked upon as an example of atrophy of a single chamber of e heart. Common causes are starvation and wasting diseases, as cancer, abetes, protracted suppuration, and, in particular, phthisis. Brown rophy of the heart is common in advanced valvular disease and old age— *senile heart.*

Symptoms.—The symptoms are those of cardiac inadequacy—feeble d rapid action, especially upon exertion, weak and irregular pulse, faint unds, and indistinct impulse. The shrunken lungs usually increase the ea of superficial dulness, but the diameters of deep dulness are reduced.

Diagnosis.—The calcification of the costal cartilages in the aged often nders the examination of the heart by percussion very difficult and satisfactory. The X-ray examination yields more definite signs of a luction in the size of the organ. The clinical phenomena are much bordinated to those of the primary affection.

Prognosis.—The outlook is that of the primary disease. The ultimate ure of the circulation is often largely due to cardiac atrophy.

III. DISEASES OF THE PERICARDIUM.

i. Pericarditis.

Definition.—Inflammation of the pericardium resulting from trau- ism, infection, the extension of inflammation from contiguous ctures, or toxic conditions.

Etiology.—Idiopathic or spontaneous pericarditis is a purely theo- al conception. The extremely rare cases of pericarditis in children out other indications of local or constitutional disease are probably to latent tuberculosis, or tonsillitis or other infection, or to an obscure mia. Traumatic pericarditis may become the subject of medical osis when the injury is from within, as in the case of the ulceration of ign body from the œsophagus, or injury by needles or pins that have swallowed. Infection is the most common cause. The greater num- f cases occur in connection with rheumatic fever. The pericarditis precede the joint affection. Next in frequency are the cases due to culosis. To this etiological group are to be referred the cases of rditis which follow blows and contusions of the chest, and those which in alcoholics. The pericarditis may, for a time, be the only clinical station of the tuberculous infection. Less commonly pericarditis is secondary to sepsis, especially that caused by acute necrosis or puerperal infection, or the toxæmia of scarlet fever and the other acute febrile infections.

Extension of the inflammation from the endocardium may account for the common association of endo- and pericarditis in rheumatic fever, or the later pericardial inflammation may be also a direct manifestation of rheumatism. This mode of infection is common in pleurisy and pneumonia, and may occur in œsophageal carcinoma, tuberculous or bronchiectatic cavities closely adjacent to the pericardium, tuberculous mediastinitis, perforating gastric ulcer, or subphrenic abscess. That form which sometimes occurs in purulent myocarditis, ulcerative endocarditis, aneurism of the aorta, disease of the ribs and sternum or the vertebræ arises in a majority of the cases by direct extension. Toxic pericarditis is not rarely a terminal condition in chronic nephritis, especially the interstitial variety. It is occasionally present but usually latent in gout, scurvy, diabetes, and arteriosclerosis. Among the infrequent causes of pericarditis are syphilis, carcinoma and sarcoma of the pericardium, echinococci or cysticerci, and actinomycosis. The micro-organisms most frequently encountered in the exudate are the ordinary pyogenic bactéria, the pneumococcus, and the tubercle bacillus.

The inflammatory exudate may be fibrinous, serofibrinous, hemorrhagic, or purulent. The terminal condition in cases that recover is that of more or less complete adhesion between the pericardial surfaces—*adherent pericardium*. It is customary to describe separately dry or fibrinous pericarditis, pericarditis with effusion, and adherent pericardium; but it is important to bear in mind the fact that these, in a majority of instances, are successive stages in a continuous process. Like other inflammations, pericarditis may be acute or chronic.

(a) FIBRINOUS, PLASTIC, OR DRY PERICARDITIS.

Pericarditis Sicca.

In the simple acute cases the inflammation involves first the epicardial or visceral layer; later the pericardial layer of the serous pericardium. The fibrinous exudate may be circumscribed or general. Its arrangement varies greatly. Sometimes it presents the appearance seen when two buttered surfaces are separated; sometimes there are hairy ridges in irregular parallel lines,—*cor villosum,*—or again there may be a stratified or a honeycombed appearance. There is, as a rule, a variable amount of fluid entangled in the meshes of the fibrin, but in chronic tuberculous cases with great thickening fluid is absent. The myocardium immediately subjacent is inflamed. The frequent coexistence of endocarditis is of clinical as well as etiological interest.

Symptoms.—Plastic pericarditis is sometimes latent. Even in marked cases the subjective phenomena may be indefinite. Pain is common. It is usually substernal or referred to the region of the apex. Less frequently it radiates to the neck and arm, especially on the left side. It may be stitch-like and lancinating, or dull and heavy; persistent or paroxysmal.

The fever of the primary disease may be aggravated, but the terminal pericarditis of nephritis may be unattended by a rise of temperature.

Physical Signs.—Upon inspection the signs are usually negative. The breathing may be rapid and shallow or there may be orthopnœa. Palpation in a considerable proportion of the cases reveals a more or less distinct friction fremitus. The pulse is usually accelerated—120 to 140 to the minute. The percussion borders of the heart are not enlarged in simple fibrinous pericarditis. In old cases the myocardium may undergo dilatation. AUSCULTATION.—The pericardial friction sound is most variable in character. It is frequently of a soft grazing or brushing quality. More commonly it is rubbing or grating and has been compared with the creaking of new leather. It appears to be superficial, as though produced close to the surface, and is increased by moderate pressure, in some cases obliterated by strong pressure with the stethoscope. Its intensity varies from a scarcely audible whiff to a loud coarse sound, directly appreciable to the ear. Its loudness is not dependent upon the amount of fibrinous exudate. It is sometimes absent when the fibrin is abundant, as in *cor villosum;* sometimes distinct when there is merely a thin layer. The intensity is modified by posture and undergoes remarkable changes from day to day in the course of the attack. The pericardial friction sound is usually to-and-fro, corresponding to the systole and diastole of the ventricles, but it does not bear the definite relations of endocardial murmurs to the cardiac cycle. It is sometimes single and in rare instances triple, having a somewhat irregular canter rhythm. Its systolic and diastolic portions have usually, but not invariably, the same quality and pitch. They are almost always of unequal length. It is heard over the body of the heart; sometimes most distinctly in the second, third, and fourth left intercostal spaces and adjacent parts of the sternum, sometimes at the base over the pericardial reflections upon the great vessels, and again in the region of the apex. It is usually limited to a small area, but may be distinctly heard over a large part of the pericardium. It is, however, always circumscribed and never transmitted beyond the boundaries of the heart in definite lines corresponding to the vessels, as is the case with endocardial murmurs. When pericardial effusion takes place the friction sound disappears over the body of the heart, but, except in large effusions, may still be heard in a limited region at the base.

Diagnosis.—The DIRECT DIAGNOSIS of fibrinous pericarditis depends upon the recognition of a friction sound having the foregoing characters, location, and correspondence to the revolution of the heart.

DIFFERENTIAL.—The distinction between endocardial murmurs and exocardial friction sounds is based upon the well-recognized characters of each. A double aortic murmur, particularly when accompanied by a thrill, may lead to error, but not if due heed be given to its sameness from time to time, lines of propagation, the correspondence of its systolic and diastolic elements with the cycle of the heart, and the associated arterial changes.

Pleurisy.—Pleural friction is not usually restricted to the cardiac borders of the lung, and when heard elsewhere serves to explain a pleuropericardial friction, which is due to movements of the pleural surfaces induced by the action of the heart. This sign is by no means infrequent at the left anterior margin of the lung in croupous pneumonia, and is sometimes encountered in phthisis. It disappears upon full-held inspiration, and on ordinary breathing is more distinct during the expiratory period. Clicking and crepitant râles, occasionally heard in the region of the apex and recurring with the ventricular systole, are readily differentiated from pericardial friction.

It is stated that pericardial friction sounds are sometimes produced by milk spots on the surface of the ventricles, concretions, and in the dry condition of the tissues occurring in cholera, but such conditions are not to be confounded with true pericarditis.

Prognosis.—The course of fibrinous pericarditis as such is favorable. In some days or weeks recovery may take place without any clinical manifestation of injury to the heart. The inflammation may indeed run its course in the absence of subjective phenomena, the friction sound being the only objective sign. More commonly there are more or less urgent symptoms. The danger of serofibrinous, hemorrhagic, or purulent effusion, of implication of the myocardium with acute symptoms, and of extensive pericardial adhesions leading to chronic myocarditis invests every case with importance. As an intercurrent affection it adds to the gravity of the primary disease.

(b) PERICARDITIS WITH EFFUSION.

Pericarditis Exudativa.

There is no abrupt line of separation between dry pericarditis and pericarditis with effusion. In the meshes of an abundant fibrinous exudate there are small collections of serum; in serofibrinous effusions the pericardial surfaces are covered with fibrin, flakes of which float free in the fluid. The effusion may be serofibrinous, hemorrhagic, or purulent.

Etiologically serofibrinous effusion is usually a so-called second stage in the evolution of the attack of pericarditis, and may arise in any of the conditions in which plastic pericarditis occurs. Blood elements are present in varying amounts. Hemorrhagic effusions owe their characteristic appearance to an excess of blood. They are met with in tuberculous and cancerous pericarditis, and in those forms which occur in hemorrhagic conditions, as scorbutus and purpura, and in the aged. The quantity of blood varies from an amount only appreciable upon microscopical or chemical examination to almost pure blood. The effusion may be purulent in tuberculous cases. It is likely to be so in those due to sepsis or internal or external traumatism, or when an effusion arises in consequence of infection from a contiguous bronchiectatic cavity or vomica. The volume of the effusion is extremely variable. Experimentally the normal sac will contain without distention 150 to 200 c.c. Upon forcible distention, with compression of the heart, from 500 to 800 c.c. may be injected. The inflamed pericardium is more distensible, and with the adjustments which take place under the gradual accumulation of an effusion, as much as 1500 or even 2000 c.c. have been observed.

Symptoms.—The condition is frequently latent. There are cases in which moderate pericardial effusions run a favorable course without heart symptoms, resorption taking place in the course of two or three weeks. More commonly the early symptoms consist of chilliness, precordial pain, and fever. In well-developed cases there are two main groups of symptoms, constitutional and local. The constitutional symptoms are very often masked by those of the primary affection, and moderate terminal effusions are more frequently recognized in the post-mortem room than in the ward. In children general symptoms, as feverishness, dyspnœa, loss of appetite, fretfulness, languor, and a rapidly developing pallor, may occur in the absence of precordial pain or other symptoms suggestive of the actual condition. Pallor, weakness, insomnia, loss of appetite, dyspnœa and orthopnœa, melancholia and a disposition to suicide, and in grave cases restlessness, somnolence, delirium, and a tendency to coma are among the symptoms of pericardial effusion. The local symptoms arise from the inflammation, from the derangement of the circulation, and from pressure. The pain is referred to the precordium; less commonly to the epigastrium. It is usually sharp and lancinating; sometimes dull and aching; exceptionally it amounts only to a sense of distress and discomfort. It is usually continuous with exacerbations, but may be paroxysmal with intervals of relief. It is intensified by pressure with the stethoscope. Derangements of the circulation are manifest in cyanosis of varying intensity, shortness of breath, anxiety, and the sensation of air hunger. The patient prefers to lie upon the left side; in large effusions he is obliged to be propped up with pillows in the semirecumbent posture or to sit up in bed. The pulse is rapid, small, and frequently arrhythmic. In large effusions with thickening of the parietal pericardium the pulse may become very feeble or quite imperceptible during inspiration—*pulsus paradoxicus*. It is sometimes smaller in the left than in the right carotid and radial arteries. The circulatory symptoms are due in part to the direct pressure of the effusion upon the heart, the effects of which are greater upon the thin-walled auricles than upon the ventricles, and to the implication of the myocardium directly in relation with the inflamed epicardium. The symptoms due to pressure upon other organs are a sense of precordial oppression and weight in the epigastrium, dysphagia, aphonia, a laryngeal cough, distention of the veins of the neck, and dyspnœa from compression of the left lung.

Physical Signs.—INSPECTION.—In small effusions there are no distinctive signs. In moderate and large effusions the respiratory excursus upon the left side is diminished in consequence of pressure atelectasis of the lower lobe. Pericardial effusions compress the left lung to a far greater extent than the right. The epigastrium is prominent, owing to the depression of the diaphragm and liver. In children and young persons precordial prominence, widening and slight bulging of the lower intercostal spaces, and in some cases a feeble wavy cardiac impulse may be present. PALPATION.—A cardiac impulse due to the contraction of the right ventricle may be feebly felt in the fourth interspace; in other cases the apex beat may be lower than normal in consequence of the depression of the diaphragm. Very often no precordial impulse can be detected. Friction fremitus vanishes as the layers of the pericardium are separated by the effusion, except at the base, where it may sometimes be felt, especially in the erect posture. Fluctuation is not a sign of pericardial effusion. In massive effusions bulging of the left retroclavicular space has been observed and elevation of the clavicle, so that the first rib may be palpated to the sternum—*first rib sign.* PERCUSSION.—This method of physical diagnosis yields most important signs. The effusion collects first in the most dependent part of the sac and gradually rises as it increases in amount. Its presence may be first appreciated by an absence of resonance at the sternal end of the fifth right intercostal space—the cardiohepatic angle. At this point in normal and dilated hearts the vertical border of the cardiac dulness and the transverse upper border of the hepatic dulness make a well-defined right angle. In early effusion and in certain cases of obesity this angle is replaced by a curve having its concavity upward and outward toward the lung—*Rotch's sign.* As the effusion increases the precordial dulness extends toward the left and upward, later toward the right, displacing the borders of the lung and forming at first a quadrilateral area of dulness with rounded corners, which with larger effusions assumes a pear-shaped outline, the larger end lying at the inferior border and extending beyond the sternal margin on the right, and beyond the position of the apex and the midclavicular line on the left. In large effusions the dulness may invade Traube's semilunar space. The truncated apex of this figure reaches into the upper sternal region. As the diameters of this figure are gradually reached, the area of superficial precordial dulness advances more rapidly than that of the deep or absolute dulness, until at length they nearly coincide. A circumscribed area of dulness or flatness may sometimes be found at the base of the left chest posteriorly between the inferior angle of the scapula and the vertebræ. Old pericardial adhesions, compression of the left lung, and coexistent pleural effusions greatly modify these changes in the percussion signs. AUSCULTATION.—The friction sound disappears over the body of the heart but may be heard at the base, very rarely at the apex. The first sound is obscure and indistinct; the second pulmo-

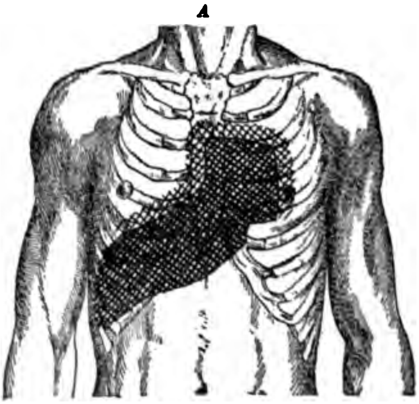

FIG. 346*A*.—Moderate pericardial effusion; quadrilateral flatness with border of relative dulness; effacement of cardiohepatic angle.

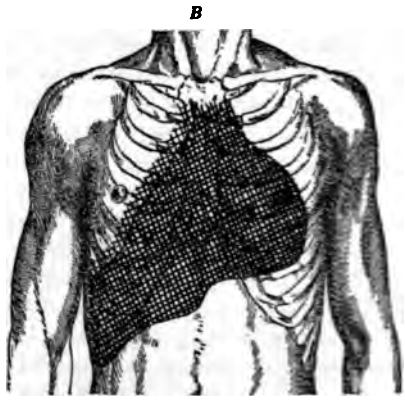

FIG. 346*B*.—Massive pericardial effusion; pyramidal area of flatness with truncated apex; right border of relative dulness; downward displacement of liver.

nary sound accentuated. The action of the heart is rapid and often arrhythmic. A systolic endocardial murmur may sometimes be detected. As resorption takes place the first sound becomes more distinct and the friction sound may be again heard.

Diagnosis.—The DIRECT DIAGNOSIS of pericardial effusion may be made without difficulty when the case has been seen from the outset and the above-described percussion signs have supervened upon pericardial friction sounds. Of especial value in the early recognition of effusion is Rotch's modification of the cardiohepatic angle. Very important is the triangular outline of dulness in large effusions. A sign too little appreciated is the progressive encroachment of the borders of the superficial dulness upon the area of deep dulness. The X-ray examination may be of service.

DIFFERENTIAL.—*Dilatation of the heart* very often presents extreme difficulty in the differential diagnosis. Careful clinicians have tapped the right ventricle instead of a pericardial sac distended with fluid. Dulness n the cardiohepatic angle, a quadrilateral area of dulness with rounded orners, the close approach of the borders of superficial to those of deep ulness, especially when the dulness extends to the left beyond the apex eat, constitute an association of physical signs of great importance. The runcated apex of the triangular area of dulness in large effusions, a cirumscribed area of dulness near the angle of the left scapula, and signs of ompression of the left lung have also diagnostic value. But most of these onditions may be present in dilated heart. In some cases of hypertrophy f the right ventricle the deep cardiac dulness due to the left ventricle xtends beyond the position of the visible impulse. An undulatory npulse seen or felt in two or more interspaces, distinct though feeble eart sounds, valvular in character and having the fetal rhythm, and stural changes in the upper borders of the dulness, are signs suggestive effusion. Overfilled veins, cyanosis, aphonia, dysphagia, and other essure symptoms are without value in the differential diagnosis, since ey may occur alike in large pericardial effusions and extreme dilatation the heart.

Left-sided Pleural Effusion.—This condition, unless the fluid be ensted, is not often mistaken for pericardial effusion, but large pericardial usions may closely simulate pleurisy. In the latter condition the heart isplaced toward the right, its impulse and sounds are distinct, the flats extends around the base of the chest, the overlying compressed lung lds tympanitic percussion resonance, Traube's semilunar space is oblitted, the spleen is displaced downward and its respiratory excursus ricted, and finally dysphagia is not a pressure symptom in pleurisy. al fremitus is usually distinct over an atelectatic lung, feeble or absent r fluid. The pericardial effusions which occasionally occur in pneuia present unusual difficulties in diagnosis. The signs are masked by e of the primary lesion. There is no border-line change from dulness learness or tympany; at the left border of the heart the extension of ess to the right of the sternum may be ascribed to the dilatation of a g right heart. The modification of the cardiohepatic angle would est pericarditis, but it is likely to be overlooked.

The recognition of tumors of the lung, pleura, or mediastinum, or of aneurisms of the aortic or pulmonary artery, depends upon a clinical course and physical signs that are widely divergent from those of pericarditis with effusion.

The character of the fluid can be determined with certainty only by an exploratory puncture. Paracentesis of the pericardium for diagnostic purposes cannot, however, be considered a justifiable procedure. When performed as a measure of treatment the gross and microscopical characters of the fluid are of diagnostic importance. Various sites are recommended for the insertion of the needle. The fourth or fifth left intercostal space near the sternum; the same interspaces to the left of the midclavicular line and within the border of the flatness on percussion; a point high in the angle formed by the ensiform cartilage and the left costal margin and the fifth right interspace 2 cm. from the sternal border when this area is flat upon percussion, are situations recommended. A small aspirator needle should be employed and strict surgical antisepsis observed. In rheumatic, renal, and tuberculous cases the fluid is usually serofibrinous; in senile, purpuric, and cancerous cases hemorrhagic; and in septic conditions it is commonly purulent. The gravity of the general symptoms usually depends upon the nature and intensity of the primary disease. When this is not the case it corresponds rather to the severity of the pericarditis, the amount of the effusion, and the rapidity with which it is formed, than to its character.

Prognosis.—The signs of effusion may, in many cases, be recognized within a few days of the detection of the friction sound. The accumulation in rheumatic fever, nephritis, scurvy, and some septic cases is rapid, while in tuberculous cases it is usually slow. Serofibrinous effusions of moderate volume frequently undergo resorption, which may be complete as shown by the retrogression of the dulness in the course of four or five weeks. Reappearance of the friction sound may occur but is less common than in pleural effusions. Occasional post-mortem findings, namely, grayish material in various stages of calcareous change in the pericardium, render it probable that under certain circumstances a purulent effusion may undergo resorption. Very large effusions show little tendency to undergo resorption, and unless removed by operative measures—dissection layer by layer, or paracentesis—rapidly prove fatal by compression of the heart and other mechanical effects. Purulent effusions, unless relieved by operation and drainage, terminate, as a rule, in death. The pericarditis with effusion in scurvy, chronic nephritis, and pyæmia is almost always a terminal condition. Heart complications are frequently present. There is myocarditis involving the myocardium directly in relation with the inflamed epicardium. Endocarditis is often also present, particularly in the rheumatic cases. Old valvular lesions with associated myocardial changes may at the same time obscure the diagnosis and unfavorably affect the prognosis. When recovery takes place the pericardial surfaces become adherent. The prognosis is relatively unfavorable as the primary constitutional condition is grave, the effusion large, and its accumulation rapid.

ii. Adherent Pericardium.

Synechia Pericardii; Obliteration of the Pericardial Sac.

Pericardial adhesions constitute a constant anatomical sequel of pericarditis, both in its latent and manifest forms. The extent of the adhesions is exceedingly variable. In many cases there are merely thread-like strings or bands of organized tissue extending from the visceral to the parietal pericardium; in others the adhesion between their membranes is universal and so close as to suggest congenital absence of the pericardium. The adjacent pleura is frequently involved and in extreme cases the heart is embedded in a dense, thick connective-tissue mass including the fibrous pericardium and the structures with which it is in relation—chronic adhesive mediastinitis.

The cases of simple pericardial adhesion may be divided into two groups:

(a) Those presenting no clinical manifestations and found upon post-mortem examination. This group includes the cases of limited adhesions and the threads and bands which scarcely affect the free movement of the heart within the sac, and some of the cases in which more general adhesions exist.

(b) Those in which, as a result of the pericarditis and adhesions, chronic myocarditis has occurred, with hypertrophy and dilatation and the symptoms of cardiac inadequacy. These cases do not always show general obliteration of the sac. A high grade of hypertrophy may occur with only partial adhesion between the layers.

Symptoms.—There are cases in which the history and physical signs are positive, but for a long period symptoms of heart disease are absent. After a time the symptoms are those of hypertrophy, then dilatation and a failing heart.

Physical Signs.—INSPECTION.—In young persons there may be prominence of the pericardium in consequence of hypertrophy, with an impulse visible in the fourth, fifth, and sometimes the sixth interspace and to the left of the midclavicular line. While the hypertrophy remains marked the impulse may be strong and heaving, but when it gives way to dilatation the impulse becomes more extended and undulatory, and there is systolic retraction in the neighborhood of the apex. The systolic indrawing may extend to other parts of the cardiac area. It is sometimes seen at the base of the heart and may be confined to this region. An energetic retraction in the parts about the ensiform cartilage upon the left is sometimes seen. In cases in which there are strong cardiodiaphragmatic adhesions a visible systolic retraction may be detected in the lower left ribs and interspaces behind—*Broadbent's sign*. Respiratory movement of the epigastrium may be embarrassed by the pericardial adhesions. *Friedreich's sign*, diastolic collapse of the cervical veins, is sometimes seen. PALPATION.—The signs obtained by inspection are confirmed and there is often to be felt a distinct diastolic shock. The movements of the apex, under the influence of gravity, upon change of posture are less marked than under normal conditions. Pulsus paradoxus may be present. PERCUSSION shows an increase in the transverse diameter of the heart, and since there are usually also pleural adhesions the area of superficial cardiac dulness may not be influenced by the respiratory movements. AUSCULTATION.—The signs are not distinctive. There is very often the murmur of an associated endocarditis, especially in the rheumatic cases. With dilatation there is usually the systolic mitral murmur of relative insufficiency. Other murmurs have been described, especially a presystolic murmur, but these are inconstant and accidental.

Diagnosis.—The direct diagnosis of pericardial adhesion rests upon the history of pericarditis and the presence of the foregoing signs upon physical examination. In some cases a positive diagnosis cannot be made; in others it can be made with certainty even in the absence of a history of pericarditis. The cases that present the greatest difficulty are those in which there are adhesions between the pericardium and epicardium without adhesions to the adjacent structures; the cases in which the diagnosis may often be made with confidence are those in which there are extensive adhesions, not only between the visceral and parietal pericardium, but also between the fibrous pericardium and the surrounding parts—chronic indurative mediastinitis. Too great importance may be given to systolic retraction of the intercostal space in the region of the apex. This, in the absence of pericardial adhesions, may be due to atmospheric pressure when the energetically contracting ventricles are not followed by the border of the lung with sufficient promptness. It may also occur in hypertrophy and dilatation of the right ventricle when the left ventricle remains small. The impulse is that of the right ventricle, and to the left of it there may in some cases be seen distinct systolic retraction. As a rule, to which there are, however, exceptions, the retraction in adherent pericardium is more energetic than that induced by atmospheric pressure. When systolic retraction occurs under observation after an attack of pericarditis the diagnosis of adherent pericardium may be made. In a doubtful case the shadow cast by the Röntgen rays may be of service. It may show irregular contour of the heart, a feeble, restricted cardiac pulsation, and diminished play of the diaphragm, especially in its central parts.

Prognosis.—The outlook in simple obliteration of the pericardium depends upon the influence of the primary pericarditis and the subsequent adhesions upon the myocardium. So long as the heart retains its function the prognosis is favorable. Upon the supervention of the signs of cardiac insufficiency it becomes unfavorable.

iii. Hydropericardium; Hydrops Pericardii.

Dropsy of the pericardium occurs in connection with general dropsy in the course of renal, less frequently of heart disease, and in association with effusions into the other great serous sacs, the pleuræ and peritoneum. In rare cases of scarlet fever this condition has been observed in the absence of dropsy in other parts. Normally the pericardial sac contains a small amount of clear yellow serum, 5–10 c.c. In hydropericardium the quantity rarely exceeds 150–200 c.c. This transudate is clear, yellowish, and may contain a few red blood-corpuscles. The pericardium is smooth and glistening. Chylous effusion is a very rare condition. Hydropericardium presents the physical signs of a moderate pericardial effusion, from which it cannot be differentiated except by the history and the associated clinical phenomena. It does not directly tend to cause death, but constitutes an additional danger in the serious affections in which it arises as a complication.

iv. Hæmopericardium.

Hemorrhage into the pericardial sac is to be differentiated from hemorrhagic pericardial effusion. It results from wounds or rupture of the heart or of an aneurism of the aorta, pulmonary artery, or coronary arteries, and has been observed as a consequence of ulceration in malignant endocarditis. As a rule, death occurs immediately with the symptoms of internal hemorrhage. In extremely rare instances the bleeding is more slow, especially in cases of rupture of the heart, and time permits an examination of the cases with a view to their diagnosis and treatment. The signs are then of a more or less rapidly accumulating pericardial effusion; the symptoms those of more or less abundant internal hemorrhage. The prognosis is in the highest degree unfavorable. Traumatic cases may recover after immediate operation—suturing the incised wall and draining the pericardium.

v. Pneumopericardium.

Air or gas in the pericardial sac is an exceedingly rare condition. t may result from external wounds, perforation of the œsophagus or stomch, subphrenic pyopneumothorax, a tuberculous cavity involving the periardium, or spontaneously without solution of continuity in the sac from he presence of the Bacillus aërogenes capsulatus. The area of cardiac dulless is replaced by tympany. The impulse in the recumbent posture disppears, though it may be felt when the patient sits up. The movements the heart are accompanied by coarse, churning, gurgling noises, and the eart sounds have a loud, metallic ring, which may be heard at some stance from the chest. Fluid is usually also present.

vi. Calcification of the Pericardium.

Deposition of lime salts sometimes takes place in tuberculous perirditis and in pyopericardium with resorption of the fluid. The condition y be partial or complete, so that the heart is encased in a sort of bony ell. Strangely enough it is usually latent, though a diagnosis of adherent ricardium has been made. The lesion is exceedingly rare.

IV. DISEASES OF THE ENDOCARDIUM.

i. Endocarditis.

Definition.—Inflammation of the lining membrane of the heart.

In by far the greater number of the cases the inflammatory process is ricted to the valves—*valvular endocarditis;* exceptionally it extends he lining membrane of the wall of the heart—*mural endocarditis.*

Two forms of endocarditis are recognized, acute and chronic.

(a) Acute Endocarditis.

Acute endocarditis is of every grade of intensity. Its milder forms run a favorable course, and the inflammatory lesions, though they impair the function of the valves, are not destructive. Its severe forms are attended with grave symptoms and usually end in death, and the lesions comprise ulceration and necrosis of the affected valves and adjacent parts. It is convenient to describe separately *acute simple or benign endocarditis*, and *acute ulcerative, infective, or malignant endocarditis*. Between these two forms there is no abrupt anatomical or clinical dividing line.

The lesions in simple endocarditis consist of minute wart-like vegetations, hence the descriptive terms *vegetative* or *verrucose endocarditis*. The left side of the heart is involved more commonly than the right, and the mitral than the aortic leaflets. These vegetations are arranged in lines upon the auricular surface of the auriculoventricular leaflets and the ventricular surface of the sigmoid cusps a little distance back of the free edges of the valves. The clinical course of the disease is determined by the subsequent changes in the valvular lesions, which may result in organization with trifling permanent alteration; in progressive sclerotic changes and deformity—*chronic valvular disease;* in the detachment of loose vegetations and embolism; or finally in an overgrowth of the vegetations and ulcerative destruction of the valve leaflets—*malignant endocarditis*. In the last, not only the leaflets but also adjacent parts may be destroyed, with perforation of a valve, the septum, or the wall of the heart, and, owing to the loosely organized character of the exuberant vegetations, multiple embolism is common.

The valve systems affected are in the order of frequency as follows: mitral alone, aortic alone, aortic and mitral together, tricuspid, and pulmonary. The walls of the heart are involved, as a rule, only in connection with the valves. Endocarditis in fetal life usually involves the right side of the heart.

Etiology. — PREDISPOSING INFLUENCES. — Acute endocarditis, both simple and malignant, has been met with under circumstances in which no antecedent or primary disease or lesion could be demonstrated. In the majority of instances it is a secondary affection. *Simple Endocarditis.*— Rheumatic fever is by far the most common primary affection. Chorea, tonsillitis, scarlet fever, and croupous pneumonia are very frequent. It is rare in enteric fever, measles, diphtheria, variola, and varicella. In gout, diabetes, chronic nephritis, and cancer simple endocarditis is occasionally observed. Acute endocarditis is common in old cases of valvular disease—*recurrent endocarditis*. *Malignant Endocarditis.*—Here also rheumatic fever, pneumonia, and other acute infections play an important part as the primary disease. But it is especially in septic processes that malignant endocarditis occurs. Recurrent endocarditis is frequently malignant in type. Malignant endocarditis constitutes a grave danger in gonorrhœal infection, especially in the male. The malignant form is exceedingly rare in enteric fever, diphtheria, tuberculosis, dysentery, and scarlet fever.

Heredity plays an important rôle in the predisposition to endocarditis. There are many families in which the liability is plainly manifest in successive generations. Rheumatic endocarditis is especially common in childhood and early adult life. It may, however, occur at any age. After forty the liability to a first attack of rheumatic fever is slight. Chorea is more common in girls than in boys, and it is in accordance with this fact that the incidence of simple endocarditis is somewhat greater in females. The especial liability to sepsis which attends the child-bearing function constitutes an important predisposing influence to the graver forms of endocarditis.

Exciting Cause.—The pyogenic bacteria which are present in the lesions of the primary disease are found in the valvular vegetations and in the infected emboli common in malignant endocarditis. One or more varieties may be identified in the same case. The more common are streptococci, staphylococci, pneumococci, and gonococci. Much less frequently the bacillus of enteric fever, diphtheria, tuberculosis, and the Bacillus coli communis have been found. In the simple endocarditis of chronic diseases and cachectic states micro-organisms are frequently absent.

SIMPLE ENDOCARDITIS.

Symptoms.—This form very often runs a latent course without modification of the symptoms of the primary affection. In other cases increased pulse-frequency, slight irregularity in the action of the heart, a sense of precordial oppression, and attacks of dyspnœa occur. There may or may not be a rise of temperature in rheumatic cases without fresh joint affection. In young children rheumatic endocarditis may occur with trifling manifestations of illness and without arthritis, the true nature of the attack being revealed by the physical signs, the subsequent valvular disease, and recurrent attacks of well-characterized articular rheumatism. Again, to these symptoms there may be added the manifestations of acute cardiac insufficiency and grave constitutional disturbances—irregular, rapid, and feeble pulse, faintness, oppression, orthopnœa, high fever not conforming to type, profuse perspirations, and extreme pallor. Such cases lie on the border-line between simple and malignant endocarditis.

Physical Signs.—A murmur may develop at one of the valvular areas. Commonly the first sound is impure at the beginning or slightly rough. This change increases to a murmur which gradually becomes distinct. The second sound may be reduplicated, its pulmonary element accentuated. There may be slight increase in the transverse diameter of the heart and displacement of the apex to the left, signs of implication of the myocardium.

Diagnosis.—Simple endocarditis in many of the cases is discovered only by systematic routine examination. Very often it is not recognized at all. Recent endocarditis is sometimes found in cases of nephritis or carcinoma in which no murmur has been heard. When heard the murmur may be due to relative or muscular insufficiency, or to valvular disease resulting from acute endocarditis in the past. If it has developed under observation the latter possibility may be excluded but not the former. If it becomes more distinct and persists beyond the convalescence from the primary disease, a diagnosis of acute endocarditis is justified.

MALIGNANT ENDOCARDITIS.

Symptoms.—There are two groups of symptoms: those due to the primary disease or the sepsis to which it has given rise, and those due to the endocarditis. Either of these groups may dominate the clinical picture. To the first belong irregular fever, copious sweating, profound anæmia, delirium, and loss of strength; to the second group a curious air hunger, paroxysmal dyspnœa, orthopnœa, palpitation, frequent and irregular action of the heart, and the phenomena caused by emboli in various tissues and organs. A very common point of entrance for the infection is in lesions of the female reproductive organs. Injuries of the integument, boils and abscesses, suppuration of the middle ear, inflamed hemorrhoids, gonorrhœa in the male, croupous pneumonia, and suppurative disease of the liver or of bone are frequent causes. Old valvular disease of the heart is very common. The anamnesis is of great value in the diagnosis. In a doubtful case this ground must be carefully gone over. Septic, typhoid, cerebral, and cardiac forms are described, but the picture is a very diverse one and the distinctions are by no means clear. THE SEPTIC FORM.—There is usually a history of puerperal infection, a neglected wound, acute necrosis, or gonorrhœa. Severe rigors, irregular pyrexia, colliquative sweating, and vomiting are common. Heart symptoms are sometimes subordinate and the signs overlooked. Embolism is common. THE TYPHOID FORM.—The temperature is high and subcontinuous or remittent in type. There are great depression, diarrhœa, sometimes tympany, drenching sweats, delirium, somnolence, and a tendency to coma. Heart symptoms are often obscure. Murmurs may be absent. THE CEREBRAL FORM.—The onset is abrupt with the signs of a basilar or cerebrospinal meningitis. Sudden violent delirium is followed by coma. THE CARDIAC OR RECURRENT FORM.—This variety occurs in individuals who are the subjects of chronic valvular disease. The symptoms are very variable. The attack may run a rapidly fatal course with septic or so-called typhoid phenomena and high fever, or recovery take place after several weeks. Repeated attacks with the clinical manifestations of an acute endocarditis may occur.

Embolism may cause the most diverse manifestations, among which are delirium, coma, hemiplegia, monoplegia, and central derangements of vision and hearing in consequence of implication of arterial branches in the brain or meninges; pain in the splenic area from infarction and perisplenitis; pain in the lumbar region and bloody urine from infarction of one or both the kidneys; and abscesses in the subcutaneous tissues, which are often multiple. The last are common in the legs and feet, less so in the arms, occasional in the buttocks or shoulders, and infrequent in the face or neck. To this cause must also be ascribed the retinal hemorrhage which sometimes occurs, and the rare complication of suppurative panophthalmitis. Erythematous and petechial rashes are common. Jaundice occasionally occurs. As in other forms of sepsis, diarrhœa is often troublesome. Leucocytosis is usually present.

Physical Signs.—There are no signs of importance upon inspection. Palpation yields valuable information as to the character and extent of the impulse, which is frequently somewhat displaced to the right and may be sometimes felt in two interspaces. Thrills in the mitral and aortic areas may be felt. Percussion shows the heart to be moderately enlarged in its transverse diameter, especially to the right of the sternal border—dilatation of the right ventricle. Upon auscultation the signs are by no means constant. Errors in diagnosis may be avoided by bearing in mind the fact that in a considerable proportion of the cases no murmur can be detected upon careful search. Usually, however, there are well-marked, often harsh, murmurs in the mitral and aortic areas, mostly systolic in time, but frequently also presystolic or diastolic as the case may be, and often changing their quality, rhythm, and intensity from time to time. This variability in the murmurs and in thrills when present corresponds to changes in the dimensions and other physical characters of the lesions, and constitutes a diagnostic sign of the highest importance.

Diagnosis of Malignant Endocarditis.—DIRECT.—In the absence of the physical signs of endocarditis and of embolism the recognition of the disease may be impossible. Sepsis, associated with murmurs which vary in character and intensity, or the signs of embolism form the basis for a positive diagnosis.

DIFFERENTIAL.—The following conditions are to be considered: *Acute Simple Endocarditis.*—The general symptoms in the malignant form are much more intense. Recurrent chills, irregular pyrexia, and profuse sweating occur. Embolic processes are far more common. There are borderline cases which may be referred to either category, but in these the absence of a focus of infection is in favor of a severe form of the benign type of the disease. In the malignant cases leucocytosis, petechial eruptions, and the urinary findings of acute nephritis are of diagnostic importance. Blood cultures may yield conclusive results. *Enteric Fever.*—Many of the cases are at first regarded as irregular forms of enteric fever. The gradual rise of temperature in the latter disease, the slowness of the pulse in proportion to the pyrexia, the greater enlargement of the spleen, the rose rash, and a positive Widal reaction constitute a symptom-complex not seen in any other affection. It is true that a rose spot or two may sometimes be found in a septic case. Grave cases of enteric fever with secondary infection may become distinctly septic and develop malignant endocarditis. *Typhus Fever.*—This now infrequent disease usually occurs in local outbreaks, and is characterized by early intense headache, stupor, a peculiar petechial rash appearing about the fourth day and all over the body except the face, and an average course of about fourteen days. *Hemorrhagic Smallpox.*—This rare malignant variety of variola occurs only in the unvaccinated and has little in common with ulcerative endocarditis except its profoundly infectious nature and rapidly fatal issue. *Malarial Fever.*—In some of the cases of malignant endocarditis the ague-like paroxysms of chill, fever, and sweating recur with a periodicity suggestive of malarial infection. The absence of the blood parasite and the total failure of quinine to influence the progress of the disease are conclusive.

Prognosis.—The immediate outlook in the simple form of acute endocarditis is favorable. In the majority of instances, however, it proves to be the point of departure for chronic valvular disease. The remote consequences are therefore often grave. An attack in early life may prove the

As to age, the greater number of the cases originating in rheumatic endocarditis are first recognized between the tenth and thirtieth years. The chronic endocarditis of early life is mostly due to acute endocarditis; that of advancing years to sclerosis. The two sexes are liable nearly to the same degree. Other predisposing influences are unimportant.

V. CHRONIC VALVULAR DISEASE.

i. Aortic Insufficiency.

Aortic Incompetence; Aortic Regurgitation; Corrigan's Disease.

The valves fail to close the aortic orifice and a portion of the blood that has passed into the aorta with the systole returns to the ventricle during diastole.

The loss of function is, in a great majority of the cases, the result of deformity of the valves; in others it is due to dilatation of the aortic ring—*relative aortic incompetency.*

The deformity may be, (a) congenital, and arise from the fusion of two semilunar leaflets at their lateral borders, or from a narrow slit parallel with and close to the free edge. Such valves frequently show sclerotic changes. (b) The result of acute endocarditis in which the insufficiency is caused by the vegetations, or by ulceration and necrosis, or by adhesions with the later changes which attend sclerosis. (c) The manifestation of progressive sclerotic processes, thickening, rigidity, incurving at the borders, and shortening of the valves. (d) Rupture of a valve segment. an accident due to excessive muscular strain, probably never occurring in previously sound valves and very infrequent in disease, if the ulcerated and necrotic valves of malignant endocarditis be excepted.

Dilatation of the outlet may occur in arteriosclerosis involving the aorta immediately above the outlet, in aneurism of the ascending portion of the aortic arch, and in advanced age as a senile change. In aortic insufficiency due to acute endocarditis there is frequently also some degree of stenosis; in the form associated with arteriosclerosis narrowing is comparatively rare.

Etiology.—Aortic insufficiency may occur at any age. It is, however, chiefly met with in middle life and is far more common in males than in females. Rheumatic fever and other acute infections associated with acute endocarditis, conditions which favor arteriosclerosis, as occupations involving continuous and prolonged excessive muscular effort, injudicious devotion to athletics, poisons such as lead and alcohol, and gout and syphilis are important etiological factors.

Direct Effects upon the Heart and Vessels.—The reflux of blood causes overdistention of the left ventricle and diminution of the normal amount in the aorta and its branches. The failure of the valves to close deprives the blood in the arterial tree of its normal base of support, which is transferred in a degree corresponding to the valvular defect to the ventricular wall. The cavity of the ventricle is overdistended. Dilatation occurs and is followed by hypertrophy. In the sclerotic forms the compensation maintained and the lesion is said to be compensated. The tendency on the part of the valvular lesion is to progress. That on the part of the compensating hypertrophy is to advance at an equal rate. Thus compensation advances hand in hand with the lesion, and symptoms are absent. This process goes on, however, at the cost of corresponding impairment of the reserve power of the heart. There may be no symptoms while the heart does only its ordinary work, but the capacity for extraordinary work is progressively impaired. A sudden violent effort, hill climbing, worry, the stress of life, an acute illness reveal beginning cardiac inadequacy. It is fortunate that in the physical signs of valvular disease we have, while compensation is still maintained, the means of recognizing the condition and :an institute measures to avert disaster. There are cases, however, in which compensation does not occur. The lesion is too great or has leveloped too rapidly, or the myocardium is unsound, and dilatation akes place at once.

After a time the compensation becomes impaired. This change may e due to further advance in the valvular lesion, with which the heart uscle is unable to keep pace, or to the insufficiency to which the hyper-ophied muscular tissue is peculiarly prone. The manifestations are not ifferent from those of chronic myocarditis due to other causes. They ary progressively in degree. Hence the terms impaired compensation, oken or lost compensation, de-compensation.

It is in valvular disease that a functional diagnosis is of the highest portance. Not so much what is the lesion or the valve system involved, how it affects the function of the heart, is the question in the individual se. The condition of the heart muscle is far and away more important an the valvular lesion. Is the compensation maintained or impaired? is is the main point. If impaired, to what extent? Upon the reply these questions the management of the case and the future of the tient depend.

When the valvular lesion is compensated the arterial pressure is normal. der ordinary circumstances there is no dyspnœa upon moderate exertion. nosis is not present. The liver is not enlarged, and the normal amount rine is voided. We think too much of the condition of the valves; too le of that of the myocardium. The former is beyond the reach of pro-laxis and cure. Intelligent attention to the latter means, in many cases, relief of distressing symptoms and the postponement of disaster. When pensation fails the heart is enlarged toward the right, there is dys-a upon slight exertion or even at rest, orthopnœa, faint cyanosis, rgement of the area of liver dulness, a feeble impulse, and a small, d, often irregular pulse,—all manifestations of cardiac inadequacy.

About 75 per cent. of the cases are due to acute endocarditis, about er cent. to arteriosclerosis, and the remainder to primary valvular osis and other causes. Of the cases resulting from acute endocarditis ly 60 per cent. are due to rheumatic fever. The distribution of the ns in valvular disease following rheumatism is, according to Romberg, llows: mitral about 59 per cent.; mitral and aortic 29 per cent.; aortic 9 per cent.; and mitral and triscupid, and with these the aortic and onary, 3 per cent.

diastolic thrill may sometimes be felt. Systolic depression, when present, is not so often the sign of pericardial adhesion as of atmospheric pressure. Upon ophthalmoscopic examination the retinal arteries are seen to pulsate. In aortic regurgitation of high grade there is sometimes seen distinct backward nodding of the head corresponding to the systole. Upon percussion, since the hypertrophied heart pushes the lung before it, the area of superficial dulness and that of deep dulness are alike greatly increased, especially downward and to the left. In extreme enlargement of the left ventricle the right heart is displaced to the right.

Upon auscultation there is to be heard at the base of the heart and downward a diastolic murmur, caused by the reflux of blood through the insufficiently guarded aortic orifice into the ventricle. This murmur is often faint at the aortic cartilage, but usually distinct or loud at the sternal end of the third left intercostal space or third cartilage, over the seat of the valve, and propagated along the left border of the sternum to the ensiform cartilage. The diastolic murmur of aortic insufficiency is, in many cases, more distinct in the pulmonary than in the aortic *punctum maximum*. It usually is loud at the beginning and rapidly becomes fainter and lasts throughout the period of diastole. The murmur of aortic insufficiency may, in some cases, be more distinct in the recumbent than in the erect posture. The second aortic sound is either wholly inaudible, being replaced by the murmur, or faintly heard over the aortic cartilage, or finally, when absent at that point, it may be heard over the carotid. The first sound may be normal at the base. A systolic murmur is not always the sign of combined stenosis. It is commonly short, and when coarse and accompanied by a thrill it may be the sign of rigid lesions projecting into the aortic space without actual narrowing, or of abrupt aortic dilatation. While compensation remains good, the first sound at the apex is normal or simply intensified and prolonged. When it fails, the systolic murmur of relative mitral insufficiency is heard.

A

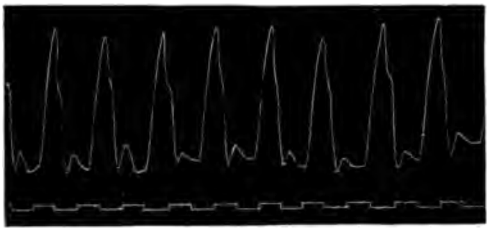

Fig. 347*A*.—Aortic regurgitation; carotid tracing.

B

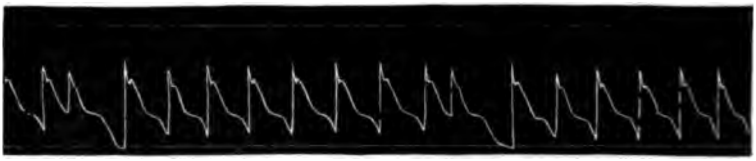

Fig. 347*B*.—Aortic regurgitation; radial tracing.

Flint's Murmur.—A coarse, rumbling murmur, presystolic in time, heard in a limited area just above the apex, and accompanied by a thrill. This murmur has the same qualities and time relation to the cardiac revolution as the murmur of mitral stenosis, but it is not associated with the sharp first sound, the abrupt impulse, and the pulsation in the second and third interspaces which characterize well-marked cases of mitral stenosis. It is not heard continuously, but comes and goes under conditions not well understood, and is met with in a large proportion of the cases of uncomplicated aortic insufficiency.

Over the larger arteries, and especially over the femoral, there is sometimes heard a double murmur—*Duroziez's murmur.* The arteries between the pulse-beats are abnormally empty and soft. The systolic blood-pressure is high and the diastolic pressure abnormally low. The sphygmogram is characterized by abrupt high ascent, sharp summit, and faintly marked dicrotic notch.

Diagnosis.—The DIRECT DIAGNOSIS of aortic insufficiency rests upon the presence of a diastolic murmur, cardiac hypertrophy, and the pulse of Corrigan—*pulsus celer.* It finds support in the occurrence of Flint's murmur, or an associated aortic systolic or mitral systolic murmur,—relative insufficiency,—and in the tendency to massive hypertrophy. There are cases in which the diastolic murmur cannot be heard, or a systolic murmur only is present, yet the pulse and cardiac hypertrophy point to insufficiency of the aortic valve. In high fever with great loss of arterial tone, intense anæmia, some cases of hysteria and neurasthenia, and the more acute forms of exophthalmic goitre, the conditions of the peripheral circulation are very suggestive of aortic insufficiency, but in all these the history of the case and the concomitant symptoms and physical signs are of help in the differential diagnosis.

Prognosis.—The compensation may be maintained for years without symptoms referable to the heart, and the patient lead a fairly active life. The outlook is better when the valvular defect follows acute endocarditis and develops early in life. Associated mitral lesions are unfavorable. Relative mitral insufficiency, by which the arterial conditions are modified, tends to transfer the stress of compensation from the left ventricle to the auricle and thence to the right heart. Sudden death is a danger. It often occurs without marked previous symptoms of heart disease. When compensation fails more gradually, the characteristic symptoms of progressive cardiac inadequacy arise.

ii. Aortic Stenosis.

Aortic Obstruction.

The aortic outlet is narrowed or constricted. A portion of the blood which should pass into the aorta with the ventricular systole is held back with every revolution of the heart. That function of the valve which consists in the retreat of its segments into contact with the wall of the aorta before the blood stream is impaired. This affection is comparatively rare.

The lesion may be, (a) congenital, in which case the cusps may be united to form a thin diaphragm-like membrane with a small slit-like opening, or there may be a subvalvular stenosis the result of prenatal endocarditis; (b) the result of an acute endocarditis with adhesions, stiffening, and vegetations which have undergone fibroid and calcareous changes; or (c) the outcome of an arteriosclerotic process. Very often the diastolic thrill may sometimes be felt. Systolic depression, when present, is not so often the sign of pericardial adhesion as of atmospheric pressure. Upon ophthalmoscopic examination the retinal arteries are seen to pulsate. In aortic regurgitation of high grade there is sometimes seen distinct backward nodding of the head corresponding to the systole. Upon percussion, since the hypertrophied heart pushes the lung before it, the area of superficial dulness and that of deep dulness are alike greatly increased, especially downward and to the left. In extreme enlargement of the left ventricle the right heart is displaced to the right.

Upon auscultation there is to be heard at the base of the heart and downward a diastolic murmur, caused by the reflux of blood through the insufficiently guarded aortic orifice into the ventricle. This murmur is often faint at the aortic cartilage, but usually distinct or loud at the sternal end of the third left intercostal space or third cartilage, over the seat of the valve, and propagated along the left border of the sternum to the ensiform cartilage. The diastolic murmur of aortic insufficiency is, in many cases, more distinct in the pulmonary than in the aortic *punctum maximum*. It usually is loud at the beginning and rapidly becomes fainter and lasts throughout the period of diastole. The murmur of aortic insufficiency may, in some cases, be more distinct in the recumbent than in the erect posture. The second aortic sound is either wholly inaudible, being replaced by the murmur, or faintly heard over the aortic cartilage, or finally, when absent at that point, it may be heard over the carotid. The first sound may be normal at the base. A systolic murmur is not always the sign of combined stenosis. It is commonly short, and when coarse and accompanied by a thrill it may be the sign of rigid lesions projecting into the aortic space without actual narrowing, or of abrupt aortic dilatation. While compensation remains good, the first sound at the apex is normal or simply intensified and prolonged. When it fails, the systolic murmur of relative mitral insufficiency is heard.

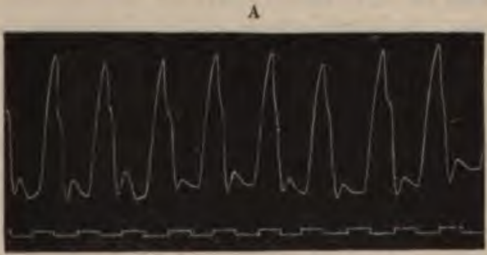

FIG. 347*A*.—Aortic regurgitation; carotid tracing.

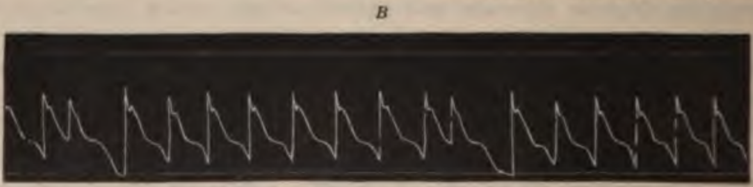

FIG. 347*B*.—Aortic regurgitation; radial tracing.

FLINT'S MURMUR.—A coarse, rumbling murmur, presystolic in time, heard in a limited area just above the apex, and accompanied by a thrill. This murmur has the same qualities and time relation to the cardiac revolution as the murmur of mitral stenosis, but it is not associated with the sharp first sound, the abrupt impulse, and the pulsation in the second and third interspaces which characterize well-marked cases of mitral stenosis. It is not heard continuously, but comes and goes under conditions not well understood, and is met with in a large proportion of the cases of uncomplicated aortic insufficiency.

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The obstruction to the outflow of the blood throws increased work upon the left ventricle, which undergoes hypertrophy. Thus compensation is established. So long as compensation is maintained, the ventricle undergoes little or no dilatation, but it appears small in view of the great thickness of the wall and is, therefore, sometimes spoken of as concentric hypertrophy in contradistinction to the ordinary eccentric hypertrophy seen in aortic or mitral insufficiency. When compensation fails the left auricle undergoes dilatation and hypertrophy of its wall, there is increase of the blood-pressure in the pulmonary circuit and stress upon the right ventricle.

Of aortic stenosis it is especially true that the valves can neither fully open nor completely close. Combined stenosis and insufficiency are therefore common; uncomplicated stenosis is rare.

Relative stenosis is that condition in which with a normal aortic ring and valve cusps there is abrupt dilatation of the aorta immediately beyond.

Etiology.—This is a rare valvular affection. Its incidence is greater in males than females, and it occurs with more frequency in old men with atheromatous arteries than at an earlier period of life.

Symptoms.—With fair compensation there are no special symptoms, and aortic stenosis may reach a high grade without marked evidences of derangement of the general health. Among the symptoms which attract the attention of the patient to his circulation are those indicative of transient cerebral anæmia—vertigo and faintness. In some cases epileptiform seizures have been observed. Palpitation and precordial pain are less common. As compensation fails the symptoms of cardiac inadequacy are progressively developed.

Physical Signs.—Inspection shows, as a rule, a heaving impulse due to the left ventricle hypertrophy, situated at the normal place or slightly to the left. As compensation fails and dilatation of the left and later of the right ventricle takes place, the impulse is displaced beyond the mid-clavicular line. A distinct thrill corresponding in time and duration to the systolic murmur may be detected at the sternal border or at the right side of the root of the neck over the carotid. The pulse is somewhat retarded. In other respects, namely, as to volume and tension, it often preserves its normal characters, though in stenosis of high grade it may be small and slow, with the filling of the arteries well maintained between the beats. The sphygmogram shows a slow rise, a broad summit, and a slow decline. There is in most of the cases in advanced life evidence of marked arteriosclerosis. Upon palpation the position of the apex beat may be obscured by pericardial adhesions or an

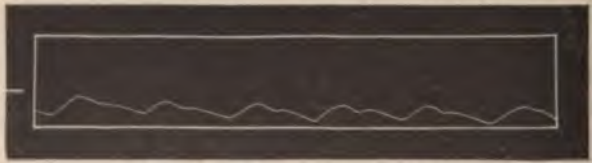

FIG. 348.—Aortic stenosis; radial tracing.

emphysematous lung. Upon percussion while compensation is still maintained, the transverse diameter of the absolute dulness and that of the relative dulness of the heart are little if at all increased. Auscultation discloses in the second right intercostal space at the sternal border a very distinct systolic murmur, usually coarse and harsh. This murmur is among the loudest of the heart murmurs and may sometimes be heard at a distance of some feet from the patient. Not infrequently it has a musical quality during some part of its course. It is distinctly transmitted to the carotids and subclavians, especially upon the right side; less plainly over the heart, but in some cases it may be heard at the apex. Very characteristic is the absence of the second aortic sound. A second sound heard at the aortic cartilage is in most cases transmitted from the pulmonary valve. When compensation fails the murmur may be faint and distant and the thrill disappear.

Diagnosis. — DIRECT. — Aortic stenosis may be recognized by the ssociation of a loud, rough, or musical systolic murmur having its point f maximum intensity at the aortic punctum maximum, and accompanied y a thrill, the signs of hypertrophy of the left ventricle, an inaudible or aint aortic second sound, and a slow, regular pulse of moderate tension.

DIFFERENTIAL.—Errors of diagnosis are common. They arise from ttaching too great importance to a systolic murmur in the aortic area the absence of actual signs of lesions of the valve and hypertrophy of e ventricle. The following conditions in which such a murmur may be ard are to be considered: *Sclerosis of the aorta* directly beyond the valve, of a cusp without narrowing of the orifice may, particularly when asso- ated with the cardiac hypertrophy of nephritis, closely simulate aortic nosis. In favor of the former condition would be an accentuated aortic ond sound, and a small, regular, and rather slow pulse. *Aneurism of Ascending Portion of the Arch.*—A history of syphilis or strain, pressure nptoms,—as pain, dyspnœa, or cough,—inequality of the pulses, displace- nt of the heart as a whole rather than hypertrophy of the left ventricle, umscribed dulness and bulging with or without a thrill, tracheal tugging, l diastolic shock, all or several, when present, justify a diagnosis of urism. An X-ray examination may be of great service in a doubtful . In *anæmic conditions* the basic murmur is often loudest in the aortic . This soft bruit is very different from the loud, harsh murmur of osis; the aortic second sound is heard, there is not usually hypertrophy he left ventricle, the pulse is more frequent, and anæmia may be dem- rated upon examination of the blood. *Relative aortic stenosis* may uspected when, with a systolic aortic murmur, the second aortic sound eserved, left ventricle hypertrophy is lacking, and signs of dilatation e aorta are found upon percussion or by palpation with the finger-tip e episternal notch.

Prognosis.—Cases following acute endocarditis in early life with good ensation may go on without cardiac symptoms for many years. e due to arteriosclerosis beginning in advancing life are of much less able outlook. The changes are essentially progressive, the coronary ies are liable to become involved, and with the development of myo- tis compensation fails. This form of chronic valvular disease is not ded with an especial liability to sudden death.

iii. Mitral Insufficiency.

Mitral Incompetence; Mitral Regurgitation.

A portion of the blood in the left ventricle, which upon systole should pass onward into the aorta, leaks back through the auriculoventricular orifice into the left auricle. Mitral insufficiency is the result of valvular disease, or it may occur without lesions of the valves in consequence of dilatation of the heart—*relative insufficiency*—or of derangement of the mechanism by which the leaflets are brought into effectual coaptation—*muscular insufficiency.*

MITRAL INSUFFICIENCY DUE TO CHRONIC VALVULAR DISEASE.—The structural defects in the valves are in the great majority of the cases the result of acute endocarditis; infrequently the outcome of primary sclerotic processes. They consist of an overgrowth of fibroid tissue with thickening and shortening of the segments, adhesions between their borders, shortening of the chordæ tendineæ, and the deposition of lime salts in the new-formed tissues. In advanced cases the altered valves are often transformed into a thick, rigid calcareous diaphragm perforated by an irregular oval opening. Owing to the nature of the lesions uncomplicated mitral insufficiency is rare, the condition being, as a rule, associated with some degree of stenosis.

The effect upon the heart is as follows:

(a) With each ventricular systole a quantity of blood, varying according to the valvular defect, is returned from the ventricle to the left auricle. This results in an overdistention of the auricle with dilatation and hypertrophy.

(b) The left ventricle undergoes dilatation in consequence of the increased volume of blood received from the overfilled auricle. It, however, empties itself in the normal time and becomes hypertrophied to meet the increased work. Notwithstanding the amount of blood returned to the auricle, the normal quantity enters the general circulation.

(c) In the minor degrees of mitral insufficiency, the dilatation and hypertrophy of the left chambers of the heart suffice for compensation.

(d) In higher grades, the increased blood-pressure due to the overfilling of the left auricle is transferred to the pulmonary veins, and by way of the capillaries to the branches of the pulmonary artery, and thence to the right ventricle, which in turn undergoes hypertrophy.

(e) So long as the compensation thus established is maintained the right ventricle does not undergo dilatation.

(f) The prolonged overfilling of the pulmonary vessels produces brown induration of the lungs.

(g) The compensation may be indefinitely prolonged, but after a time it fails, the left ventricle no longer discharges the normal amount of blood into the aorta, the right ventricle begins to dilate, there is relative insufficiency of the tricuspid valves, and the right auricle becomes dilated. The pressure is transferred to the venous system, and the passive visceral congestions, dropsies, anæmic and other symptoms of cardiac dyscrasia begin to show themselves.

(b) Muscular insufficiency may involve the mitral or the tricuspid valve system. It is due to a derangement of the mechanism by which the closure takes place. There may be evidences of moderate dilatation of the auriculoventricular ring, but these are often absent. Pathological changes in the myocardium are, however, present. There is myocarditis involving the ring muscle at the base of the ventricle and the papillary muscles. The occasional occurrence of transitory systolic mitral murmurs in otherwise healthy men, in the absence of a history of acute cardiac dilatation, justifies the assumption that muscular insufficiency may sometimes be purely functional. It may aid the understanding of this somewhat obscure subject to recall the fact that the auriculoventricular valves are held in close contact during systole, not merely at their margins but throughout their auricular faces, by the pressure of the blood upon their ventricular surfaces.

Diagnosis.—The DIRECT DIAGNOSIS of mitral insufficiency may be attended with difficulty. A systolic murmur having its point of maximum intensity at the apex, propagated to the axilla, and heard at the angle of the scapula; accentuation of the pulmonary second sound; and the signs of hypertrophy of the left ventricle, namely, increase in the transverse diameter to the left, and a strong impulse, are important but not in every case conclusive.

The DIFFERENTIAL DIAGNOSIS between valvular and relative and muscular insufficiency cannot always be made. Here the anamnesis is very useful. In the latter groups of cases a history of acute illness, as diphtheria, enteric fever, or influenza, but no history of recent rheumatism or scarlet fever, recent blood loss or other cause of secondary anæmia, and primary anæmias, as chlorosis or pernicious anæmia, are very suggestive. The signs of arteriosclerosis and of chronic nephritis must be considered when the diagnosis is obscure. These conditions are frequently associated with myocarditis and muscular insufficiency. The almost constant association of some degree of stenosis with insufficiency gives to a presystolic murmur and thrill great value in a doubtful case.

Prognosis.—Mitral insufficiency is the most common and the least deadly of the chronic valvular diseases. Whether or not minor lesions ever terminate in recovery may well be questioned. It is certain, however, that there are many cases in which full compensation is early established and maintained through life. The signs persist, but cardiac symptoms are absent. The outlook is more favorable in the cases which follow acute endocarditis than in those originating in sclerotic processes; when the disease begins in adolescence or early adult life than in childhood or the aged; in those who are able and willing to lead quiet, orderly, and well-disciplined lives than in those whose circumstances demand unremitting toil, or whose habits are irregular and self-indulgent. The extent of the leakage, as indicated by the degree of dilatation and hypertrophy, the completeness of compensation, the presence of secondary morbid conditions or of other valvular lesions or extensive pericardial adhesions has an important bearing upon the prognosis. The liability to recurrent acute endocarditis adds to the gravity of the condition. Among the common causes of death are cardiac insufficiency in the sense of complete loss of contractile power, pulmonary infarct, cerebral embolism, nephritis, and acute intercurrent disease.

somewhat increased in frequency, and arrhythmic. The irregularity persists when compensation is restored, and the pulse of mitral insufficiency once irregular is almost always irregular. The transverse dulness is increased toward the left and to some extent upward. It does not usually extend to the right while compensation is maintained. Exceptionally in large hypertrophy of the left ventricle the right ventricle may be displaced beyond its normal position toward the right. With failing compensation the dilated right ventricle gives increased dulness to the right of the sternal border. Upon auscultation there is heard a systolic murmur having its point of maximum intensity at the apex and being transmitted in all directions, but most distinctly in the direction of the axilla. In some cases this murmur is loudest along the left border of the sternum or in the pulmonary area. It may also be heard along the lower part of the inner border of the left scapula. It is sharp, less frequently soft and blowing, and sometimes musical, especially toward the end. Its intensity varies from time to time. It may be more distinct in the erect than in the recumbent posture, but it is usually audible in both attitudes. It may accompany or wholly replace the first sound of the heart. The second pulmonary sound is accentuated. A more or less distinct presystolic murmur is the sign of a combined stenosis but it is by no means always to be heard.

A

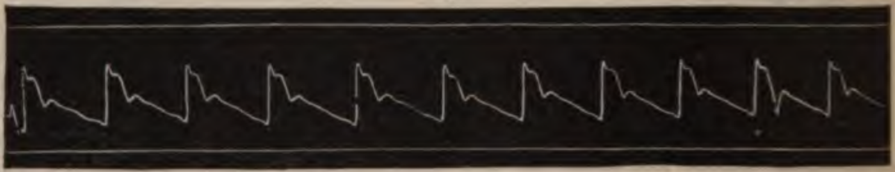

Fig. 349*A*.—Mitral regurgitation; good compensation; radial tracing.

B

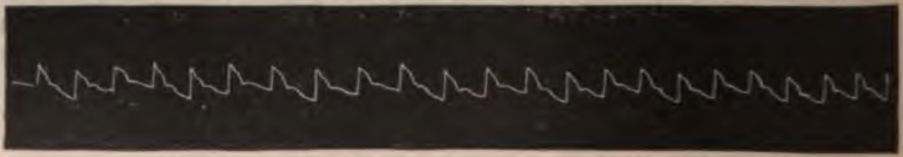

Fig. 349*B*.—Mitral regurgitation; radial tracing.

Relative and Muscular Insufficiency. — (a) Relative insufficiency of the left auriculoventricular valve—*mitral valve system*—results from overdistention of the auriculoventricular ring. It occurs in acute dilatation of the heart,—*heart-strain*; the heart starvation of acute illness and chlorosis or anæmia,—and is then described as primary; and in aortic regurgitation and aortic stenosis upon failure of compensation, when it is known as secondary relative insufficiency.

The etiological factors are those of acute or gradual dilatation. The pathological condition is stretching of the ventricular ring; the derangement of function, incompetence of the valve system. The rational symptoms and physical signs are those of valvular insufficiency.

(b) Muscular insufficiency may involve the mitral or the tricuspid valve system. It is due to a derangement of the mechanism by which the closure takes place. There may be evidences of moderate dilatation of the auriculoventricular ring, but these are often absent. Pathological changes in the myocardium are, however, present. There is myocarditis involving the ring muscle at the base of the ventricle and the papillary muscles. The occasional occurrence of transitory systolic mitral murmurs in otherwise healthy men, in the absence of a history of acute cardiac dilatation, justifies the assumption that muscular insufficiency may sometimes be purely functional. It may aid the understanding of this somewhat obscure subject to recall the fact that the auriculoventricular valves are held in close contact during systole, not merely at their margins but throughout their auricular faces, by the pressure of the blood upon their ventricular surfaces.

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mur is rough and vibratile in character, increasing in intensity toward its close, and terminates abruptly in the tap which constitutes the first sound. It occurs in four modifications. In the greater number of cases it occupies the entire period of the diastole, and is increased in intensity at the time of the auricular contraction which immediately precedes the ventricular systole; it may be heard at the beginning and at the close of the diastole; it may be present in the mid-diastolic period; and finally it is in some cases heard only in the moment immediately preceding the first sound. The intensity of this murmur is extremely variable. It may be the loudest of heart murmurs, or very soft and faint, or, finally, it may be wholly inaudible when the patient is at rest, and only heard after exertion. It may at times have a peculiar rumbling quality. Its loudness depends in part upon the character of the lesions and in part upon the force of the blood stream. It is in accordance with this fact that, as compensation fails, the murmur can no longer be heard, but only the sharp first sound in the mitral area, or the *one, two, three* of the gallop rhythm. If, however, under rest and treatment compensation is for a time re-established the murmur reappears. This change is often met with in actual practice and has been the cause of many disputes as to diagnosis.

The first sound is short, valvular, and very loud. It has in many cases the character of the second sound and is frequently mistaken for it. Under this error the murmur is naturally assumed to be systolic and the condition that of mitral insufficiency. This mistake is to be avoided by determining the time of the auscultatory signs by palpation of the cardiac impulse or carotid pulse at the moment of auscultation. The alteration in the first sound is due to the quick contraction of the left ventricle upon an abnormally small blood content. The second pulmonary sound is strongly accentuated.

Diagnosis.—The DIRECT DIAGNOSIS is in well-marked cases not difficult. It rests upon the association of the presystolic thrill and murmur with the signs of hypertrophy of the right ventricle, the absence of signs of enlargement of the left ventricle, the loud snapping character of the first sound, and the accentuation of the pulmonary second sound.

Prognosis.—In general the outlook is less favorable than in mitral insufficiency. This form of chronic valvular disease is only second to aortic insufficiency in the gravity of the prognosis. Sudden death is somewhat more frequent than in mitral insufficiency, but occurs usually after the compensation has become greatly impaired. Among the more common causes of death are progressive cardiac asthenia, pulmonary infarction or œdema, and acute intercurrent disease. More than any other chronic valvular disease of the heart, mitral stenosis is associated with tuberculosis, and to the latter the termination must in many of the cases be attributed.

v. Pulmonary Insufficiency and Stenosis.

Lesions of the pulmonary valves are extremely rare. Murmurs having their point of maximum intensity in the pulmonary area are common. They are usually systolic. They are sometimes present in health, especially in children, and are best heard during expiration and in the recumbent

Symptoms.—Fair compensation is often maintained for years, during which symptoms are absent. As it gradually fails dyspnœa upon exertion first attracts attention. Pressure paralysis of the left recurrent laryngeal nerve from the enlarged auricle has been observed. Cerebral embolism is by no means rare. The frequency with which hæmoptysis occurs while compensation is yet fair is interesting. The cases are often mistaken for incipient phthisis. The tendency to dropsy is less marked than in mitral insufficiency.

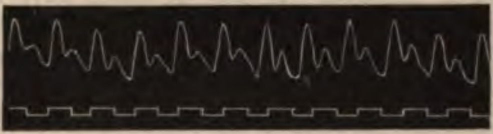

Fig. 350.—Mitral stenosis; carotid tracing.

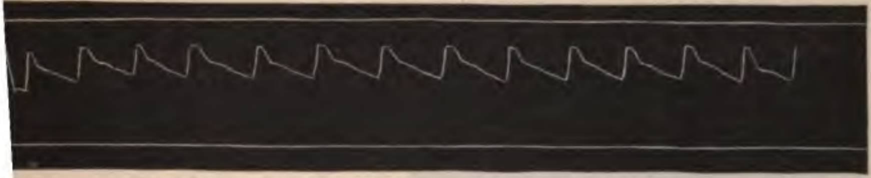

Fig. 351.—Mitral regurgitation and stenosis; radial tracing.

Physical Signs.—Upon inspection there is very often, especially in the young, precordial prominence due to hypertrophy of the right ventricle. The apex beat is commonly displaced but slightly toward the left, and may be indistinct, the visible impulse being at the lower end of the ternum and extending to the eft costal cartilages. The pul- ation of the conus arteriosus nay often be visible at the ster- al end of the third and fourth ft interspaces. As compensa- on fails, the impulse loses its ower, and signs of back pressure in the systemic veins appear, as distention the superficial veins, especially the jugulars, with pulsation due to con- action of the right ventricle and enlargement of the liver. Upon palpation ere is recognized in at least three-fourths of the cases a very distinct rill. This sign is usually coarse in character, diastolic or presystolic in ne, confined to an area above the apex, usually in the fourth and fifth terspaces, and circumscribed. It is more intense and slightly more tended during expiration and runs up to a short, sharp apex beat. This ll is of the highest diagnostic significance. It is the tactile equivalent he characteristic murmur of mitral stenosis. It may, however, fre- tly be felt when no murmur can be detected at this time in the cardiac e. In stenosis of moderate grade, so long as compensation is good the has no special characters. Upon failure of compensation it becomes l, soft, and arrhythmic. Upon percussion the dulness in fair compen- n extends but slightly to the left of the normal line, rarely much beyond nidclavicular line, and little to the right of its normal limits. When, ver, compensation is broken, the transverse diameter of deep cardiac ss is decidedly increased, reaching beyond the midclavicular line on ft and beyond the parasternal line on the right. Auscultation reveals racteristic murmur which corresponds in situation and in time of its ence in the revolution of the heart to the thrill which has been bed above, and which is its auditory equivalent. This mur-marked upon the right side. The venous pulse is synchronous with the carotid pulse and apex beat. It is sometimes transmitted to the subclavian and axillary veins. It may, by way of the vena cava and hepatic veins, reach the liver and cause the phenomenon known as pulsating liver, an expansile pulsation in the organ best appreciated upon bimanual palpation, and to be differentiated from the "jogging" liver, which rises and sinks under the influence of the movements of the heart or aorta but does not expand and contract.

vii. PHYSICAL SIGNS OF UNCOMBINED VALVULAR LESIONS OF THE LEFT HEART, COMPENSATION BEING MAINTAINED.

	Mitral		Aortic	
	Insufficiency	Stenosis	Insufficiency	Stenosis
Impulse	Increased in extent. Displaced to left	Often ill-defined and extended, but not much beyond the midclavicular line. Preceded by thrill	Extended, heaving, forcible and displaced downward and to the left	Variable; sometimes feeble and indistinct; often slow, heaving and forcible. Moderately displaced to the left.
Cardiac dulness	Increased transversely and downward. It may extend beyond the right border of the sternum and beyond the left midclavicular line	Increased to the right of the sternum and along its left border; not usually beyond the midclavicular line	Increased to a greater extent than in any other valvular lesion. Principally downward and to the left	Moderately increased to the left.
Murmurs	Systolic at apex accompanying or replacing the first sound. Transmitted toward left axilla. May be heard at the back; sometimes widely over chest	Coarse, presystolic, terminating in the first sound and limited to the region of the apex	Diastolic, prolonged, accompanying or wholly replacing the aortic second sound, and propagated from the base of the heart downward along the sternum and to the left	Coarse, systolic, of maximum intensity at aortic cartilage and propagated into the great vessels.
Associated signs	Accentuation of pulmonary second sound	Presystolic thrill; impulse of conus arteriosus of right ventricle in fourth and third, sometimes in second left interspaces. A clear, well-defined first sound. Accentuation of pulmonary second sound. Reduplication of second sound	A systolic shock in the larger arteries and sometimes a double murmur—Durosiez's sign. The aortic second sound may be heard over the right carotid when absent in aortic area	Systolic thrill in aortic area, absent or feeble aortic second sound. Occasionally a diastolic murmur.
Pulse	Full, regular, frequent and usually of low tension. Upon failure of compensation there is usually arrhythmia which is commonly persistent	Smaller in volume than normal and frequently arrhythmic	Water-hammer, collapsing—Corrigan's pulse	Small, of fair tension, regular, and usually somewhat slower than normal.

Stenosis.—Tricuspid stenosis is a rare form of valvular disease. It may be congenital, in which case it is almost invariably associated with other cardiac anomalies; or acquired, when it presents deformities similar to those seen in mitral stenosis. It is usually associated with some degree of incompetence of the tricuspidalis and with diseases of the other valve systems, most commonly the mitral. In a large proportion of the cases there has been a history of rheumatic fever; in a few a history of syphilis. This form of chronic valvular disease is far more common in females than in males. It has, in a majority of the cases, been first observed early in adult life. It is so rarely an isolated lesion that its symptoms are commonly obscured by those of the associated affections. They are those of general venous engorgement. Distention of the jugulars, cyanosis of the lips and face, tenderness over the liver, and dropsy are common. The physical signs are also usually subordinate to those of the associated valvular disease. Percussion shows increased dulness to the right of the sternum. When, in a case of mitral stenosis, there is a second presystolic murmur best heard at the base of the ensiform cartilage at the right, associated with a presystolic thrill, and these are of different character from those in the mitral area, tricuspid stenosis may be diagnosticated. The clinical diagnosis is, however, uncertain. Almost all the cases have been discovered upon the post-mortem table, the condition not having been suspected during life.

viii. Combined Valvular Diseases.

There are two cardinal facts in regard to chronic valvular disease. First, that the lesions are very rarely uncomplicated, and second, that the condition of the myocardium is much more important than the state of the valves. While the recognition of these facts renders heart disease complex and difficult as regards diagnosis, it greatly simplifies it in respect of prognosis and the recognition of the indications for treatment. The valvular damage is irreparable, but injury to the myocardium may be postponed or to some extent repaired. The diagnosis, to be of service, must be at once anatomical and functional. It undertakes to determine what valve system is defective; whether the defect gives rise to obstruction or incompetence or both, and in the last of these conditions which defect predominates; whether more than one valve system is involved, and in what manner; and finally the condition of the myocardium—is its contractile function increased to the point of full compensation? Is the increase maintained? Is it failing? From this point of view the combined lesions acquire especial clinical importance. In aortic and mitral insufficiency resulting from acute endocarditis a progressive stenosis may favorably modify the condition of the heart by diminishing the regurgitation. Aortic lesions and, in particular, aortic regurgitation are more frequently simple than mitral lesions. Relative and muscular mitral and tricuspid insufficiency are frequently uncombined.

Especially common is the combination of mitral insufficiency with stenosis of the mitral orifice. The greater the one, the less the other of these defects. This combination is very often associated with other valvular lesions. Next in order of frequency is the association of aortic insufficiency and mitral insufficiency, or combined mitral insufficiency and stenosis. Combined aortic insufficiency and stenosis is less common but not rarely associated with other valvular lesions. In proportion as the stenosis is marked the insufficiency is diminished and a progressive narrowing of the orifice in insufficiency may be conservative.

The frequent association of tricuspid disease with mitral lesions has already been noted, especially that of tricuspid and mitral stenosis. The diagnosis of these and other combined lesions depends upon the recognition of the associated signs and demands the painstaking study of every phenomenon in any particular case.

VI. CONGENITAL LESIONS OF THE HEART.

These lesions are of two kinds, developmental and inflammatory.

DEVELOPMENTAL DEFECTS.—To this group are to be referred the following anomalies:

(a) Defects of the whole heart, as acardia, double heart, ectopia cordis—conditions not, as a rule, of diagnostic interest. (b) Defects of the septa, which may, by the absence of both the auricular and ventricular septum, convert the heart into a double chamber—the bilocular heart; or by the absence of the ventricular septum only, convert it into a three-chambered viscus—the trilocular heart. (c) Patent foramen ovale. (d) Defects of the valves, which may be increased or diminished in number, or adherent. These anatomical defects involve the semilunar cusps of the aortic and pulmonary valves, but not the segments of the auriculoventricular valves. (e) Transposition of the large vessels.

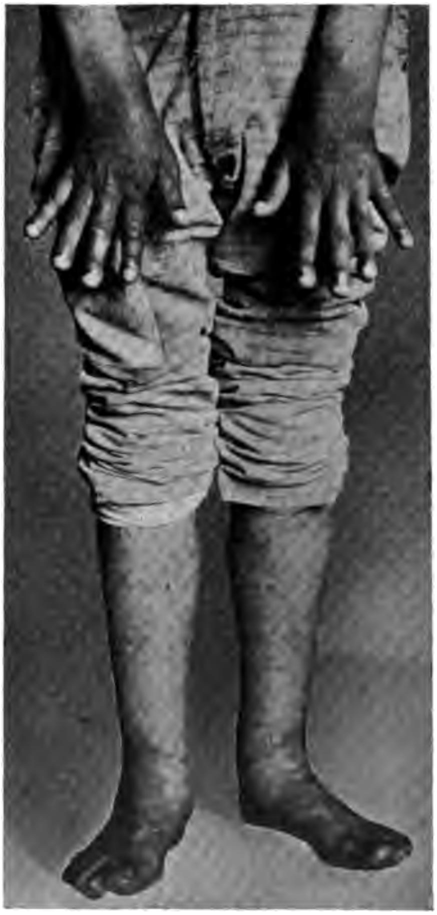

FIG. 353.—Clubbed fingers and toes.—German Hospital.

FETAL ENDOCARDITIS.—The right heart is usually affected. Pulmonary stenosis is a common result. Complete obliteration of the pulmonary orifice is associated with persistence of the ductus arteriosus and patulence of the foramen or other defect in the ventricular wall. Congenital lesions at the aortic orifice are rare.

Developmental anomalies are usually multiple. They not infrequently are the seat of sclerotic processes.

Symptoms.—Cyanosis, general duskiness of the surface, a persistent low external temperature, dyspnœa and cough, increase in the red blood-corpuscles, the drum-stick deformity of the fingers and toes, and finally retarded physical and mental development are usually present. Associated developmental defects in the mouth, genitalia, or elsewhere are common. In infants the presence of endocardial murmurs with or without enlargement of the heart constitutes an important diagnostic criterion.

Diagnosis.—The diagnosis rests upon the association of several or all of the above conditions in a child or young adult in whose case there is a history of having been a "blue baby," or having had "the blue disease." The differential diagnosis of the various congenital defects must be determined by a special study of the physical signs in individual cases. In many of the cases a positive diagnosis of the particular lesions cannot be made.

VII. HEART BLOCK; THE STOKES-ADAMS SYNDROME.

Definition.—A condition characterized by bradycardia, with transient attacks of vertigo and syncope, and momentary epileptiform seizures.

Erlanger's classification based on that of Wenckebach (Osler) is as follows:

Arrhythmia resulting from decreased conductivity in the auriculoventricular function—*heart block*. Characteristics: auricular rhythm perfect, rate normal or accelerated; ventricular rhythm may or may not be perfect; if perfect its rate will be one-half of that of the auricles, or less; if not perfect the irregularities will bear some direct relation to the contractions of the auricles.

(a) Partial heart block: (1) occasional ventricular silence; (2) regularly recurring ventricular silence, either one ventricular beat missed in 7, 6, 5, 4, etc., auricular beats, or a 2 : 1, 3 : 1, 4 : 1 rhythm, or either of these alternating.

(b) Complete heart block: auricular and ventricular rhythms perfect but independent.

(c) Paroxysmal bradycardia (Stokes-Adams disease affecting the ventricular rate alone).

Etiology.—Arteriosclerosis and syphilis play an important part in the causation of heart block. The lesions involve the auriculoventricular bundle of His, a narrow neuromuscular band constituting the only muscular connection between the auricles and ventricles, which serves as a pathway for the stimulus, by which the heart contracts, from the auricles, in which it originates, to the ventricles. Robinson found in the Museum of the Pennsylvania Hospital a heart in which a gumma is situated upon the septum in such a position as to involve this muscular bundle, and upon looking up the clinical records discovered, after the lapse of twenty-five years, that bradycardia had been a prominent symptom in the case. There is a group of cases in which no lesions are found after death and in which the condition appears to be a neurosis. The cases due to syphilis have occurred in early adult life; those due to sclerotic processes at a later period. One of my cases, a woman, was fifty-two; another a man of seventy.

Symptoms.—Bradycardia is usually persistent, sometimes paroxysmal. The ventricular beats may fall to 20, or as low as 5 per minute. The pulse is usually tense. Its frequency is not increased, as under normal conditions, by posture, exercise, excitement, or stimulants. A feeble venous pulsation may be detected in the right jugular synchronous with the auricular contractions. The cerebral symptoms are attributable to the delay between the ventricular contractions. Momentary vertigo and syncope are common, the attacks recurring many times in the course of twenty-four hours. The loss of consciousness is often attended by slight muscular twitchings which especially involve the face and arms.

Diagnosis.—The Stokes-Adams disease is to be distinguished from the physiological bradycardia occasionally seen in pregnancy, from that of old age, and that which occurs in the inanition of hunger. It must also be differentiated from the pathological slow pulse of convalescence from acute disease, of certain gastric affections, especially ulcer and carcinoma, of jaundice, of fatty heart, and of uræmia. Abnormal slowness of the pulse is present also in some cases of anæmia and chlorosis, and in diseases of the nervous system, as brain tumor, affections of the cervical cord, and apoplexy. Certain poisons, as opium, alcohol, lead, and digitalis, cause bradycardia. The differential diagnosis between heart block and any of these conditions rests upon the essential difference between the frequency of the auricular contractions as manifest in the jugular pulsation and those of the ventricles as felt in the radials and at the apex, the occurrence of cerebral symptoms, and the exclusion of other causes. In many cases of cardiac insufficiency a considerable proportion of the ventricular systoles fail to transmit a pulse-wave to the wrist. The apex beats must be counted in all cases of slow pulse.

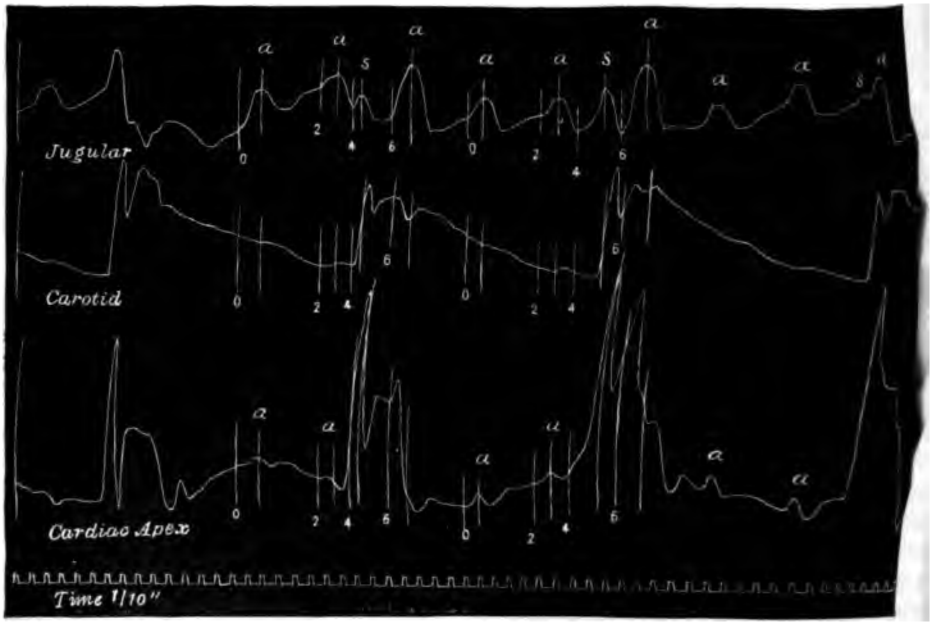

FIG. 354.—Stokes-Adams syndrome. Tracing of the jugular, carotid and apex beat. *a*, auricular contraction; *s*, ventricular contraction.—Jefferson Hospital.

Prognosis.—The outlook is favorable in the neurotic cases. Energetic antiluetic treatment should be instituted when a history or manifestations of syphilis are in evidence. In aged persons with arteriosclerosis, treatment has little effect and the condition continues until death.

VIII. ANGINA PECTORIS.

Stenocardia; Angina Pectoris Vera.

Definition.—True angina pectoris is a symptom-complex occurring in individuals who suffer from sclerotic changes involving the ascending aorta and coronary arteries, and characterized by recurrent paroxysms of agonizing pain in the precordial region, extending to the neck and arms, especially upon the left side, and often accompanied by a sense of impending death.

Etiology.—PREDISPOSING INFLUENCES.—All conditions which tend to the production of arteriosclerosis may be looked upon as influences predisposing to angina pectoris. Age is of great importance. The great majority of the cases first declare themselves after the fiftieth year. Attacks occasionally occur at an earlier age, but they are comparatively infrequent, while the few cases which have been recorded in childhood do not militate against the general rule. Sex exerts a remarkable influence, not in itself, but in the bearing which it has on the mode of life of the individual. Heredity plays an important part. It is by no means rare for angina pectoris to occur in successive generations. To gout, syphilis, and alcohol are justly attributed causal influences of weight. Diabetics are prone to the attacks. It has occurred after influenza.

EXCITING CAUSE.—Any exertion, especially any sudden exertion which calls upon the heart for increased effort, may bring on the attack. The effort is usually a familiar one, as hurry to catch a car, ascending a flight of stairs, or stooping over to lace the shoes. Emotional excitement is a common exciting cause. A fit of anger may precipitate a fatal attack. The paroxysm frequently follows a hearty meal. I often say to patients: Do not hurry, do not worry, and do not eat too much. Exposure to cold is a common cause of the attack. Several of these causes are often associated, as hurry and exposure to cold, or some annoyance after a full meal. The attack seldom comes on when the patient is at rest both physically and mentally. On the other hand there are cases in which the attack occurs immediately upon waking from sleep.

The Paroxysm.—The attack begins suddenly, very often without warning, and in a few moments attains its full intensity. There are three essential phenomena: (1) Pain, commonly described as agonizing, referred to the precordium and mostly to the region beneath the manubrium. This pain radiates to the left side of the chest, to the neck, and to the ulnar distribution in the left arm. In rare cases the pain is first felt in the wrist or arm. The right side is in some instances also affected. (2) The stenocardia of Heberden. The sensation is that of being crushed in a vice, or as if the heart were being "grasped by a mailed hand." It is to this sensation that the fixation of the muscles of the patient is probably due, for at the height of the paroxysm he is usually motionless, almost rigid, while at the beginning and end he is agitated, restless, and anxious. (3) The sense of impending death. The face denotes the anguish which the patient feels. It is pallid, gray, and bathed in sweat. The pulse is often full and slow; its tension greatly increased. It may, however, be nearly normal. The heart sounds are commonly feeble, but clear, and not rarely a soft apex murmur may be heard. Vomiting is not unusual, and death may occur during the act. The duration of the attack does not usually exceed four or five minutes, but recurrences are common, so that the agony may last, with remissions all too brief, for several hours. This is, however, unusual. Nocturnal attacks are usually very severe and longer in duration than those which occur by day. Instant death during the attack is not uncommon. In other cases the patient may fall into a syncope and not revive. The paroxysm subsides as suddenly as it came on, often with belching, the passing of a large quantity of clear urine, exhaustion, and asthmatic symptoms. There are other cases in which the patient, while much distressed and exhausted, is, in the course of an hour or two, able to resume his occupation. The first paroxysm has in many cases proved fatal; in other cases the attacks recur at varying intervals for many years. The first attack may not be followed by a second for a period of months or years, and there are instances of a series of violent attacks followed by no recurrence for a long period.

ANGINA SINE DOLORE.—Gairdner used this term to designate "an element of subjective abnormal sensation present in cardiac diseases, which when it is not localized through the coincidence of pain is a specially indefinable and indescribable sensation." There are undoubtedly cases of heart disease in which paroxysms of profound disturbance of the nervous system referable to the heart occur in the absence of pain, but with an agonizing fear of impending death.

There are many explanations concerning the attack of angina pectoris, among which that of paroxysmal ischæmia or intermittent claudication is the best as a working hypothesis, but none of which as yet fully meets the requirements of the conditions. Mackenzie suggests that the heart muscle induces pain on the principle of the summation of stimuli.

Diagnosis.—The direct diagnosis rests upon the association of paroxysms having the foregoing characteristics with the evidences of general arteriosclerosis and chronic myocarditis. Increased arterial tension, a history of gout, alcoholism, or syphilis, and advanced life are of the nature of corroborative evidence.

FUNCTIONAL ANGINA PECTORIS; PSEUDO-ANGINA PECTORIS.

Two principal groups are described: the neurotic and the toxic.

Neurotic Angina Pectoris.—This form is common in hysterical and neurasthenic women. It may occur at any age. The attacks are not precipitated by muscular effort or cold, but by the multitudinous worries to which such persons subject themselves, and not rarely by injudicious eating. They recur with a periodicity which is remarkable and are more common in the night, a point in which they especially differ from angina associated with coronary or aortic sclerosis. They are usually attended by nervous symptoms—flatulent distention of the stomach and belching—and are often prolonged for several hours, with continuous agitation and restlessness. There are forms in which the paroxysm is characterized by coldness and numbness of the extremities, with agonizing substernal pains, great restlessness, and a tendency to syncope—vasomotor variety.

Toxic Angina.—This form is attributed to excesses in tea, coffee, and tobacco. It includes "tobacco heart." As a rule pain is less marked than sensations of anxiety and precordial oppression, with derangements of the cardiac rhythm. Attacks occur which are characterized by vertigo, pallor, a small, tense pulse, faintness, precordial distress, perspirations, and coldness of the hands and feet. There are three groups of cases: first, the irritable heart of smokers; second, cases characterized by precordial pain, which may be persistent and severe but is not agonizing; and third, cases in which the symptoms are those of an organic angina and there are evidences of arteriosclerosis. In the first two of these groups recovery follows abstinence from the narcotic, but in the third recovery is only partial.

The **diagnosis** of functional angina pectoris depends upon the presence of the neurotic constitution, the absence of the signs of arteriosclerosis or chronic myocarditis, the character of the paroxysms, which, however severe they may be, have neither the short duration nor the essential features of angina vera. The younger age, the sex, and the different nature of the causes by which the paroxysms are excited have diagnostic significance. There are varying grades of intensity, both in organic and in functional angina pectoris, as shown in the recurring paroxysms in the same patient, and it is not the intensity but the character of the attack which is diagnostic.

IX. THE FUNCTIONAL AFFECTIONS OF THE HEART.

The Cardiac Neuroses.

Definition.—The functional affections of the heart include those motor and sensory derangements which occur in the absence of demonstrable anatomical changes in the organ.

The qualifying adjective "functional" is used in its common sense, to denote the absence of anatomical lesions demonstrable during life or after death. It is appropriately employed in this connection to designate, not lesions of the heart itself, but rather disorders of its innervation. Hence these affections are also properly spoken of as cardiac neuroses.

It is important to observe that all the morbid phenomena noted in functional disorders may attend the structural diseases of the heart.

The functional affections of the heart are:

A. Motor:
  1. Derangements of rhythm.
    (a) Arrhythmia.
    (b) Rapid heart—tachycardia.
    (c) Slow heart—bradycardia (brachycardia).
  2. Momentary arrest—syncope.

B. Sensory:
  Subjective sensations referred to the precordia.
    (a) Heart consciousness.
    (b) Precordial distress.
    (c) Precordial pain.

C. Motor and sensory combined:
  Palpitation.

Etiology.—PREDISPOSING INFLUENCES.—These comprise, (a) a weak and delicate organization associated with an impressionable nervous system; (b) anæmic conditions; (c) lithæmia and allied derangements of metabolism and excretion; and (d) morbid conditions directly affecting the nervous system, as organic diseases of the brain and cord, chorea, epilepsy, and the acute and chronic infections.

Certain of these conditions are inherited, others acquired. Thus the descendants of nervous or insane parents, those begotten of elderly persons, those who have in infancy been exposed to privation and neglect, or who have suffered from serious or protracted disease, are especially prone to functional disturbances of the heart. To a less extent is this true of gouty, tuberculous, and syphilitic persons.

Functional derangements of the heart are much less frequent in childhood than in adult life.

The EXCITING CAUSES include: (A) those acting upon the nervous system, (a) directly, as intense mental emotion, fear, anger, grief; or (b) reflexly, as gastro-intestinal irritation, intestinal parasites, or foreign bodies in the intestinal canal; and (B) those acting, by means of mechanical disturbance of the circulation, upon the heart, as violent exercise or exertion.

Symptoms.—In general terms the symptoms of the functional disorders of the heart consist in derangement of the motor functions and abnormal sensations referred to the precordia. These motor and sensory derangements are not always associated. More commonly the movement of the heart is deranged, its action being accelerated, retarded, or irregular, without abnormal sensations; occasionally derangement of rhythm occurs in connection with precordial distress or pain or a sense of oppression; and in comparatively rare instances precordial pain occurs in the absence of motor disturbance.

When the functional disorder is paroxysmal or of a high grade of intensity, it is usually accompanied by increased frequency of respiration, and very often by pallor of the face and slight cyanosis. Especially is pallor associated with syncope.

When the derangement is not paroxysmal, but persistent, the rhythm of the respiration is not usually disturbed.

Physical examination yields a limited number of definite signs. The frequency of the heart's action and the degree and character of the arrhythmia are recognized upon palpation. By this method of examination we also detect, especially on palpation, the change in the character of the impulse, which is increased in force. We observe also by this means and by inspection that the impulse is extended. We determine by the position of the apex beat, and may confirm the observation by percussion, that the heart is not enlarged. Upon auscultation the first sound is found to be sharp and valvular and shortened in duration, while the second sound remains distinct or is accentuated. In very rapidly acting or very irregular hearts transient murmurs, usually mitral systolic, are common.

A. **Motor:** 1. DERANGEMENTS OF RHYTHM.—The cardiac mechanism is now regarded as myogenic rather than neurogenic; that is to say, inherent in the heart muscle rather than due to the influence and control of the vagi and sympathetic nerves under the influence of higher centres. The following classification of the forms of arrhythmia is that of Joseph Erlanger, based upon that of Wenckeback:

I. *Arrhythmia Resulting from Decreased Conductivity in the Auriculoventricular Junction—Heart Block.*
  A. Partial heart block. B. Complete heart block. C. Paroxysmal bradycardia.
II. *Arrhythmia Resulting from Increased Irritability of the Heart.*
  A. Ventricular extra-systoles, characterized by an early systole, which is associated with the phenomena of a retrograde impulse. There may be one or more extra-systoles following a normal systole; when regularly recurring, one or more extra-systoles after 5, 4, 3, 2, or 1 normal systoles, the last giving the bigeminal or trigeminal pulse, or there may be irregularly recurring extra-systoles causing delirium cordis.
  B. Auricular extra-systoles.
III. *Arrhythmia Resulting from the Influence of Extrinsic Nerves upon the Heart-rate.*—(1) Vagus effects. (2) Accelerator effects.
IV. *Arrhythmia Resulting from Disturbed Diastolic Filling of the Heart.*
  A. Disturbed filling resulting from violent respiratory movements; may give the paradoxical pulse.
  B. Disturbed filling from adherent pericardium or mediastinal tumor; may give the paradoxical pulse.
  C. Associated respiratory and cardiac rhythm. Alternating pulse (?).

(a) *Arrhythmia* in time and volume and intermission of the pulse is due to extra-systoles, which occur in various groupings and combinations, giving rise to pulsus bigeminus, trigeminus, and so on, according to the transmission of the extra pulse-beats to the peripheral arteries. Extra-systoles occur under the most varying conditions. Arrhythmia may be manifest as an individual peculiarity, wholly independent of health or disease. There are individuals who have marked cardiac irregularity,—arrhythmia, intermission,—wholly without derangement of the function of the circulation and usually without consciousness of the abnormality. Extra-systoles are common in irritable heart, such as occurs in debilitated and neurasthenic persons; in overtrained athletes; in those who are given to the abuse of narcotics, as tea, coffee, tobacco, and alcohol, or to sexual excesses; and in the subjects of autointoxications of various kinds. They are common in overdistention of the stomach with food, and in various forms of dyspepsia, especially gastric flatulency—probably as manifestations of reflex irritation. They may be the result of abnormally high blood-pressure. Finally, they are common in organic disease of the heart, as myocarditis, dilatation, sclerosis of the coronary arteries, and rupture of compensation in chronic valvular disease of various kinds. It is evident that the extra-systoles of organic disease are far more important and significant than those due to irregular physiological stimuli or abnormal reflex or toxic stimuli, that are controllable or transient.

(b) *Rapid Heart; Tachycardia.*—Frequent revolution of the heart is sometimes an individual peculiarity. The pulse may be 100 per minute, and more frequent after meals and under excitement or active exercise. The rapid pulse caused by fright may continue for an indefinite time. The accelerated pulses symptomatic of fever and of exophthalmic goitre are of toxic causation and cannot be regarded as cardiac neuroses, nor does the frequent pulse sometimes present at the menopause, or that due to reflex influences from disease of the pelvic organs, belong to this category.

Paroxysmal or Essential Tachycardia.—This remarkable affection occurs in the absence of the ordinary nervous or organic conditions which give rise to symptomatic heart hurry. It may begin early in life and continue for years without manifestations of organic disease of the heart. The attacks occur abruptly in the midst of ordinary health, and continue for some minutes, hours, or days, and cease as suddenly as they began. They are characterized by faintness, oppression, moderate palpitation, pallor, dyspnœa, and a pulse-frequency that may exceed 200 per minute. The heart sounds are of fetal type. Occasionally simple intermissions occur. In some cases there is distinct arrhythmia. At the close of the attack the pulse becomes relatively slow and full, sometimes not exceeding in frequency half or even one-third that of the paroxysm. The attacks recur at intervals of days or years, the general condition of the patient being meanwhile normal. They are brought on in many cases by physical effort or emotional stress. In other instances they occur in the absence of recognizable exciting cause. The attack is rarely if ever fatal. In exceptional cases the disease ceases after a series of paroxysms.

(c) *Slow Heart; Bradycardia.*—Slow heart may be an individual peculiarity. The physiological and pathological conditions under which slowness of the pulse may occur have already been considered. It is of cardinal importance to determine the heart frequency by palpation of the apex beat or auscultation of the heart sounds, since there are cases in which half the beats do not transmit a pulse-wave to the wrist.

A form of paroxysmal bradycardia has been described (Hoffman).

2. MOMENTARY ARREST; SYNCOPE.—Faintness is common in nervous and impressionable persons. It may result from sudden shock or intense excitement. Blood loss (even when slight), profuse diarrhœa, extreme fatigue, and severe pain may cause faintness or actual syncope. Emotional children may faint at the sight of blood. A boy of seven fainted at the sight of the denuded spot upon his arm caused in vaccination. A healthy girl of eight, of shy and timid disposition, fainted at the dinner table upon being suddenly addressed by a person whom she did not know.

B. **Sensory.**—Subjective sensations referred to the precordia.

(a) HEART CONSCIOUSNESS.—Neurotic and neurasthenic individuals frequently complain of disagreeable sensations in the region of the heart. They feel the extra-systole and the compensatory pause, and use such phrases as "the heart stumbled," or "the heart turned over," to express the sensation. But in many cases of arrhythmia there are no subjective sensations. It is a mistake to call the attention of patients to arrhythmia or intermissions of which they are unaware, since the consciousness of such irregularities is often a cause of great distress.

(b) DISTRESS.—Precordial distress is common in neurotic individuals, both in functional and organic disease of the heart. It is extreme in the overacting heart of violent exertion and is the essential element in palpitation. It is common in intense emotion. It may be reflex in character and is frequently due to gastro-intestinal derangements. It is often transient.

(c) PRECORDIAL PAIN.—This neurosis is common in nervous individuals. It occurs in valvular disease of the heart, especially in aortic and mitral stenosis and the later periods of aortic insufficiency, and in the forms of myocarditis associated with sclerosis of the coronary arteries. It is the chief element in angina pectoris and an important symptom in aneurism of the aorta.

C. **Motor and Sensory Combined.**—PALPITATION.—The most extreme motor disturbance of the cardiac rhythm may occur in the absence of consciousness of any cardiac irregularity, and distressing precordial sensations may, on the other hand, be experienced while the heart's action is regular and orderly. In palpitation there is irregular or forcible action, perceptible and usually distressing to the individual. This combination of motor and sensory derangements constitutes palpitation. It is of frequent occurrence in neurotic individuals, and is common at puberty, the grand climacteric, and during menstruation. It is frequently encountered in hysteria and in neurasthenia, and constitutes an important element in the symptom-complex known as cardiac neurasthenia. It may occur in normal individuals in consequence of violent emotion. It is a symptom of irritable heart whether due to over-exercise, excesses in venery, or over-indulgence in tea, coffee, tobacco, or alcohol; and finally it is a common factor of cardiac insufficiency from any cause. As a rule, palpitation is more violent and distressing in irritable heart than in valvular or myocardial disease. The symptoms vary from a mere fluttering of the heart to a violent pulsation with great distress and anxiety. In the latter case the heart's action may be slow or rapid; regular or irregular. The sounds may be clear and ringing, the second accentuated, and murmurs wholly absent. In some cases there are basic or apex murmurs, which disappear when the attack is over. The attacks are of variable duration, but do not often exceed an hour or two.

DISEASES OF THE ARTERIES.

I. ARTERIOSCLEROSIS.

Definition.—A disease of the arterial system characterized anatomically by nutritional changes in the media and adventitia with compensatory thickening in the intima, which thickening subsequently involves all the coats; and clinically by functional derangements in the various viscera.

The anatomical changes may be diffuse or localized. In the diffuse variety the arteries are dilated and tortuous, their walls thickened and inelastic, and the intima the seat of irregular thickening, calcareous plates, and atheromatous ulcers; in the localized or circumscribed form yellowish-white, rounded, nodular patches, raised above the surface, are irregularly scattered along the intima. Diffuse and nodular sclerosis are commonly associated.

In senile arteriosclerosis the arteries are dilated and tortuous, the walls thin and rigid, and the subendothelial tissue the seat of circumscribed collections of softened or broken-down material—*atheromatous abscesses*. When these collections rupture into the lumen of the artery they give rise to atheromatous ulcers.

In the various forms the degeneration of the media may be marked in the smaller arteries. The capillaries are thickened and may be obliterated. The connective-tissue overgrowth leads to more or less complete atrophy of the muscular and connective-tissue elements. In some cases characterized by general or local increase in blood-pressure the muscular fibres of the media may be preserved or even hypertrophied.

In consequence of these changes the larger and middle-sized arteries are dilated; the smaller, by reason of the thickening of the intima, are narrowed and very often wholly obliterated—*endarteritis obliterans*.

The ramifications of certain arteries are involved with greater frequency than others. In the series of cases studied by pupils of Thoma and analyzed by Bergmann, sclerotic changes were found in the ulnar artery in 94 per cent., the tibialis antica 93, the subclavian 88, arteries of the brain 87, internal carotid 87, radial 86, splenic 82, popliteal 79, external carotid 78, axillary 71, femoral 69, common carotid 68, ascending aorta 67, abdominal aorta 64, external iliac 58, and brachial 55. The minute arteries of the various organs are involved in the arteriosclerotic process. These lesions are especially common in the heart, brain, kidneys, liver, and pancreas. Sclerosis of the pulmonary artery and its branches is a constant concomitant of lesions such as chronic disease of the mitral valve, emphysema, and fibroid phthisis, which cause persistent increase of the blood-pressure in the pulmonary circulation. The artery may be dilated, with insufficiency of the semilunar valve system, its primary and secondary branches the seat of aneurismal dilatation, and its smaller branches highly sclerotic. The sclerotic process frequently extends to the capillaries and may also affect the veins, which not infrequently are sclerotic in the absence of similar changes in the arteries.

Etiology.—The following factors are of importance in the causation of arteriosclerosis:

1. *Persistent High Blood-pressure and Sudden, Frequent, and Extreme Alternations of Pressure.*—There are many conditions which bring about habitual strain upon the arteries. Among the more important are: (a) Habitual hard work. This accounts for the great frequency of arteriosclerosis among the laboring classes, and the fact that working men more frequently suffer from sclerotic changes in the upper extremities and women in the same walk of life in the lower extremities, while such changes in the arteries of the extremities are infrequent in persons whose occupations are not laborious (Romberg). The muscular effort habitually increases the peripheral resistance and raises the intra-arterial pressure. (b) Nervous influences—the strenuous life in which physical effort, mental stress, and excitement combine to tax alike the brain and the heart. In this connection the frequency with which arteriosclerosis is present in neurasthenia is to be considered. (c) Obesity. The increased effort demanded by the ordinary movements of life and by the larger volume of the circulating

blood tends to arteriosclerosis, and especially to arteriosclerosis of the coronary arteries. (d) Chronic interstitial nephritis is associated with persistent increase of blood-pressure and sclerotic changes in the arterial walls. (e) Frequently repeated and extreme changes in temperature, such as are necessary in certain crafts, tend by the abrupt contraction and dilatation of the superficial vessels to the production of arteriosclerosis. (f) The strain upon the arteries in aortic insufficiency rapidly brings about sclerosis of their walls.

2. *Chronic Intoxications.*—(a) The abuse of tea, coffee, tobacco, and alcohol is credited with a causal influence which is doubtless over-estimated. These narcotics, and especially alcohol, may exert an indirect effect by increasing nervous excitability. (b) Lead, diabetes, and gout play an important rôle. The mode of action has not been explained. Their association with interstitial nephritis is to be considered. (c) Renal disease. There are two groups of cases, primary interstitial nephritis and nephritis associated with general arteriosclerosis. (d) The infectious diseases, especially malaria, rheumatic fever, and enteric fever, appear in some cases to be the starting-point of progressive arteriosclerotic changes. (e) Excesses at table. There can be no doubt of the importance of over-eating as a factor in the causation of arteriosclerosis, and that the results of such excesses belong in the category of the chronic intoxications is equally beyond question.

3. *Syphilis* is a causal factor of great moment. In syphilitic subjects arteriosclerosis develops early and attains a high grade. The distinction between arteriosclerosis and the specific vascular lesions of syphilis is to be made.

4. *Heredity.*—The predisposition to arteriosclerosis varies greatly in different families. Inherited anatomical peculiarities, as congenital narrowness of the arteries and thickness of their walls, are attended with the liability to early sclerosis. These arterial changes appear early among the peasant immigrants from Italy and other countries of Southern Europe.

5. *Age.*—The view is generally entertained that arteriosclerosis is a manifestation of senile involution. There are, however, aged persons in whom little or no evidence of the disease is apparent. It is probable that in many cases the arteriosclerosis of old persons is a late result of the action of other causes operative in earlier life. In fact several of the above etiological factors are usually to be recognized in the anamnesis. This is especially the case in connection with syphilis.

It is in accordance with the above facts that arteriosclerosis is more common after middle life than at an earlier period, and in men than in women.

Symptoms.—Arteriosclerosis begins insidiously and may long remain latent. There are, however, cases in which the vascular changes attain a high grade in the course of a very few years. When symptoms become manifest they may be general or local, according to the vascular distribution chiefly affected.

The general symptoms are in many cases not different from those of old age. The skin becomes harsh, wrinkled, and inelastic; subcutaneous fat is reduced over the chest and extremities and accumulates upon the abdomen; the muscles waste; the viscera undergo atrophic changes; and there is manifest loss of bodily and mental power. Dyspeptic symptoms are often prominent. Characteristic phenomena relate to the circulation. The blood-pressure is increased, the superficial arteries are thickened and hard, there is hypertrophy of the left ventricle and accentuation of the aortic second sound, which has often a clear, ringing quality. There are, however, cases in which the blood-pressure remains low and the hypertrophy of the left ventricle is moderate. An increased flow of urine of low specific gravity and transient traces of albumin, together with occasional hyaline casts, is not uncommon.

The local symptoms depend upon the grade of the vascular lesions and the organ or structure principally involved, as the heart, brain, kidneys, or extremities.

1. Cardiac Symptoms and Signs.—The effect of persistent resistance to the flow of the blood in the peripheral arteries is hypertrophy of the left ventricle, with the symptoms and physical signs of that condition. When the coronary arteries are involved, local or general myocardial degenerations occur with their attendant symptoms, which are those of cardiac insufficiency. Angina pectoris, aneurism of the heart, local softening, rupture, or sudden death may result. When dilatation supervenes the systolic apex murmur of relative mitral insufficiency is heard, and the symptoms of extreme cardiac insufficiency arise, dyspnœa while at rest, somnolence, scanty urine, dropsies and effusions into the serous sacs. Dilatation of the ascending aorta may give rise to tympany or dulness at the sternal end of the second right intercostal space or palpable pulsation in the episternal notch and a soft systolic murmur with an accentuated second aortic sound when the aortic valves are capable of closure, and may be well shown by the Röntgen rays.

2. Cerebral Symptoms.—Among the early symptoms are sensations of fulness and pressure in the head, insomnia, distress, anxiety, and vertiginous attacks. As the vascular lesions progress mental symptoms develop. The attention flags, the recollection of recent events fails, the patient becomes indifferent about his personal appearance and less considerate of others. Self-restraint may be impaired. Actual vertigo, transient loss of consciousness, and temporary derangements of speech are among the symptoms of advanced arteriosclerosis. Repeated attacks of hemiplegia, monoplegia, and aphasia may occur with the signs of organic lesions and terminate in complete recovery in the course of a few hours or a day or two. The Stokes-Adams syndrome is occasionally observed. Tinnitus cerebri and tinnitus aurium are common and often distressing symptoms. The cerebral symptoms of arteriosclerosis include those of the lesions of the brain due to persistent obstruction of the smaller vessels, and accidents, such as thrombosis, embolism, and hemorrhage.

3. Renal Symptoms.—These correspond to those of chronic interstitial nephritis. The association of this form of renal disease and arteriosclerosis is common. Arteriosclerosis predisposes to nephritis; chronic nephritis to general sclerosis. When the patient has not been under observation from the beginning it is impossible to determine which is the primary, which the secondary, affection. In either case the renal symptoms may

dominate the clinical picture; more commonly, however, the cardinal symptoms are those of myocardial disease. The condition constitutes one of the forms of cardiorenal disease. Glycosuria is a frequent indication of sclerosis of the pancreatic arteries.

4. VASOMOTOR SYMPTOMS.—Sensations of cold and numbness in the hands and feet, fulness or lightness in the head, tingling and pulsation in the fingers, dynamic pulsation in the abdominal aorta, and congestion of the feet and legs are very common.

5. THE EXTREMITIES.—The symptoms due to sclerosis of the peripheral vessels are comparatively infrequent. The diminished capillary circulation gives rise to more or less marked pallor, which is not rarely an early symptom. Its association with loss of weight and strength suggests anæmia or even the development of visceral cancer, especially in cases in which the signs of changes in the heart or brain are not prominent.

Intermittent Claudication; Dysbasia Angiosclerotica; Crural Angina. —This constitutes a somewhat common and very striking clinical manifestation of sclerosis in the arteries of the lower extremities. It depends upon the fact that while at rest the blood supply to the muscles, diminished as it is by the narrowing of the lumen of the peripheral vessels, is yet sufficient for their physiological requirements or even for moderate use, but when muscular effort is made the blood supply is inadequate and severe cramp-like contractions ensue. After walking a short distance the patient experiences in one or both legs sensations of numbness, tingling, heat or cold, tension and pain. The skin becomes pale and cyanotic. If the effort is not discontinued more or less severe cramp and muscular disability follow. These symptoms cease upon rest, only to return upon further effort. In extreme cases the spasms recur upon moderate effort and the muscles undergo gradual atrophy.

Diagnosis.—The direct diagnosis of arteriosclerosis may be made when increased blood-pressure, thickened and tortuous superficial arteries, hypertrophy of the left ventricle, and accentuation of the aortic second sound are present. The thickened radial artery can often be rolled under the finger upon the radius like a whip-cord, or it may show irregular, nodular projections along its course like a string of wampum, or present little aneurismal-like circumscribed dilatations. A high degree of sclerosis in the superficial arteries may exist without a corresponding condition in the arteries of the parenchymatous organs, and the converse is true. Another important fact in diagnosis is that advanced arteriosclerosis may involve a vascular territory or organ, as the kidneys or brain, without, in the absence of general sclerotic changes, increasing the blood-pressure, and, therefore, without causing left ventricle hypertrophy or marked accentuation of the aortic second sound. When there is marked deposition of lime salts in the affected arterial walls in the legs and feet, the X-ray examination yields positive results, but it is useless in the deeply seated arteries of the trunk, with the exception of the aorta, the shadow of which is broadened and intensified in places by the presence of thickened atheromatous plates. Etiological considerations are important in the diagnosis.

Prognosis.—The course of arteriosclerosis shows extreme variations. The disease may involve the peripheral circulation and be wholly latent as to symptoms. Cases of this kind go on from year to year without serious impairment of health. It may, on the other hand, rapidly give rise to distinctive changes in organs that are necessary to life. Finally, it not uncommonly sets up a vascular dyscrasia in which the entire body and its organs are involved in progressive nutritional and atrophic changes, which terminate in death in the course of a few years. In selected cases of peripheral sclerosis the removal of the cause and regulation of the mode of life very often favorably influence the progress of the disease and may arrest it. In cases due to syphilis an energetic antiluetic treatment may be followed by an arrest of the sclerotic process.

II. ANEURISM.

Definition.—A circumscribed dilatation of an artery. This anatomical condition is to be distinguished from the diffuse widening of the larger arteries which occurs in arteriosclerosis.

Aneurisms are divided:

(a) According to their form into *fusiform* or *cylindroid*, in which there is uniform dilatation of the vessel, and *sacculated*, in which there is a lateral bulging or protrusion of the wall.

(b) According to the composition of the wall into *true*, in which the wall is composed of one or more of the coats of the artery, and *false*, in which there is a rupture of all the coats and the blood is confined by the surrounding tissues. This distinction is not important, since a false aneurism is in reality a hæmatoma and the differentiation between true and false aneurisms cannot in all cases be made after death, much less during life.

(c) According to other anatomical peculiarities into *cirsoid*, in which an artery and its branches are involved in the dilatation, *dissecting*, in which the blood collects between the coats of the artery—this type occurs in the aorta and occasionally forms a complete double tube—and *arteriovenous*, in which there is a communication between an artery and a vein. There may be a sac between the artery and vein, but more commonly the communication is direct and the vein which yields to the intra-arterial pressure is dilated, tortuous, and pulsating—*aneurismal varix*.

i. Aneurism of the Aorta.

Aneurisms of the aorta are usually fusiform or sacculated. The latter is more common. The combination of these forms is occasionally encountered. Dissecting aneurisms are rare. Still more rare are arteriovenous aneurisms. The essential anatomical condition is dilatation, under the intra-arterial pressure, of the vessel wall, weakened by disease. In fusiform dilatation all three coats of the vessel are dilated. In sacculated aneurism the intima may extend into the sac some distance; the media undergoes atrophic changes and extensive destruction. The wall of the sac is in the greater part of its extent formed by the adventitia, which is thickened, infiltrated, and fused with the surrounding tissues. The communication with the aorta is by an opening of variable size. The interior of the sac is lined by superimposed laminæ of coagulated blood, these which are peripheral being dense and of a whitish color; those which are central being soft and red. The arrangement of these coagula is neither symmetrical nor constant, and important modifications of the pressure symptoms and physical signs arise in consequence of the yielding of the sac to the internal pressure in various directions at different periods in the course of the affection. In rare instances, small aneurisms having narrow communications with the lumen of the aorta are completely filled with coagula and thus undergo spontaneous obliteration with the formation of a small nodular tumor. As the conditions which give rise to sacculated aneurism involve different portions of the wall of the aorta multiple aneurisms are by no means rare. In size sacculated aneurisms vary from a walnut to a large cocoanut; in shape they are globular, but as they increase in size the wall yields more at one point than another in such a manner that irregular protrusions occur. Aneurisms may occur in any part of the aorta, from just above the ring to the iliac bifurcation. The most common site is in the ascending portion of the arch to the right.

Etiology.—Arteriosclerosis, syphilitic aortitis, and trauma are the most important etiological factors. The great majority of aortic aneurisms are due to those causes acting singly or in combination. Far less frequent are cases due to the action of micro-organisms.

(a) *Arteriosclerosis* leads to diffuse weakening of the wall, which yields to the pressure of the blood at a period when compensatory thickening of the intima has not yet occurred, and undergoes dilatation. Sacculated aneurism may occasionally have its origin in arteriosclerosis.

(b) *Syphilis* plays a most important rôle. Sacculated aneurism is in a large proportion of the cases syphilitic. Whether the process has its beginning comparatively early in the changes in the wall due to gumma or at a later period in consequence of the loss of elasticity in scar tissue remains undetermined. The lesions of a patch of syphilitic aortitis constitute the point of diminished resistance.

(c) *Traumatism.*—When the media and adventitia are weakened, a sudden increase of the blood-pressure may lacerate the intima. A violent contusion, a fall, a blow upon the chest may be followed by the gradual development of an aneurism.

(d) *Micro-organisms.* — Multiple aneurisms of the aorta and other arteries have occasionally been observed in connection with malignant endocarditis. Various bacteria have been found in the lesions. Weakening of the wall of the vessel in consequence of an ulcerative process analogous to that affecting the valves doubtless constitutes the early lesion.

(e) *Traction Aneurisms.*—An exceedingly rare form of aneurism is caused in the concavity of the aortic arch at the point of insertion of an insufficiently elongated remnant of the duct of Botallo.

Among other predisposing influences of secondary importance are the following: *Age.*—Aortic aneurism is much more common between the fortieth and sixtieth years than at any other period of life. *Sex.*—Men suffer more frequently than women in the proportion of 4 or 5 to 1, a preponderance due not to inherent anatomical differences but to the far greater exposure to the common causes. *Habits.*—The direct influence of alcohol has probably been over-rated. Its indirect influence in leading to exposure to the danger of contracting syphilis is very great. *Occupations* which involve great muscular effort and those which are attended with the danger of violent blows and contusions of the chest may be regarded as predisposing to thoracic aneurism.

An aneurism of the aorta is a vascular tumor which may be wholly latent or manifest itself by symptoms, usually effects of pressure, and physical signs which differ according to its situation and the relative amount of stratified clot and blood which it contains. It is convenient to consider separately aneurism of the thoracic aorta and aneurism of the abdominal aorta.

(a) ANEURISM OF THE THORACIC AORTA.

Symptoms.—The relative prominence of subjective symptoms and physical signs depends upon the situation and size of the aneurism. The cases may be arranged in four groups.

1. The Aneurism is Latent.—Symptoms are wholly absent, or vague and not suggestive of the actual condition. There are no physical signs. This group includes the cases of moderate fusiform dilatation, small sacculated aneurisms in the ascending aorta, and especially those immediately above the sinuses of Valsalva. The last not rarely rupture into the pericardium. The diagnosis is made after death.

2. Symptoms are Prominent.—Pressure symptoms are present, but the nature of the lesion cannot be determined. To this category are to be referred small sacculated aneurisms of the transverse and descending portions of the arch compressing the trachea or left main bronchus and the recurrent laryngeal nerve, and larger sacs in various situations, containing much laminated clot and yielding the signs of a solid tumor rather than those of an aneurism, and sacs upon the descending aorta just above the diaphragm, especially those eroding the vertebræ.

3. Signs are Prominent.—There are associated subjective symptoms but they are distinctly subordinate to the objective manifestations of expansile pulsation, circumscribed dulness, thrill, diastolic shock, and tracheal tugging. This group comprises certain aneurisms of the convexity of the ascending portion of the arch which project to the right and exert moderate pressure chiefly upon the right lung, and some large aneurisms in this situation which have eroded the chest wall and formed projecting external tumors, with great relief from the pressure symptoms of an earlier period in the course of the disease. In both these sub-groups the contrast between the prominence of the physical signs and the moderate intensity of the symptoms may be very marked. Broadbent's division into the aneurism of symptoms and the aneurism of physical signs serves an important purpose in indicating the data upon which to base a diagnosis.

4. Symptoms are Severe and Signs are Distinctive.—Under this heading may be grouped a great majority of aneurisms of the ascending and transverse portions of the arch which have attained considerable size and are sufficiently free from coagula to constitute pulsating tumors. Both those still within the chest and those which have perforated the chest wall and form external masses belong to this group; also those aneurisms

of the descending aorta which compress the left lung or erode the vertebræ, and in particular those which erode the ribs and appear as external tumors at the back.

The connective-tissue overgrowth in the inflammatory capsule involves and compresses the nerve-trunks with which the tumor comes into contact. To this fact is to be attributed the pain so characteristic of growing aneurismal tumors, the palsies of the recurrent laryngeals and pneumogastric, and derangements of the sympathetic.

When neighboring blood-vessels are implicated in the growth, to which the venous trunks and the pulmonary artery are particularly liable, they are narrowed and the veins may be completely compressed, or perforation into the superior vena cava may take place. In a similar manner the trachea, a main bronchus, or the œsophagus may be involved and compressed, or the sac may rupture into these organs.

A

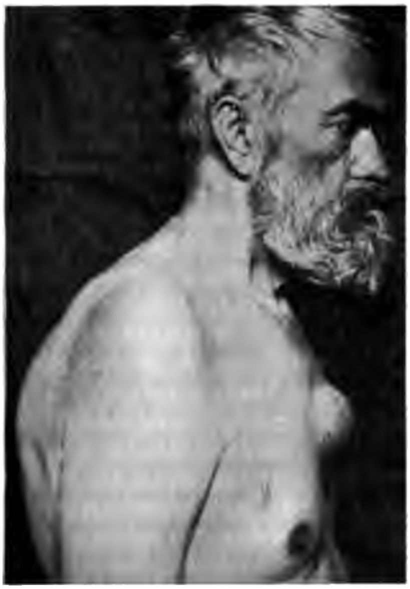

Fig. 355*A*.—Aneurism of the arch of the aorta protruding through the sternum.—German Hospital.

B

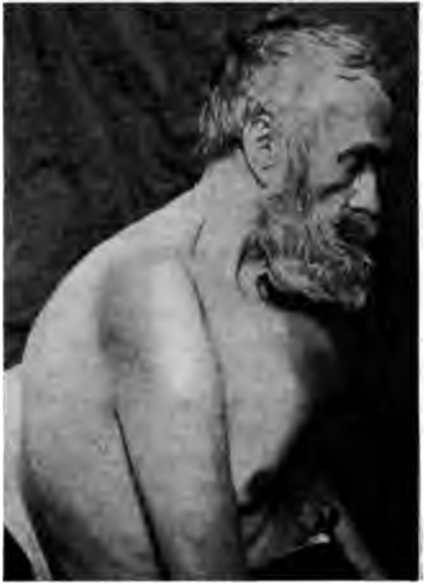

Fig. 355*B*.—Aneurism of the arch of the aorta protruding through the sternum. The same patient one year later. Death occurred suddenly from external rupture through the skin.

The sternum, costal cartilages, ribs, and the bodies of the vertebræ undergo more or less extensive erosion under the pressure of the distending sac. In rare instances portions of the bony wall of the chest are enclosed in the advancing sac, and a fragment of a rib or the end of a partly eroded clavicle is found within its capsule.

Hemorrhage.—The adventitia, even when reinforced by encapsulating connective tissue, may, in the course of a short time, yield to the blood-pressure. When the rupture takes place into the trachea, a bronchus, the œsophagus or stomach, the pleura or pericardium, death usually occurs at once. If the sac is surrounded by dense connective tissue a hæmatoma is formed immediately at the seat of rupture and infiltrating the adjacent parts. Under these circumstances the bleeding may be arrested for a period, only, however, to recur from time to time until fatal hemorrhage ultimately takes place. The blood loss depends upon the amount of thrombus within the sac. If there is a considerable quantity of stratified clot, as is usually the case in aneurisms that have perforated the wall of the chest and ruptured externally, the bleeding may be moderate and for a time controllable. Recurrent hemorrhage frequently takes place in such cases for weeks or months. Protracted or recurrent small bleedings may occur under similar conditions into the trachea, bronchi, or œsophagus.

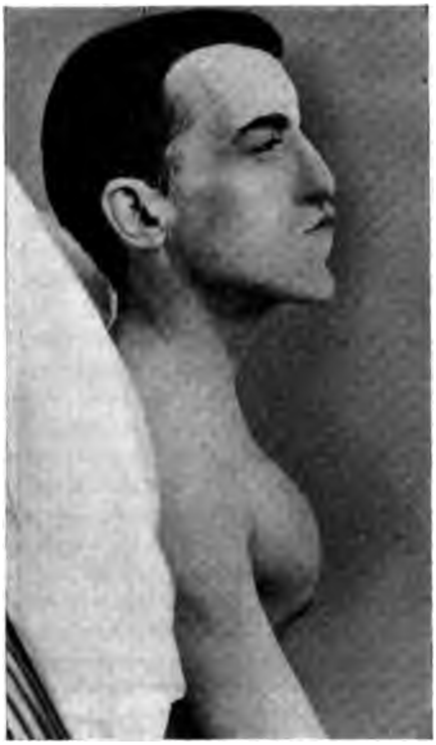

FIG. 356.—Aneurism of ascending aorta. Relief of pain upon appearance of external tumor, which occurred under observation.- Jefferson Hospital.

Pain is an early and important symptom. It may be in the sac itself,—*intrinsic*,—due to irritation of the sac or internal pressure. This pain is dull, aching, and substernal. More commonly it is in the adjacent parts,—*extrinsic*,—due to irritation of nerve-trunks implicated in the advancing capsule or subjected to pressure. This kind of pain is continuous, with paroxysmal exacerbations of great intensity, and is particularly severe at the time of erosion of the vertebræ or the wall of the chest. It is described as sharp, lancinating, cutting, boring, and the like. It often radiates along the intercostal nerves, or into the side of the neck and down the left arm. When the bodies of the vertebræ are eroded, the pain radiates in the course of the intercostal nerves and is often intense, suggesting herpes zoster or intercostal neuralgia. The pain of aneurism, usually spoken of as neuralgic, is in point of fact a symptom of pressure neuritis. When the aneurism is situated at the root of the aorta, attacks of angina pectoris of varying intensity may occur, with radiation to the left side of the neck and arm.

Cough is a common symptom. It may be caused by irritation pressure upon the vagus or recurrent laryngeal nerve, compression of the trachea or a main bronchus, in which event it is often associated with stridor, or by bronchitis. The expectoration is often blood-stained from interference with the venous circulation or from granulations at a point of impending rupture. In bronchitis and bronchiectasis it is thin and abundant. It is sometimes purulent and offensive.

Dyspnœa may be laryngeal, tracheal, or pulmonary, and these forms are sometimes present in the same case. Laryngeal dyspnœa is due to irritation of the recurrent nerve and is usually associated with a ringing brassy cough, aphonia, and hoarseness. The tracheal form has its origin in direct compression of the windpipe or left primary bronchus and is accompanied by stridor; while pulmonary dyspnœa may be caused by compression of one or both lungs by an enormous aneurism of the lower or posterior wall of the transverse arch.

Dysphagia may arise when the sac develops in relation with the gullet. It may be due to œsophagismus or stenosis from compression. A clinical rule, in no case to be disregarded, is never to pass an œsophageal bougie if there is reason to suspect thoracic aneurism.

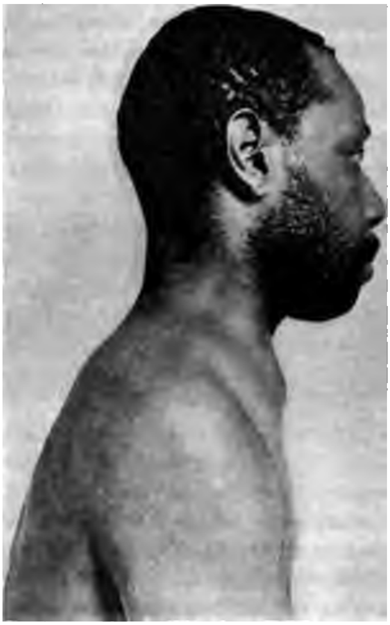

FIG. 357.—Aneurism of the aorta and innominate artery.

The signs of a thoracic aneurism are due, (a) to the tumor itself, and (b) to pressure upon adjacent structures.

Physical Signs.—INSPECTION.—In a large proportion of the early cases, and in many throughout the whole course of the disease, this method yields no physical signs. The tumor while still wholly intrathoracic may cause local bulging of the chest wall with widening of the intercostal spaces and visible pulsation. The signs may elude observation save in a good light and with oblique illumination. When perforation of the chest has occurred there is a more or less prominent external pulsating tumor, at the summit of which, in late cases, the skin may be livid or necrotic and the seat of hemorrhagic oozing. Such tumors are most common at the upper part of the sternum and to the right, and are sometimes of large size, extending also to the left. When the innominate is involved the pulsating prominence projects above the right clavicle and episternal notch. An aneurism of the descending aorta may give rise to a tumor to the left of the spine. Pressure upon venous trunks frequently causes congestion of the face and eyes, and enlargement of the superficial veins of the trunk and arm. These signs are much more common upon the right side. In consequence of pressure upon the sympathetic occasional differences in the pupils arise. Irritation of the upper dorsal or lower cervical ganglion upon the affected side is attended with dilatation of the pupil, which may or may not be accompanied by pallor of the face upon the same side; while destructive pressure is followed by contraction of the pupil and in some cases by flushing and sweating upon the same side of the face. The larynx may be seen to be displaced to the left, much more frequently to the right, by the pressure of an aneurismal tumor, and in large aneurisms of the arch tracheal tugging may sometimes be seen. The visible apex beat is displaced downward and to the left, indicating more commonly dislocation of the heart from pressure than dislocation of the apex from hypertrophy.

Palpation.—This method confirms the signs obtained upon inspection. The extent, force, and expansile character of the pulsation are determined by this method of physical diagnosis. Intrathoracic aneurisms of considerable size may cause general expansion of the upper part of the thorax in its anteroposterior diameter, which is best recognized by bimanual palpation, one hand being firmly applied to the back, the other to the front of the chest. There may be merely a diffuse pulsation. An aneurism which has perforated the wall of the chest forms a distinct tumor with expansile pulsation, the denseness of which is determined by the amount of coagula. Very rarely the sac is thin-walled and fluctuating. There is usually a distinct diastolic shock, which may be intense and is of diagnostic significance. A systolic thrill may often be felt but is to be distinguished from the thrill of a dilated aorta and from the thrill which is present in aortic stenosis. Retardation of the pulse is common. The radials may be unequal in time and volume. When the sac is very large the pulse in the arterial trunks beyond it may be scarcely perceptible.

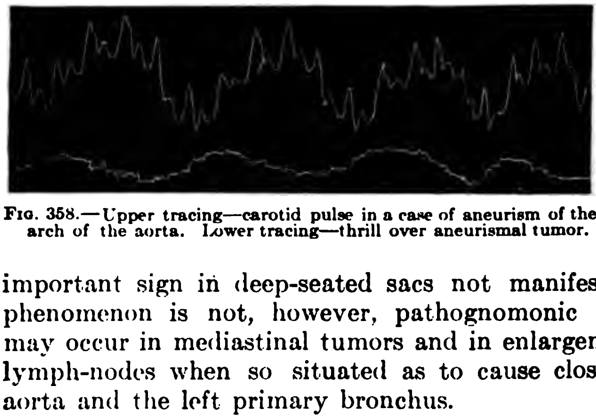

Fig. 358.—Upper tracing—carotid pulse in a case of aneurism of the arch of the aorta. Lower tracing—thrill over aneurismal tumor.

Tracheal tugging is common in aneurisms of the transverse arch and is a very important sign in deep-seated sacs not manifest by other signs. This phenomenon is not, however, pathognomonic of aortic aneurisms. It may occur in mediastinal tumors and in enlargement of the peribronchial lymph-nodes when so situated as to cause close adhesions between the aorta and the left primary bronchus.

Percussion.—Deeply situated aneurisms of small size yield no signs. The percussion signs of larger aneurisms which approach the chest wall depend upon the fact that they displace the lungs. The pulmonary resonance is replaced in aneurisms of the ascending limb of the arch by circumscribed flatness, usually to the right of the manubrium sterni; in aneurisms of the transverse arch by flatness in the upper sternal region to the left of the sternal border; and in those of the descending aorta by flatness along the left side of the spine and in the scapular region. The area of flatness corresponds to the region of contact between the tumor and the inner wall of the thorax, and is surrounded by a very narrow border of dulness and an outer border of tympanitic resonance, the width of which is determined by the extent to which the circumjacent lung is compressed.

Auscultation.—Murmurs may be absent even in aneurisms of large size. The greater the amount of stratified coagula the more nearly the aneurism resembles a solid mediastinal tumor. An accentuated second sound is a common and significant sign. A systolic murmur is often heard; less frequently a diastolic murmur, which is not a sign of the aneurism but of an associated aortic insufficiency. An extremely rare continuous murmur with rhythmical whiffs corresponding to the systole heard over the manubrium is a sign of an arteriovenous aneurism or communication with the pulmonary artery. A systolic murmur may sometimes be heard over the trachea or at the open mouth of the patient.

The following clinical phenomena associated with aneurisms of different parts of the aorta are of diagnostic importance:

Aneurism of the Ascending Portion of the Arch.—Small sacs close to the root of the aorta are latent. Larger aneurisms arise most frequently from the convexity and develop toward the right. Deep-seated pain is an early symptom. It is most severe behind the manubrium and radiates to the neck, shoulders, and arms, more frequently the left than the right. Flatness in the sternal end of the second, third, and sometimes also the first intercostal space indicates the extension of the tumor to the right. In this region the heart sounds are loud and the second aortic sound is commonly ringing and accentuated. Expansile pulsation is present. The apex of the heart is displaced downward and to the left. Aneurisms of this part of the aorta may, in rare instances, communicate with the superior vena cava or compress the inferior vena cava, causing œdema of the lower extremities, and ascites. An aneurism springing from the concavity of the ascending portion may extend beyond the left sternal border. The right recurrent laryngeal nerve is exposed to pressure. Sclerosis of the coronary arteries, sclerotic lesions of the aortic cusps, and relative insufficiency are common. When a sacculated aneurism in this position attains considerable size, the overlying manubrium and cartilages and ribs to its right form a distinct, rounded, pulsating prominence, which in the course of time may cause absorption of the wall of the chest and appear as an external tumor. Systolic murmurs are common and are occasionally audible at a distance from the chest. Compression of the neighboring veins occurs early. The jugulars, especially the right, are prominent. The little venous twigs overlying the tumor are enlarged. The trachea and left bronchus are frequently compressed. Tracheal tugging may occur when firm adhesions with the trachea or bronchus are established. Difficulty in swallowing is not common in aneurisms in this situation.

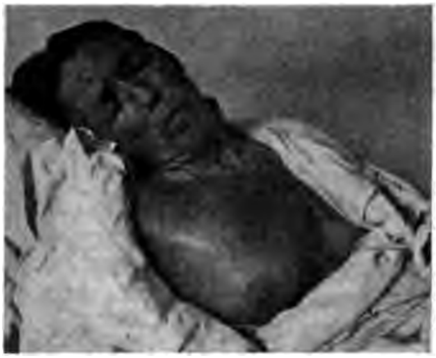

FIG. 359.—Aneurism of ascending portion of the arch of the aorta.

Aneurism of the Transverse Portion of the Arch.—The sac may cause prominence of the manubrium and the cartilages and ribs to its right, as in aneurisms of the ascending aorta. The innominate is frequently involved, with the appearance of an external pulsating tumor at the sternoclavicular articulation and extending upward into the neck. Compression of the left recurrent laryngeal nerve is common. An early symptom is hoarseness with a curious high-pitched vibrating quality of the voice. In slight palsy and in older cases the voice may be unchanged. Pupillary derangements are sometimes observed. Compression of the trachea and left bronchus, and tracheal tugging are very common. Dysphagia is more frequent than in aneurisms in other portions of the aorta. The sac sometimes includes portions of the ascending and transverse arch, causing dulness to the right and upward, and can often be felt pulsating in the episternal notch.

Sacculated aneurisms in the concavity of the arch are always difficult of recognition. Even when of moderate size they may cause persistent, even fatal, hæmoptysis. They elude physical examination and rarely attain a size sufficient to displace the heart. This form of aneurism may be suspected when, with persistent or recurring hæmoptysis in the absence of recognizable cause, are associated paralysis of the left recurrent nerve, dislocation of the trachea and larynx to the right, tracheal tugging, stridor, and dysphagia; but none of these is constant.

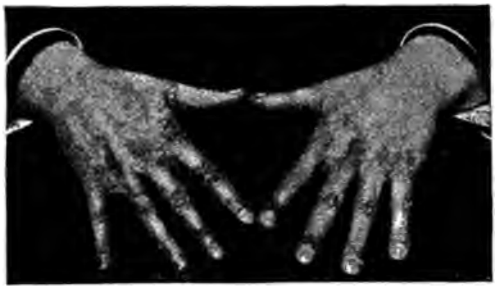
FIG. 360.—Unilateral clubbed fingers in aneurism of the descending arm of the aortic arch.—Groedel's case.

Aneurism of the Descending Arch.—These tumors may also be latent. Pain is a common symptom. It may be intrinsic. Very often, however, it is due to erosion of the dorsal vertebræ. Dyspnœa and stridor from compression of the left bronchus and lung, bronchitis, bronchiectasis, and bronchorrhœa may occur. Left-sided recurrent nerve palsy or paralysis, difficulty in deglutition, left pupillary phenomena are of diagnostic importance. When the sac is large, retardation of the crural pulse as compared with the radials or the apex beat is a less important sign. At the left sternal border in the first and second intercostal spaces there may be dulness, with distinct heart sounds, murmurs, and pulsation. Perforation may take place and an external tumor present in this region—an uncommon event.

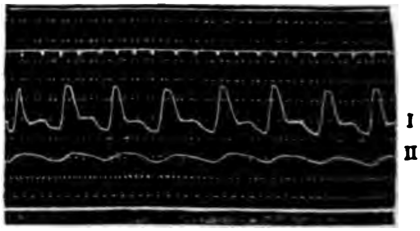

FIG. 361.—Sphygmograms in aneurism of the descending arm of aortic arch. I, right radial pulse; II, left radial. Same case as Fig. 360.—Groedel.

Aneurisms of the Descending Thoracic Aorta.—The sac is usually low down, resting upon the diaphragm and the left side of the bodies of the lower dorsal vertebræ, which are eroded. Among the pressure phenomena are dysphagia, intense pain radiating to the left, pleuritic friction, impaired resonance, together with feeble respiratory sounds and small mucous râles in the lower scapular region near the spine. A pulsating external tumor may present in the back. Pain is usually present, but the other phenomena may be wholly absent and the condition overlooked.

As a rule large aneurisms are sacculated and increase in size without involving other portions of the aorta than that from which they spring.

Exceptionally an aneurism arising from one portion enlarges by involving adjacent parts of the aorta until several parts are successively implicated, as the ascending portion, the transverse arch, and to some extent the descending arch. Under such circumstances the rational symptoms and physical signs of aneurism in the various localities are successively developed. Still more rarely multiple aneurisms are present. In the latter case one may be recognized by suggestive symptoms and characteristic signs, while the others may escape detection.

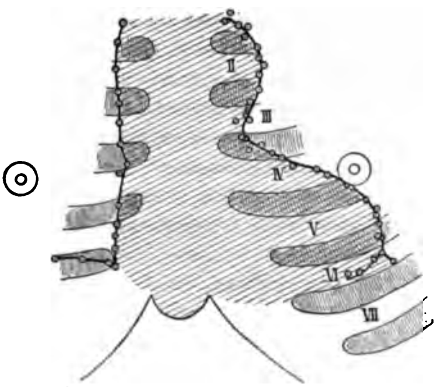

FIG. 362.—Orthodiagraphic outline of left border of aneurism of descending limb of arch of the aorta, showing displacement of the heart downward and to the left. Same case as Fig. 360.—Groedel.

Diagnosis. — DIRECT. — There are cases in which a positive diagnosis cannot be made. Aneurisms of the root of the aorta and of the concavity of the arch, when small, are usually wholly latent. The anamnesis is important. Syphilis, occupation, strain, blows and contusions of the chest are highly suggestive. Alcoholism is of secondary importance. Middle age and the male sex are predisposing influences of weight. Among the symptoms those which arise from intrathoracic pressure are significant. These comprise pain, dyspnœa, stridor, cough, dysphagia, pupillary differences, and a peculiar hoarseness with a high-pitched, shrill, vibrating quality of the voice. Among physical signs which point strongly to aneurism are, in the areas named, circumscribed flatness shading off to dulness, prominence with pulsation, a systolic murmur, systolic thrill, diastolic shock, displacement of the apex downward and to the left (especially when the signs of cardiac enlargement are absent), enlargement of superficial veins, inequality of radial pulses, a retardation or absence of crural pulsation and tracheal tugging. None of these clinical phenomena is diagnostic. The association of all of them is conclusive. When several of them are present the diagnosis becomes probable. An external tumor with distinctly expansile pulsation and diastolic shock justifies a positive diagnosis. Two facts are to be borne in mind. First, an enlarged lymph-node lying directly over a large vessel may pulsate synchronously with the action of the heart, but the pulsation is not expansile; and second, tracheal tugging may be present when, in mediastinal tumor or enlarged peribronchial lymph-nodes, there are close adhesions between the aorta and the left bronchus. Tracheal tugging is, therefore, not a pathognomonic sign of aneurism. The X-rays are of great value as showing, upon fluoroscopic

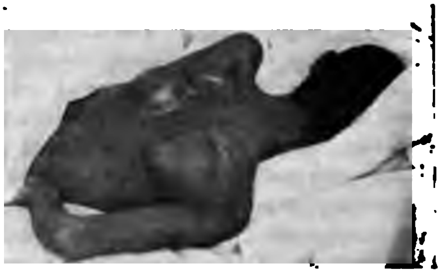

FIG. 363.—Aneurism of the descending thoracic aorta.

examination, a shadow in an abnormal situation, the borders of which expand and contract with the diastole and systole of the heart. Such a shadow is to be differentiated from the non-expansile movement which attends the advance and retreat of a tumor moved by the pulsation of the aorta or heart. The shadow of an aneurism attached to the trachea or left bronchus may rise with the act of deglutition, while tumors, in consequence of their firm attachments to the surrounding structures, are not affected by swallowing.

The situation of an aneurism may be determined by the foregoing symptoms and signs. But there are exceptions to this rule. Thus a sac springing from the ascending portion of the arch may present at the left border of the arch, while one connected with the descending portion may extend to the right of the manubrium and cause pulsation in the right interscapular space; and the possibility that an elongated aneurism of the ascending portion may cause dulness and pulsation to the right of the lower part of the sternum must be considered.

DIFFERENTIAL.—Sclerosis of the aorta can scarcely be differentiated from fusiform aneurismal dilatation of the aorta. The fluoroscopic shadow is circumscribed in saccular aneurisms; diffuse and uniform in arteriosclerosis and the dilatation which occurs in functional derangements, as some cases of neurasthenia and exophthalmic goitre. Pain, which is an early and continuing symptom in aneurism, is not a prominent symptom in sclerosis. *Dynamic Pulsation.*—Increased dulness at the level of the upper part of the manubrium and pulsation in the episternal notch are sometimes present in cases in which, at the autopsy, no dilatation of the aorta is found. This form of widening of the aorta, if persistent, cannot, during life, be differentiated from fusiform aneurism. *Dislocation of the Aorta in Spinal Curvature.*—The convex border of the ascending limb of the arch may be displaced in such a manner as to cause dulness and forcible pulsation beyond the right border of the sternum and simulate an aneurism. Pressure symptoms and pain are usually absent, the pulsation is not expansile, and there is no diastolic shock. *Solid Intrathoracic Tumors.*—These are very often malignant. They take origin in the mediastinal lymph-nodes, the pleura or lungs, the thyroid body, or the œsophagus. A persistent thymus may be greatly enlarged. Such new growths commonly show a tendency to develop both to the right and left of the sternal borders, and not, as is usually the case in aneurism, upon one side. The heart sounds are not so loud as in aneurism and diastolic shock is not felt. Murmurs may occur but are far less common. Differences in the radial and retardation in the crural pulse are likewise absent. Glandular metastases may be present in the neck—a sign of great significance. Pulsation is common but not expansile. Symptoms of pressure upon the recurrent laryngeal nerves are common, but other pressure symptoms are less prominent than in aneurism. The outline of an aneurismal sac is rounded and usually regular; that of a tumor uneven and irregular. The course of malignant disease is rapid, emaciation pronounced, and cachexia early developed; that of aneurism relatively slow, and the fatal issue may occur while the general nutrition is yet fair. There may be the history of a primary growth which has been removed, or the indications of its presence in a distant organ or part. Thoracic tumors and aneurisms containing much stratified clot cannot always, when deep seated, be differentiated. *New Growths Involving the Wall of the Chest.*—Osteosarcoma may involve the sternum or ribs. The overlying veins are greatly enlarged and tortuous. The osseous structures are palpably enlarged and involved. There is local prominence, but the symptom-complex of aneurism is not present. Caries, osteomyelitis, or actinomycosis may cause a rounded, fluctuating tumor near the sternum or ribs. In aneurism the bony structures of the chest wall may be recognized as overlying the tumor, and, when perforation has taken place, as entering into the formation of the opening through which the tumor protrudes, while in abscess they may be recognized as underlying the fluctuating tumor and separating it from the cavity of the thorax. In the very rare cases of mediastinal abscess the onset is abrupt, substernal pain is intense and persistent, and there are grave constitutional symptoms. *Pulsating Empyema.*—Intrathoracic pulsating empyemata give rise to more or less diffuse pulsation upon the left side in the anterolateral aspect of the chest. The signs of pleural effusion are present, the heart is usually displaced to the right, and Traube's semilunar space modified. Empyema necessitatis, when the tumor is in relation with the heart and pulsates, may simulate aneurism. The signs of left-sided pleural effusion, the fact that the tumor diminishes upon inspiration and increases upon expiration, the absence of heaving, forcible impulse, and diastolic shock, of the pressure symptoms incident to aneurism, and of tracheal tugging are conclusive. *Aortic Stenosis.*—A systolic murmur and thrill may suggest aneurism, but a consideration of the history of the case, the nature of the subjective symptoms, and the objective signs of the two conditions render the differential diagnosis an easy matter.

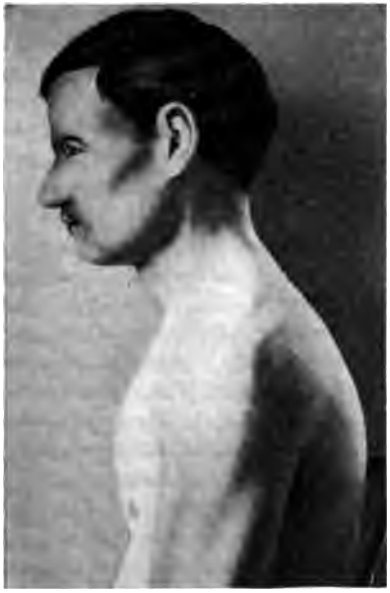

FIG. 364 — Bulging of the anterior wall of the chest in a case of mediastinal tumor. —Jefferson Hospital.

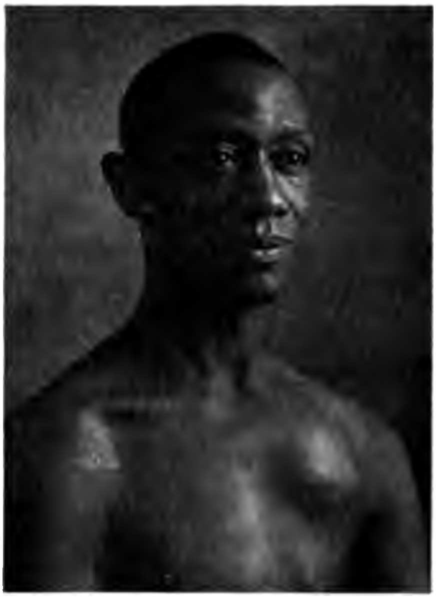

FIG. 365.—Abscess of the chest wall.—Jefferson Hospital.

The Course.—The progress is irregular. Symptoms usually precede signs. There are periods of arrest. The contour of the sac and the pressure phenomena undergo from time to time changes due to changes in the accumulation of clot, its organization or failure to organize at different places, and uneven yielding of the wall under arterial pressure in various regions. In sacs that have perforated the chest wall such local expansions and retractions not rarely take place under the eye. They may be invoked in explanation of changes in the degree of dysphagia, the dyspnœa and stridor, the pupillary differences, and the locality and intensity of the pain. A knowledge of these facts justifies caution in ascribing diminution or even disappearance of certain pressure symptoms to an actual improvement in the underlying condition. Sacs that rapidly increase in size give rise to more urgent pressure symptoms and are more liable to early perforation than those whose growth is slow. The urgency of pressure symptoms depends in part upon the size of the sac and in part upon its location. Aneurisms of the ascending aorta cause compression of the trachea, a primary bronchus, or the œsophagus, only when they have attained considerable size; while a small sac in the concavity of the arch may compress the left bronchus, or a medium-sized sac springing from the inner aspect of the descending portion may cause difficulty in deglutition. Perforation occurs earlier in dissecting than in other forms of aneurism. In sacs containing much clot the perforation may be small and the bleeding moderate. The blood loss is often, however, rapid and fatal. It may be spontaneously arrested by a layer of clot which may permanently close the rent. As a rule, when hemorrhage is arrested it recurs from time to time and finally proves fatal. Rupture takes place most frequently into the pleural sac or a bronchus; less commonly externally, into the pericardium, the retroperitoneal connective tissue, or the peritoneal cavity; not often into the œsophagus, stomach, or intestine; and extremely rarely into the descending vena cava, the pulmonary artery, or an auricle. When the hemorrhage is free, death occurs at once with symptoms of internal bleeding. Perforation into the pericardium is usually fatal at once; into the retroperitoneal tissues it is usually delayed for a longer period. In the latter situation perforation is attended with intense pain and may at first simulate perforation peritonitis or embolism of the mesenteric artery. When the rent is small or the bleeding restrained by the adjacent structures, there are signs of internal hemorrhage, but life is prolonged for a period. I saw a patient who lived eight hours. Bleeding into a pleural sac causes the signs of an effusion; into a bronchus, hæmoptysis which may suggest phthisis or be moderate and prolonged, giving rise to a suspicion of pulmonary cancer; into the stomach, hæmatemesis; into the gut, bloody stools. Fragments of a thrombus may be detached, causing embolism, most commonly of a cerebral artery.

Prognosis.—Spontaneous cure cannot occur in fusiform aneurisms. Small sacs with narrow openings in rare instances become filled with clot which undergoes organization. In dissecting aneurisms a distant communication with the lumen of the aorta may be established, thus forming along the old course a new channel for the blood stream. These are rare events. In a majority of instances, aneu[illegible] terminates in death in the course of six months to three years. The average duration is about one year. Exceptionally life may be prolonged for several years. A time prognosis in individual cases is hazardous. Slowly developing and arrested aneurisms are of relatively favorable prognosis as compared with those of rapid growth, but unforeseen accidents may at any moment occur. Favorable conditions of life and treatment render, in exceptional instances, the prognosis less unfavorable.

(b) ANEURISM OF THE ABDOMINAL AORTA.

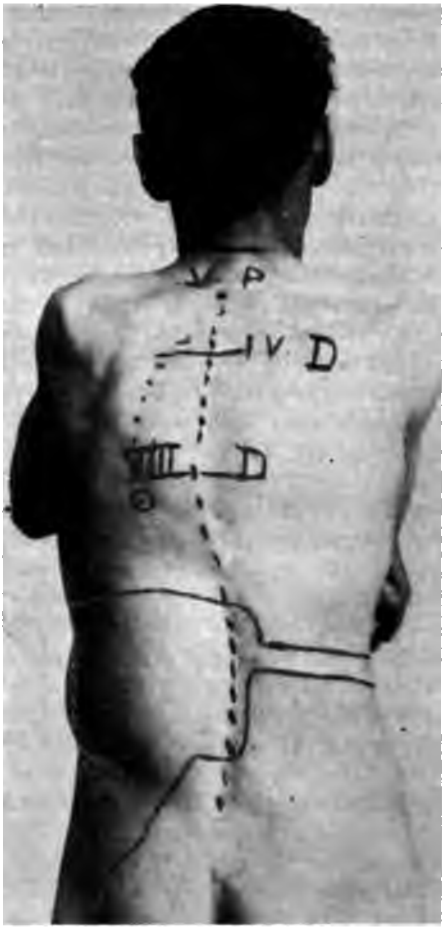

FIG. 366.—Aneurism of the abdominal aorta.—*International Clinics.*

Aneurism of the aorta is far less common below the diaphragm than above it. It may be fusiform or sacculated. In rare instances there are two or more—multiple aneurism. Still more rare in this region is dissecting aneurism. The most common situation is directly below the diaphragm and upon the anterior wall of the aorta, where the sac forms a distinct rounded tumor in the epigastric region in the median line and extending to the left. As the sac enlarges it projects into the left hypochondrium and may occupy a large part of the left side of the abdomen. When it projects posteriorly it causes erosion of the vertebral bodies, or may give rise to distinct spinal symptoms, and, increasing in dimension, may extend into the chest and rupture into the pleura or form an external tumor in the lumbar region. Perforation into the retroperitoneal space may give rise to a progressively enlarging hæmatoma extending into the left lumbar region and simulating a sarcoma.

Symptoms.—Pain is an early symptom. It is referred to the back and is often persistent, radiating to the left flank and marked by intense exacerbations, suggestive of intercostal neuralgia, or renal colic. In many of the cases there are pain-free intervals, or a dull, deep-seated pain in the back increased by movement or jarring of the body. The pain not rarely extends into the abdomen with exacerbations which simulate colic—belly-ache.

Physical Signs.—Upon INSPECTION epigastric pulsation may be present, frequently a diffuse prominence, and occasionally a distinct circumscribed tumor. PALPATION reveals a tumor mass, rounded, smooth, and the seat of forcible, expansile pulsation. A systolic thrill is common. PERCUSSION.—A large sac approaches the anterior wall of the abdomen and causes dulness, which may be continuous with that of the left lobe of the liver. AUSCULTATION reveals a systolic murmur usually best heard over the tumor and transmitted into the crural arteries, sometimes more distinct over the lower dorsal and upper lumbar vertebræ. In some cases there is also a diastolic murmur. These murmurs do not accompany the heart sounds, but follow them. Large aneurisms of the abdominal aorta displace the stomach and to some extent also the liver downward. Distinct retardation of the pulse in the crural arteries occurs when the sac is large.

Diagnosis.—DIRECT.—A distinct circumscribed epigastric tumor in the median line and extending to the left, which can be grasped and which is the seat of expansile pulsation, justifies a positive diagnosis of abdominal aneurism. In the absence of this symptom-complex the diagnosis cannot be made. It is of use to note that in aneurism the pulsation is epigastric, while dynamic pulsation is most marked immediately above or at the umbilicus.

DIFFERENTIAL.—*Dynamic Pulsation.*—The throbbing aorta is very common in nervous women. This pulsation is often very forcible and to a slight extent distinctly expansile, but it is not associated with a tumor, and the course of the aorta may often be felt upon palpation. A systolic murmur may be easily produced by the pressure of the stethoscope. Associated nervous symptoms, and the disappearance of pulsation under the influence of suggestion or other powerful psychic influences are of diagnostic importance. *Tumors of the Pylorus, Pancreas, or the Left Lobe of the Liver.*—New growths overlying the aorta in the epigastrium rise and sink with the movements of the arterial wall and are frequently mistaken for aneurism. The fact that the tumor is not expansile is of diagnostic importance. The absence of murmurs points to tumor rather than aneurism. The pressure of a tumor in contact with the aorta may cause stenosis and a systolic murmur. The crural pulse is not affected. The disappearance of pulsation in the knee-elbow posture, when the tumor falls away from the aorta, is an important sign. The X-rays are of less value as aids to diagnosis in abdominal than in thoracic aneurisms.

Prognosis.—Aneurisms of the abdominal aorta almost always end in death. In rare instances small sacs with a narrow communication with the lumen of the aorta have undergone obliteration by the deposit and organization of clots. Death commonly follows rupture, which may take place into the retroperitoneal tissues, pleura, peritoneum, or intestine. Far less frequently death is due to embolism of the superior mesenteric artery, complete occlusion of the aorta by clot, or paraplegia following erosion of the spine and compression of the cord.

ii. Aneurism of the Cœliac Axis and its Branches.

This branch is frequently involved in aneurism of the abdominal aorta. A negro, twenty-six years old, who was syphilitic, suffered from intense paroxysmal pain radiating to the back and both sides of the abdomen. No tumor could be discovered. Death occurred suddenly with symptoms of internal hemorrhage. At the autopsy there was found an aneurism of the axis the size of a small orange, which had ruptured into the peritoneum. The splenic artery is occasionally the seat of small, sometimes of large, aneurismal sacs. Epigastric pain, vomiting, hæmatemesis, and hemorrhage from the bowel may occur. A deep-seated tumor extending to the left with or without pulsation, and dulness reaching to the spleen, are significant. Rupture into the colon may occur. Aneurism of the superior mesenteric artery may cause a movable pulsating tumor, which is often the seat of a systolic murmur but is not associated with retardation of the crural pulse. Symptoms of mesenteric infarction may occur. Aneurism of the hepatic artery is extremely rare. The symptoms are obscure and a positive diagnosis is impossible. Rupture into the bile passages may occur. The differential diagnosis between aneurisms of these arteries and aneurisms of the aorta is commonly attended with insurmountable difficulties.

iii. Arteriovenous Aneurism.

That form which results from the rupture of an aneurism of the ascending portion of the arch of the aorta into the descending vena cava is of special clinical interest. It is, however, extremely rare. The symptoms usually occur abruptly and consist of marked dilatation of the veins of the upper part of the body, with cyanosis and œdema. A continuous murmur with systolic intensification and a systolic thrill may be recognized.

iv. Periarteritis Nodosa; Congenital Aneurism.

The medium-sized arteries, especially those of the muscles, and the heart, spleen, liver, kidneys, intestines, and the skin are the seat of whitish nodular masses, varying in size from a small shot to a large pea, and great numbers of small aneurismal dilatations. The disease has occurred in both sexes. It manifests itself most commonly in early and middle adult life. It has been ascribed to syphilis and to septic conditions. It is extremely rare. The prominent symptoms are weakness, anæmia, and rapidity of the pulse. There is at first fever, which presently falls without a corresponding decline in the pulse-frequency. When the muscular branches are involved, pain, weakness, and atrophy occur. When the arteries of the gastro-intestinal canal are the seat of the lesions, epigastric pain, thirst, anorexia, nausea and vomiting, and diarrhœa or constipation are prominent symptoms. Hemorrhage from the bowels has been observed. Scanty urine, of low specific gravity, albumin, and casts occur. Urea is diminished. Anæmia is marked. Leucocytosis is common. The course of the disease is progressive, and death occurs at the end of the second or third month. Recovery is exceptional. The diagnosis is usually post mortem. The nature of the affection may be suspected when, in connection with the foregoing symptom-complex, nodular thickenings may be felt in the course of accessible arteries.

XIV.

THE DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

I. MENINGITIS.

Inflammation of the membranes of the brain is common to a variety of affections. Thus, there are purulent meningitis, tuberculous meningitis, syphilitic meningitis, and meningitis due to injury, or associated with disease of bone, or with general paresis. For descriptive purposes we distinguish between inflammation of the dura—*pachymeningitis*—and inflammation of the pia,—*leptomeningitis,*—although the two membranes are often involved together.

Inflammation of the dura mater may occur from septic infection, and may be purulent. The commonest causes are injury, infection, and necrosis of bone. Thus disease of the middle ear is the most frequent cause, but cases may arise from infection through the nose by way of the cribriform plate. Fracture of the skull may sometimes be the starting-point. Large quantities of pus may be present, and the pia mater and brain substance may be involved as well as the dura. There is a variety in which the pus is localized or pocketed between the skull and the dura, usually secondary to injury of the skull or to caries or bone syphilis—the so-called *pachymeningitis externa.*

A distinct form of pachymeningitis is the disease known as hæmatoma of the dura mater—the *pachymeningitis hæmorrhagica* of Virchow. It occurs especially in the chronic insane and in old alcoholic patients. There is observed beneath the dura a layer, quite thick, which looks like organized blood-clot; this often exists, in fact, in several layers, as though caused by successive hemorrhages. Sometimes the appearance is that of a very delicate vascular membrane, enclosing in its meshes blood which is more or less organized. Cysts may be found and other evidences of breaking down of the new tissue.

The **symptoms** of pachymeningitis hæmorrhagica are obscure, being masked by the general condition of the patient, who is usually a chronic lunatic or a confirmed inebriate. A similar lesion has been described after sunstroke, and may account for the headache and mental changes.

Simple, idiopathic, or isolated inflammation of the pia mater is such a debatable condition as scarcely to be considered a clinical entity. The forms of leptomeningitis which we usually distinguish at the bedside are those that occur in septic infection, such as in otitis media, and in cerebrospinal fever, syphilis, and tuberculosis. A very characteristic form is that seen in general paresis, in which the pia mater is thickened and opaque and so adherent to the summits of the convolutions that it is stripped with difficulty, and usually carries away with it a portion of the brain substance. Quincke has described a *serous meningitis* in which the pia-arachnoid especially is involved; it is characterized by mild symptoms, slight fever, and headache, with some stiffness of the back of the neck; and optic neuritis is not uncommon with it.

Symptoms.—The general symptoms of leptomeningitis are headache, fever, delirium, convulsions, rigidity of the neck and face muscles, exaggerated reflexes, followed later by paralysis and coma.

Tuberculous Meningitis (see page 790).

Epidemic Cerebrospinal Meningitis (see page 730).

Septic meningitis may be caused by otitis media, in which case the microbe is usually a streptococcus or staphylococcus; or it may be caused by the pneumococcus. This meningitis is associated with pus formation, also with pachymeningitis and purulent cerebritis, and in not a few cases leads to brain abscess.

Diagnosis.—Meningitis, whether in the dura or the pia mater, depends upon so many causes and is associated with so many pathological processes that it is difficult to lay down uniform rules for the diagnosis. The first essential is to seek for the cause, such as a middle-ear disease, tuberculosis, meningococcic or other infection, injury to the bones of the skull, caries, sunstroke, syphilis, etc.

Symptoms of irritation appear early. These are headache simulating neuralgia, vomiting, optic neuritis, fever, stiffness of muscles, and perhaps convulsions, with irritability of temper, change of disposition, and even somnolence, delirium, or stupor. Later the symptoms are indicative of pressure and profound toxæmia: thus we see various paralyses, increasing stupor merging into coma, rapid pulse, and failing powers.

Meningitis of various kinds may be mistaken for mere neuralgia, but the history and associated symptoms of organic disease should prevent error. Where headache, vomiting, and constipation are early symptoms, as in tuberculous meningitis, the case may suggest gastric or intestinal disorder, but the onset of more pronounced brain symptoms, such as convulsions, stupor, and various paralyses, is unmistakable. The best single general rule for the distinguishing of meningitis is to be on the lookout in suspected cases for the signs of infectious diseases of the brain, and to trace these to their cause. Lumbar puncture may shed important light in cases of infection.

II. ACUTE HEMORRHAGIC ENCEPHALITIS.

The disease is characterized by multiple foci of congestion and both punctate and massive hemorrhage, with infiltration of leucocytes, minute emboli, and localized necrosis of the brain-tissue. The membranes are usually not involved. The reported cases have mostly followed the infectious diseases, such as influenza, mumps, erysipelas, pneumonia, typhoid and typhus fever, and malaria. Nephritis has been observed in some cases. The disease is probably caused by the local action of micro-organisms or by their toxins. Oppenheim called attention to the resemblance of the disease process to the polioencephalitis superior of Wernicke and to the polioencephalitis anterior of Strümpell.

Symptoms.—The symptoms are suggestive of meningitis, which, however, is not present. There are headache, vomiting, convulsions, localized palsies, and affections of consciousness. Rigidity of the neck sometimes occurs. Fever is not constant, and there may even be subnormal temperature. The pulse at first may be slow, but in fatal cases the pulse becomes rapid, the respirations shallow, coma sets in, and death occurs in a few days or weeks. Recovery has been claimed in a few cases.

Diagnosis.—The disease is likely to be confounded with meningitis, and the distinction between the two is not of practical importance, as the course and treatment are nearly the same. In most cases an accurate diagnosis has only been made after death. This form of encephalitis may be suspected when grave cerebral symptoms occur in the course of, or immediately following, any of the infectious diseases mentioned.

III. PURULENT MENINGO-ENCEPHALITIS AND BRAIN ABSCESS.

Pus-forming inflammation within the cranium assumes several forms. There may be a diffuse purulent meningitis, or, as is more common, a meningo-encephalitis; and there may be abscess. The clinical distinction between these forms is not easy.

Etiology. — The causes are the various pyogenic micro-organisms. One of the commonest is seen in purulent otitis media; and more rarely other cranial bones, especially the bones of the nose and the orbit, and their sinuses, may be the starting-point. Accessory sinuses of the nose are frequently infected, and this infection may travel by way of the frontal sinus, sphenoidal sinus, ethmoid cells, or the antrum of Highmore. The streptococcus is the chief germ in otitic brain abscess. Septic infection, arising from any focus in the body, may cause a metastatic brain abscess, but this complication is rather more common in ulcerative endocarditis, abscess of the liver, abscess and gangrene of the lung, and empyema. Trauma of the skull may also cause abscess of the brain. Suppurative meningitis occurs in tuberculosis and in cerebrospinal fever.

Pathology.—The membranes, especially the pia-arachnoid, are inflamed and opaque in purulent meningitis, and pus is diffused beneath them. This pus often follows along the perivascular spaces and the fissures of the brain, appearing as white streaks. In some cases it is more copious, and forms collections beneath the membranes, especially at the base. The cerebral tissue is often involved, the gray and white matter being infiltrated, œdematous, and softened in places. There may also be sinus thrombosis, with engorgement of veins on the outside surface of the skull. When abscess forms it is either diffuse or circumscribed. In the former case the collection of pus is merely an accident of a more or less diffused purulent meningitis. The circumscribed abscess is contained within a limited area, and its walls may even be thickened, forming a barrier between the pus and the brain tissue. These circumscribed abscesses are most common in the temporal lobe of the cerebrum and in the cerebellum. In some cases multiple abscesses are formed, especially in metastasis.

Symptoms.—These are general and focal. Among the general symptoms we include those which indicate a grave cerebral disorder; and among the focal symptoms those which indicate its location in the brain. The *general* symptoms are fever, headache, vomiting, convulsions, paralysis, optic neuritis, and affections of consciousness. Fever is not constant nor always of one type; it may be slight and easily overlooked; again it may be more pronounced, and accompanied with chills and sweating. It may depend as much on the primary condition (otitis media, abscess of the lung, etc.) as on the brain lesion. *Per contra,* in some cases of brain abscess the temperature is subnormal and the pulse very slow. Headache is usually present in suppurative meningitis, but it is not an altogether constant symptom in brain abscess. It was present in 78 out of 169 cases in Allport's table. In some cases, however, it is an early indication of an insidious onset. Its localizing value is not always .great; in cerebellar abscess, for instance, the pain has been observed in other parts of the cranium, even in the frontal region. Occasionally, however, the pain is strictly localized at the region of the abscess, and pressure and percussion on the skull at that point may be painful. Vomiting is frequent in cerebellar abscess, but more rare in diffuse purulent meningitis; and it is usually of the cerebral type—propulsive and not dependent on food in the stomach. Convulsions may or may not be present; there is no positive rule about them. They sometimes have localizing value. The same can be said of paralysis. Optic neuritis is a most important symptom of abscess of the brain, although not present in all cases. In children, if the pus formation be rapid, optic neuritis will soon ensue. It has no localizing value, but its presence in cases of latent or suspected abscess is most significant. Affections of consciousness are common in all forms of suppurative disease of the brain, and they range from mere apathy and stupor to wild delirium and profound coma. In cases of latent abscess changes in the temper and personality, such as irritability and depression, are observed.

By the *focal* symptoms we attempt to determine the site of the abscess. These symptoms are mostly pain, paralysis, and convulsions.

Pain, as already said, is not altogether reliable as a guide. Both in cerebellar and temporosphenoidal abscess the pain is sometimes frontal, or it may be more generally diffused. When sharply localized, as in the occiput or nuchal region, especially if it is increased by percussion, it may prove a safe guide.

Paralysis may be clearly indicative of the site, especially if the abscess involves the motor regions, as the pre-Rolandic area and its subcortical connections. This may occur either in frontal or parietal abscess, the pus extending backward or forward respectively. We then have hemiplegia or monoplegia (facial, brachial, or crural), according to the centres involved, and in left-sided lesions there is aphasia. Sensory aphasia, especially word deafness, is strongly indicative of a temporal abscess on the left side, such as is common to ear disease, and it has led the way to successful operation. Temporal abscess may also give rise to hemiplegia by pressure across the Sylvian fossa upon the internal capsule; hence this symptom may be misleading. Hemiplegia has even been caused by abscess of the cerebellum. The hemiplegia in such cases is probably caused by pressure on the pons or medulla oblongata. In this way also cerebellar abscess may cause other pontile symptoms, as paralysis of the fifth, sixth, seventh, and eighth nerves, and the mid-brain may even suffer, with a consequent partial ophthalmoplegia. According to Allport's table strabismus occurred in 10 out of 98 cases of abscess of the brain, not all cerebellar. From these various data it is seen that paralytic symptoms must be interpreted with care. For instance, thrombus of the cavernous sinus, which might occur in any purulent process in the brain, causes an ophthalmoplegia, as may also an abscess of the temporal lobe by pressure on the third and sixth nerves.

Involvement of the sensory tract, causing hemianæsthesia and hemianopsia, has been reported, especially in abscess of the right temporal lobe. Various affections of the visual fields may be caused by abscess in the occipital lobe.

Focal epilepsy usually indicates a lesion in or about the motor region, but this symptom is not so common as in brain tumor. Unilateral convulsions have been reported in cases of abscesses at various sites, even in the cerebellum. Tonic spasm in the nape of the neck may be caused by abscess of the cerebellum.

When a thrombus forms in a sinus the veins on the outside of the skull may be congested: thus in thrombus of the cavernous sinus the veins of the orbit are engorged; and the veins at the base of the nose and on the brow are sometimes congested from thrombus of the superior longitudinal sinus.

Among other noteworthy symptoms are vertigo, incoördination, loss of equilibration, especially but not always in cerebellar abscess, and abolished knee-jerks. Macewen has noted this last symptom in cases of cerebellar abscess, and it has been seen in cerebellar tumor, especially of the middle lobe. Occasionally it is not continuous, the reflex disappearing and reappearing. Knapp has seen a case of cerebellar lesion in which only one knee-jerk was lost, and that on the side opposite to the lesion. Abscess of the frontal lobe may cause obscure mental symptoms, especially retarded cerebration. In Allport's collection of 98 cases of abscess of the brain, 40 occurred in the temporal lobe, 31 in the cerebellum, 7 in the parietal lobe, and the remainder in various other regions. In 5 cases there was diffuse subdural abscess.

Diagnosis.—Purulent meningitis and brain abscess, although included here under one heading and having much in common, should be distinguished from each other if possible. The two conditions may coexist, or the one merge into the other, and the dividing line is not easily detected. Focal symptoms are not likely to be seen in diffuse purulent meningitis, and the course of this disease is more acute and rapid; in fact, death may occur in a few days. Abscess is likely to be much slower, especially the circumscribed abscess with well-formed walls; and in this connection the latent abscess must not be overlooked; this may endure for weeks, even months, with only very obscure symptoms. In fact, a latent or premonitory stage is not uncommon in solitary abscess of the brain, but in diffuse abscess, and especially multiple abscess, such as is caused by metastasis from some septic process outside the brain, the case is likely to be more rapid and more like a diffuse suppurative meningo-encephalitis.

Some authors attempt to distinguish extradural abscess, such as may occur in trauma and even in otitis. The local symptoms of such an infection are usually evident, as pain, swelling, venous engorgement. The presence of an external wound is important.

Tumor of the brain is distinguished from abscess by its different clinical history, its slower onset, the absence of a septic process of origin, and, as a rule, of chill and fever. But in rare cases tumor of the brain is complicated with suppuration. Optic neuritis is rather more common in tumor, but only little reliance can be placed on that fact. The evolution of symptoms is usually more gradual and progressive in the case of tumor.

Cerebellar abscess may simulate Ménière's disease by vertigo and occasional deafness. The clinical history, however, is different, and in case of abscess there are likely to be fever, headache, and mental changes.

Cerebral hemorrhage and softening may simulate abscess when the latter is fully formed, but the clinical history is so entirely different that the distinction is easy, as a rule. An abrupt onset of symptoms may occur in case of latent abscess, if the pus breaks from its cavity, causing paralysis, convulsion, coma, etc.; and in such a case the differentiation is perplexing.

In all cases of septic infection, such as suppurating otitis, abscess of the lung, etc., the onset of cerebral symptoms should excite suspicion, and the case should be carefully scrutinized. Lumbar puncture may give valuable information.

IV. SINUS THROMBOSIS.

Thrombosis of the cranial sinuses is either primary or secondary. In the former the thrombus arises from some general blood state, in the latter from some disease, usually septic, in the immediate neighborhood.

Etiology.—Primary thrombosis is seen in conditions of exhaustion, often as a terminal symptom; thus it occurs in advanced stages of tuberculosis, carcinoma, the infectious diseases, as typhoid fever, and in the diarrhœas of infancy. It is favored by a weakened heart and by the sluggish circulation in the sinuses. It has been seen also in chlorosis and anæmia.

Secondary thrombosis results from disease of the walls of the sinus, hence especially from injury or caries of bone. Its commonest cause is otitis media, in which case the lateral and transverse sinuses especially are involved. It also arises by way of the nasal bones, and in fact, though rarely, from any other bones of the cranium which become the seat of caries. It is sometimes caused by fractures; and it also arises from septic processes within the skull, such as a purulent meningitis from any cause, or from a general septicæmia or pyæmia arising from causes outside the cranium. Facial erysipelas may be a cause.

Pathology.—In septic or secondary thrombosis the sinus is partly or entirely filled with a white or grayish-white mass, adherent to the walls.

This mass may be purulent and sanious, and the walls of the sinus are inflamed, infiltrated, and discolored, while the contiguous bone in many cases, as in otitis, is carious and softened. In recent cases the thrombic mass is soft and easily broken up, but in older cases it is quite firm and fibrous. It may extend for some distance through the sinus, even into some of the tributary veins, thus causing engorgement of veins, with swelling and œdema, on the outside of the skull. These septic thrombi are not seldom associated with other septic lesions, such as purulent meningitis, purulent encephalitis, and even brain abscess.

Symptoms.—These are general and local. In the case of the secondary septic thrombi the *general* symptoms may be masked by those of the general pyæmia; thus there is fever, usually of a septic type, with headache and changes in consciousness, and there are not seldom the evidences of meningitis. Convulsions sometimes occur, and various paralyses, and occasionally a high grade of choked disk. In the case of primary thrombosis we have to consider the original disease and its exhausting effects, but superadded to these we observe grave cerebral symptoms of sudden onset, such as headache, vomiting, stupor, followed by a gradually deepening coma, possibly with convulsions; but local paralytic symptoms are rare in this form. There may, however, be hemiplegia, or even a general flaccid paresis.

The *local* symptoms are sometimes conspicuous. They depend usually upon engorgement of tributary veins on the outside of the skull and upon paralysis of certain cranial nerves.

In thrombosis of the cavernous sinus there are protrusion of the eye, swelling and discoloration of the tissues about the eye, engorgement of the veins of the orbit and the frontal veins, which communicate through the orbit with this sinus, possibly choked disk, and paralysis of the third, fourth, and sixth nerves, which run through the sinus. There may also be pain or anæsthesia in the ophthalmic division of the fifth nerve. The central retinal vein may also be the seat of a thrombus.

Thrombosis of the superior longitudinal sinus may cause cyanotic swelling and œdema on the brow, and in rare cases nose-bleed. Infection of this sinus may occur through the nasal bones and be accompanied with a purulent meningo-encephalitis. The commonest site of cranial thrombosis is in the lateral and transverse sinuses in cases of otitis media. The local symptoms are disguised by the local bone disease. The most significant are pain and œdema over the mastoid. Otitis media is the most common cause of grave general infection of the cranial contents, the most serious being abscess. Irritation from this focus may cause recurring epileptic fits. The jugular vein, external or internal, may be the seat of thrombus, which may even be palpable. A gravity abscess may simulate phlebitis of the jugular; and the glossopharyngeal, vagus, accessory, and hypoglossal nerves have been paralyzed in some of these cases.

Diagnosis.—A local diagnosis is hardly practicable for any other of the cranial sinuses than those mentioned above. It is, of course, much simplified in cases in which circumscribed œdema occurs. The general diagnosis is often difficult and problematical. Thrombosis is to be suspected when grave cerebral symptoms, such as headache, vomiting, convulsions, stupor, and unconsciousness, occur suddenly in cases of septic infection, such as otitis media and facial erysipelas, and in wasting diseases, such as tuberculosis, carcinoma, infectious diseases, and infantile diarrhœa. But a differential diagnosis from meningitis, abscess, and softening is not always possible, and in fact some of these conditions may be associated with thrombosis. Thrombi of the cerebral veins are sometimes the cause of hemiplegia and diplegia in young children. Smithers has recently called attention to hemiplegia in typhoid fever, caused by thrombi in the cerebral arteries.[1]

V. CEREBRAL HEMORRHAGE.

Etiology.—The cause of this accident is primarily some disease of the blood-vessels, excluding, as we do here, hemorrhage from trauma. The diseases of the blood-vessels are chiefly arterial sclerosis or atheroma, occurring usually after middle life, and more rarely syphilis, occurring at any period of life and not rarely in young adults. It is not to be overlooked, however, that the hemiplegia of syphilis is usually due to an inflammation and thickening of the walls of a blood-vessel rather than to a hemorrhage.

Pathology.—Atheroma of the blood-vessels is a common affection in later life. It is rare to see an autopsy in a person past fifty without some evidences of it, and in persons of sixty and seventy it is not unusual to see the circle of Willis at the base of the brain so thickened and hardened that the vessels are like pipe-stems. From these main arteries at the base the branches that pass up through the anterior perforated space to the lenticular nucleus and internal capsule are especially liable to suffer. More rarely the branches from the posterior cerebral or those from the basilar, vertebral, and cerebellar arteries are affected. The atheromatous arteries are frequently the seat of minute dilatations, aneurismal in character, and it is one of these that is likely to give way. The hemorrhage is usually within the substance of the brain; meningeal hemorrhage from arterial disease being rare, although common from trauma. Occasionally, however, a hemorrhage breaks through to the surface. The most common seat of hemorrhage is in the lenticular nucleus, which is a part of the basal ganglion (corpus striatum) within the brain. The hemorrhage occurs in such a way as to press upon or destroy the internal capsule which contains the motor and sensory tracts. The weakened artery at this point was called by Charcot the "artery of cerebral hemorrhage."

In recent cases the blood is either still fluid or partly clotted, and it occupies a ragged cavity which it has torn out of the substance of the brain. In old cases this cavity is often found walled off, forming a cyst, filled with a reddish or yellowish fluid. In rapidly fatal cases it is sometimes found that the blood has broken through into the lateral ventricle, or even to the outer surface of the brain. Multiple hemorrhages may occur, and sometimes the hemorrhage is in the parietal or occipital lobe, or even in the pons. Hemorrhage in the cerebellum is less common than in the cerebrum.

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[1] Journal of the Am. Med. Assn., Aug. 3, 1907, p. 389.

Instead of hemorrhage a diseased blood-vessel may cause thrombosis. There is then secondary softening, but clinically the two conditions are much alike and it is quite impossible, as a rule, to distinguish them. Softening is also caused by embolism, with very similar results.

In long-standing cases of hemiplegia there occurs a descending degeneration of the motor tract, which may be traced through the peduncle, the pons, the decussation in the medulla, and the spinal cord.

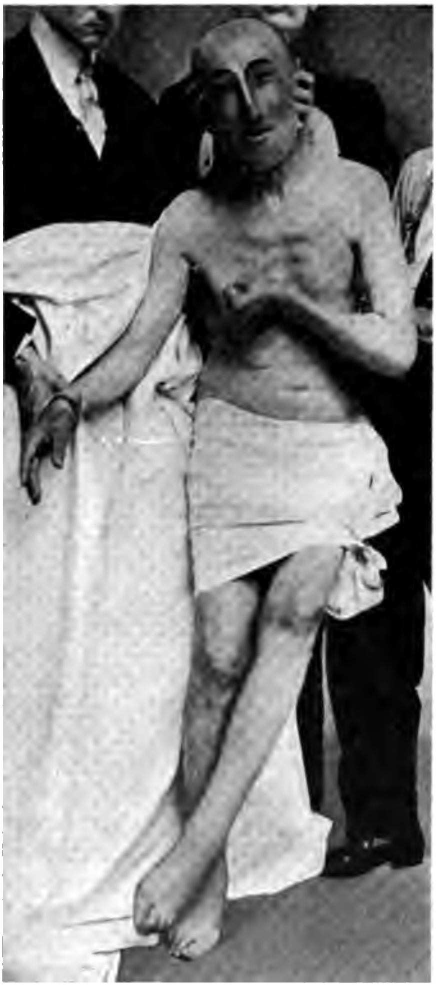

FIG. 367.—Old left hemiplegia with contractures. Lloyd.

Symptoms.—Cerebral hemorrhage causes what is popularly known either as a "stroke" or an "apoplexy." These two conditions are distinguished chiefly by the state of the consciousness; in the former the mind may be clear, in the latter there is stupor or coma. In either case there is likely to be paralysis according to the site of the lesion.

In the lenticular nucleus and internal capsule, the most common site, hemorrhage causes hemiplegia on the opposite side. The arm and leg are paralyzed, the arm rather more so, and in some cases the lower part of the face and one side of the tongue. The upper portion of the face is not involved, so the patient can still shut his eyes and wrinkle his forehead. The tongue, if involved, is protruded toward the paralyzed side. In some cases there is hemianæsthesia, and even hemianopsia, the affection of the sensory fibres showing that the clot has involved the posterior portions of the internal capsule. There may also be various forms of aphasia if the lesion is in the left hemisphere.

When the hemorrhage involves the island of Reil and the posterior end of the third frontal convolution on the left side there is motor aphasia. When the left superior temporal convolution is involved there is auditory aphasia, word-deafness and object-deafness; and when the left angular gyrus is invaded there is visual aphasia, word-blindness and object-blindness. Various mixed forms of aphasia, the so-called sensorimotor aphasia, may occur from hemorrhage into various portions of these speech centres (the so-called language zone) and their subcortical connections in the left hemisphere.

Hemorrhage in the frontal lobe, if it does not involve the motor centres or tracts, may cause very obscure symptoms, more especially mental changes, such as retardation of the mental processes, loss of the power of attention, etc.

Hemorrhage in the superior parietal lobule may cause ataxia of the limbs on the opposite side, and sensory changes, especially astereognosis.

Hemorrhage in the occipital lobe may cause hemianopsia and other partial defects in the visual fields, and also some inability to recognize and name objects by sight.

In the cerebellum hemorrhage may cause intense vertigo, loss of equilibration, forced and pitching movements, and vomiting; and if the clot is big enough to make pressure on the mid-brain and pons, there may be hemiplegia, hemianæsthesia, and involvement of the oculomotor, fifth, sixth, seventh, and eighth nerves; but these latter symptoms are rare, and are rather indicative of either a mid-brain or a pontile lesion. Thus hemorrhage in the pons causes the hemiplegia alternans, in which the arm and leg are paralyzed on the opposite side while the sixth, seventh, eighth, and possibly the fifth nerves are paralyzed on the side of the lesion; this is so especially if the lesion is low in the pons. If the lesion is high, the cranial nerves named may be paralyzed on the opposite side, that is, on the same side as the hemiplegia. In rare cases a very circumscribed lesion that involves the nucleus of the sixth nerve may also cause diabetes or polyuria.

In hemorrhage in the mid-brain (corpora quadrigemina and cerebral peduncles) the oculomotor and fourth nerves may be paralyzed on the side of the lesion, while the hemiplegia is on the opposite side, presenting a type of hemiplegia alternans which is sometimes called the "syndrome of Weber."

Hemorrhage in the medulla oblongata is extremely rare, and is incompatible with prolongation of life if the respiratory centres are involved.

In the apoplectic state consciousness may be partially or entirely lost; the breathing becomes stertorous, the cheeks puff out with every breath, and the pulse may be full and strong. If the case advances toward an unfavorable ending, the pulse becomes thin and rapid, the temperature rises, unconsciousness is profound, the pupils do not react to light and may be unequal. Cheyne-Stokes respiration may set in, and death is often hastened by an œdema of the lungs. In the apoplectic cases the hemiplegia can sometimes be determined by the loss of resistance to passive motion on the paralyzed side.

In some cases the reflexes are not at first greatly affected. In cases of massive hemorrhage with shock and unconsciousness, the knee-jerk on the paralyzed side may be abolished. In patients who survive and partially recover, the deep reflexes become exaggerated on the paralyzed side, the muscles are contractured, and there results a characteristic hemiplegic attitude and gait. Ankle clonus and Babinski's reflex are usually present in these patients.

The state of the pupils is not constant; in the early stages the light reflex may be preserved; but with deep unconsciousness it is usually abolished. The pupils are sometimes slightly unequal; or they may be of normal size or even dilated. In pontile hemorrhage they may be contracted.

Lateral deviation of the head and eyes is sometimes seen in the apoplectic cases, the head and eyes being turned away from the paralyzed side—conjugate deviation. If a spastic state sets in from irritation of the brain-cortex or motor tracts, especially if convulsions occur, as sometimes happens, the head and eyes are forcibly drawn towards the paralyzed side. In rare cases there results a "posthemiplegic chorea"—a bad term, as the disorder is not a true chorea, but a wide to-and-fro tremor.

Diagnosis.—Cerebral hemorrhage, especially when it causes unconsciousness, requires to be distinguished from uræmia, diabetic coma, postepileptic coma, opium poisoning, alcoholic drunkenness, and trauma. The problem is sometimes a difficult one. As a general rule hemorrhage causes a hemiplegia, which can usually be determined, even in cases of unconsciousness, by some difference in the resistance to passive motion on the two sides: on the paralyzed side the limbs are entirely flaccid and fall dead, while on the other side there is usually some resistance. In profound unconsciousness, however, the difference may be difficult to recognize. In cases of a simple paralytic "stroke" without unconsciousness the problem is much simplified, as the history of a sudden attack of hemiplegia is usually determinative. Even in these cases there may be at first some confusion of mind and clouding of consciousness.

Uræmic coma is often ascertainable from the history of the case. The presence of albumin and casts in the urine cannot determine the question positively because a patient with nephritis may have a cerebral hemorrhage or thrombus, while a patient with apoplexy may have albuminuria. Moreover, there are sometimes seen in nephritis attacks of hemiplegia, which pass away with other uræmic symptoms. Transient aphasia and brachial monoplegia of uræmic origin sometimes occur and may simulate organic lesion in the brain. In uræmic coma there may be prolonged subnormal temperature. In spite of the exceptions noted, uræmic coma is, on the whole, indicated by the state of the urine, the history of the case, the subnormal temperature, the usual absence of hemiplegia and other symptoms of a focal lesion, and sometimes by the uræmic odor. Convulsions are in favor of uræmia, although they sometimes occur in hemorrhage. Albuminuric retinitis is also in favor of uræmia. There are doubtful cases which only time can solve.

Diabetic coma is indicated by the glycosuria, diaceturia, and acetonuria, the history of the case, and the absence of hemiplegia. The crisis may be ushered in with headache and delirium, and the peculiar dyspnœa, called by Küssmaul "air-hunger," but these prodromes are not seen in every case.

Postepileptic coma usually clears up in a few hours at most. The history of the case is significant. Convulsions may occur in cerebral hemorrhage, but they are rare. Focal epilepsy, in which a hemiplegia or monoplegia may persist for some time, may be puzzling, but the history of the case and the course should prevent error, especially if focal symptoms are caused by organic lesion, such as tumor. In epileptic "status" the fits recur at frequent intervals (as many as twenty-five and even more in a day), the patient being profoundly unconscious between the paroxysms, with weak pulse, and sometimes with high temperature.

Opium poisoning is, as a rule, easily recognized by the history, the contracted pupils, and the slow respiration; in cases in which the history is unknown mistakes are possible. Massive hemorrhage, especially ventricular hemorrhage, may cause profound unconsciousness, immobile rather than contracted pupils, and labored breathing, while the hemiplegia may be masked; pontile hemorrhage is said particularly to simulate opium poisoning, especially in the contracted pupils. The extremely slow breathing of opium narcosis, however, is not likely to be seen in hemorrhage; while in the poisoning there is never hemiplegia, nor conjugate deviation of the head and eyes; and, finally, the extreme bilateral myosis is hardly equalled in cases of apoplexy, in which inequality of the pupils is more common. The pupil dilates as death approaches in opium poisoning.

Alcoholic intoxication is known by the history, by the odor of alcohol, and by the fact that the stupor or unconsciousness is usually not so profound as in apoplexy. None of these data is entirely reliable. A drunken man may have a cerebral hemorrhage, hence the odor of alcohol on the breath is a most unreliable test for this, as for any case. Hemiplegia is, of course, conclusive as against mere alcoholism, and unequal pupils suggest apoplexy. A few hours usually determine whether a doubtful case is one of drunkenness.

Trauma may cause a condition closely simulating or even identical with apoplexy, as, for instance, when it causes a cerebral hemorrhage. The history is most important. In all cases a careful inspection of the scalp and skull should be made in order to detect contusion or fracture.

Hysteria may possibly simulate apoplexy, but only superficially. The unconsciousness is usually not profound; the patient is often open to suggestion; there may be characteristic stigmata; the pupils respond freely to light; and deep ovarian pressure usually brings some response.

The differential diagnosis between cerebral hemorrhage and embolism is difficult and may be impossible. At most there are suggestions, not positive grounds, for an opinion. Hemorrhage usually occurs in persons at and beyond middle life; embolism in persons at any age in whom there has been a vegetative endocarditis. Hemorrhage is rather more prone to cause loss of consciousness than is an embolus. Associated cardiac and renal disease, being productive of diseased blood-vessels, may cause hemorrhage. The same is true of syphilis, although syphilitic hemiplegia is more frequently caused by meningitis and endarteritis at the base of the brain.

VI. CEREBRAL SOFTENING.

Softening may result from any process that obstructs a blood-vessel; hence an endarteritis, causing thickening of an artery, as in syphilis; a thrombus from disease of the arterial walls, as in atheroma; and finally, an embolus, from the vegetations of endocarditis—all these may cause cerebral softening.

Pathology.—Thrombus and embolus produce results so nearly identical that they cannot be distinguished clinically. The special importance of embolus, from the clinical standpoint, lies in the fact that it may occur in young persons and produce all the symptoms of an apoplectic or hemiplegic stroke. It occurs in vegetative endocarditis. It is also common in malignant endocarditis, and sometimes occurs in other forms of sepsis. Thus hemiplegia may happen in the puerperium. Thrombosis occurs in some blood states other than sepsis, such as anæmia and chlorosis; it is most common, however, in disease of the coats of the blood-vessels, as atheroma and syphilis. The vessels most involved are those that form the circle of Willis or some of their branches, especially the middle cerebral artery. Softening of the cerebellum and pons is occasionally seen.

The area involved does not always break down at once; in fact a comparatively long time may elapse before it softens. This leads to deceptive appearances at autopsies. When the tissue has once become soft it may be quite diffluent, and in color may be white, yellow, or red, according to the amount of blood elements contained in it.

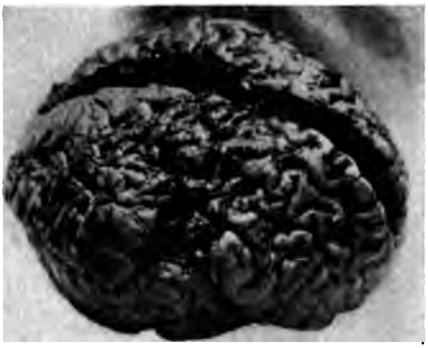

FIG. 368.—Softening of the brain in the motor area of the right hemisphere, due to embolus.—Lloyd.

Symptoms.—These are similar to those caused by hemorrhage. Hemiplegia is the commonest result, but other paralyses, such as monoplegia, hemianæsthesia, and hemianopsia, occur, as in hemorrhage, according to the seat of the lesion. Aphasia is not uncommon when the softening occurs in the lenticular nucleus or the cortical speech centres or in their subcortical connecting tracts in the left hemisphere. Apoplectic symptoms, with confusion, stupor, or loss of consciousness, may be present if a large area is involved, but they are rather more uncommon than in hemorrhage. Occasionally the onset of symptoms is gradual and the course progressive, especially if small successive thrombi occur, but usually in the case of embolus the onset is sudden. Prodromes also occur in case of atheroma, such as vertigo, headache, and failure of memory and other mental powers. In some cases we see transient hemiplegia and other paralyses, due doubtless to the fact that the circulation, after being obstructed, may be restored. But in most cases there is left some permanent loss, such as hemiplegia and aphasia. Thrombus of the carotid or of the basilar artery causes grave symptoms, such as profound unconsciousness and failure of respiration; and in the case of the basilar artery there may at first be staggering, ataxia, or even a cerebellar gait.

Diagnosis.—The distinction between hemorrhage and embolus largely depends on the presence of a lesion in the heart producing emboli, and this is more common in young persons. Hemorrhage due to atheroma is an affection of advanced life. Apoplectic symptoms, such as coma, are more common in hemorrhage; but the distinction between these two states is often problematical. Sinus thrombosis or syphilitic endarteritis may cause a hemiplegia. Syphilitic hemiplegia cannot always be recognized unless from the history or the Wassermann test. If there are headache, involvement of cranial nerves, especially the third, and an irregular mode of onset, the diagnosis is much more probable. In a young adult the absence of a cardiac lesion, such as could cause embolism, is further suggestive of syphilis as the cause of the hemiplegia.

VII. THE CEREBRAL PALSIES OF CHILDREN.

Children are sometimes the victims of hemiplegia, diplegia, paraplegia, and speech defects, due to affections of the brain. As these conditions present some special features they demand special notice apart from similar affections in adults.

Pathology.—Cerebral hemorrhage is rare in children, unless in the case of accident or trauma, especially at birth. These birth palsies are usually due to meningeal hemorrhage, caused by a general asphyxia, which in its turn is caused by prolonged pressure on the placenta. This is indicated by the fact that hemorrhage in the new-born is sometimes observed beneath other serous membranes, as, for instance, the capsule of the liver. Hence intracranial hemorrhage is not necessarily caused by the forceps, although this may be a factor in some cases. Hemorrhage may also be caused by the paroxysms of whooping-cough. A cerebral sclerosis occurs in young children; and Strümpell suggested that there may be also a polioencephalitis. Vascular lesions, as periarteritis and embolism, are doubtless the causes of extensive destructive changes in the brains of children, following upon the infectious diseases. It is not to be forgotten that hereditary, or even acquired, syphilis may cause cerebral palsies in children. Thrombosis of the cerebral veins may cause juvenile hemiplegia and diplegia. It occurs occasionally in typhoid fever, measles, etc. Thrombosis of the cranial sinuses is also occasionally observed. Hemorrhage is sometimes present beneath the membranes of the spinal cord.

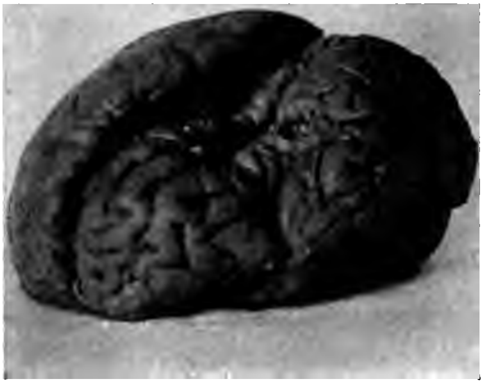
FIG. 369.—Porencephalus.—Lloyd.

A destructive lesion may result in the formation of a cavity in the cerebrum—the so-called porencephalus.

Symptoms.—The commonest forms of these palsies in children are hemiplegia, diplegia, and paraplegia.

The onset of the affection may be insidious, or at least not promptly recognized, as in very young children, and especially in the birth cases. When the onset is acute the affection may be ushered in with convulsions and coma, but this is by no means a universal rule. The "stroke" caused by embolus is sudden, just as in adults. Among other and minor symptoms are slight fever, vertigo, and vomiting. Confusion and delirium are sometimes seen.

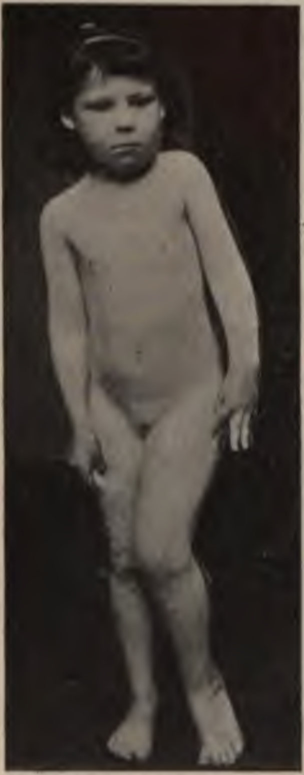

FIG. 370.—Old infantile hemiplegia.

HEMIPLEGIA is the most common form. At first the paralysis is flaccid, but in a later stage contractures set in and the paralyzed limbs are much hampered and even deformed. They do not grow quite normally, but true muscular atrophy, as seen in spinal cases, is not present. The deep reflexes are exaggerated. In some cases athetosis is present. If the lesion is in the left cerebral hemisphere grave speech defects are present, but these differ somewhat from genuine aphasia, because if the lesion comes on in very early life before the child has learned to talk, the speech is undeveloped rather than impaired. The face in old standing cases is usually not paralyzed, and the arm is more paralyzed and contractured than the leg. As a rule there is no hemianæsthesia. The limbs may be cold and blue, but the reactions of degeneration are not present, and fibrillation is not seen. The gait is typically hemiplegic, and the arm is usually carried flexed and contractured at both the elbow and wrist. The bladder and bowel are not paralyzed.

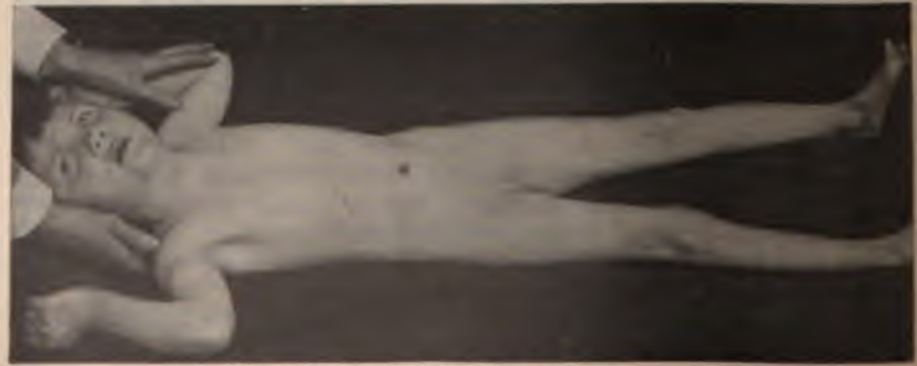

FIG. 371.—Congenital diplegia; early stage.—Young.

DIPLEGIA is simply a double hemiplegia, and is sometimes called bilateral spastic hemiplegia. The spasticity of the limbs is especially noticeable, not because the contractures are worse than in hemiplegia, but because, being on both sides, they give the patient a characteristic aspect. In one respect, however, the contractures in diplegia appear to differ from those of hemiplegia—they are somewhat more marked in the lower limbs. This gives the child a characteristic gait, if he is still able to walk. The limbs are usually adducted and extended, and the feet may be crossed and held in the position of equino-varus. The deep reflexes are exaggerated; sensation is unimpaired; and the upper limbs share in the rigidity. In these cases of double hemiplegia there may be imbecility or idiocy. Epilepsy and athetosis may complicate the case; speech defects are common; and strabismus and nystagmus are sometimes seen. The condition depends on a lesion which involves both hemispheres, and the destruction of brain tissue is sometimes great.

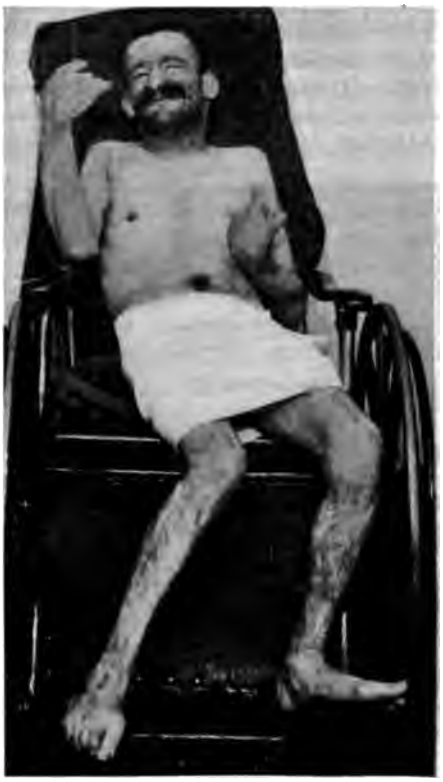

Fig. 372.—Spastic diplegia; athetosis.—Lloyd.

Paraplegia of cerebral origin has been described only in recent years, and its pathology is still a matter of some obscurity. Some authors claim that it depends upon a limited brain lesion, in which the leg areas alone are involved, while others attribute it to a primary lateral sclerosis. It is practically identical with the spastic paralysis of the legs as seen in diplegia, but the arms are not involved. There may be epilepsy, athetosis, and idiocy, just as in the other forms of cerebral palsy in children. Paralysis of the bladder and bowel is not a necessary part of the symptom-complex, as in spinal paraplegia, but incontinence may result from the mental defects.

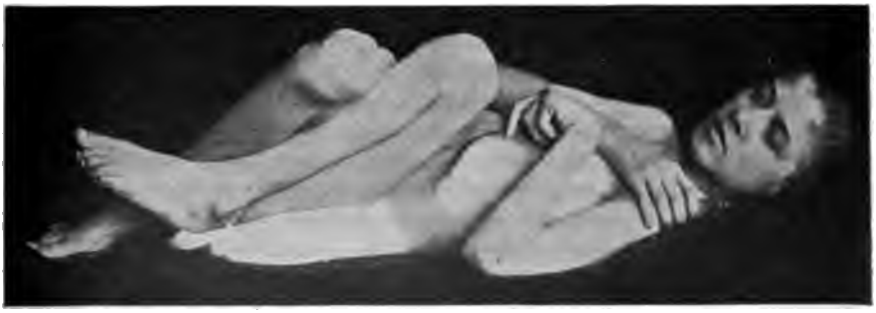

Fig. 373.—Spastic diplegia; epilepsy; idiocy. Lloyd.

Monoplegia, either brachial or crural, is a rare form of cerebral palsy in children. In this form one arm or one leg alone is involved.

In this connection brief mention may be made of *Little's disease.* The affection is in fact a form of cerebral palsy, in which the motor-conducting paths from the brain are injured, diseased, or undeveloped. Hence its clinical form is that of a diplegia or a paraplegia, according to the extent of the injury. It occurs especially in children who are prematurely born. Some writers seem inclined to limit the term to mild cases in which the cerebral faculties are not much involved, convulsions are absent, and the tendency to improvement is marked, but there seems to be no good reason for retaining it as a designation for a distinct disease.[1]

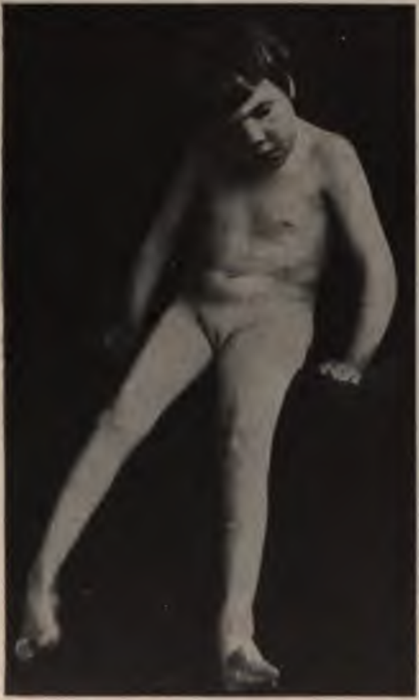

Fig. 374.—Attitude in cerebral palsy; paraplegic type.—Young.

Sachs has described a condition which he calls *amaurotic family idiocy,* in which the child, soon after birth, becomes weak and lethargic; blindness, due to degenerative changes in the optic nerves, ensues; and spastic paralysis with increased tendon reflexes may be added. In some cases nystagmus, strabismus, and deafness are noted. There are no convulsions, but there is well-marked idiocy; and death occurs in early childhood. Several cases have been observed in one family. The condition is one of failure of development (agenesis) of the nerve-centres; its causation is obscure.

Diagnosis.—The diagnosis is, as a rule, not difficult. The spastic paralysis, usually hemiplegic or diplegic in type, with exaggerated reflexes, absence of muscular atrophy and electrical changes, and the associated mental defects, often with athetosis and epilepsy, distinguish these cases clearly from diseases of the spinal cord, especially anterior poliomyelitis. Gross lesion of the brain, such as tumor, might simulate these cases, but the history and course are different, and optic neuritis is often present in the case of tumor.

Paraplegia of cerebral origin may simulate a spinal paraplegia, but in the latter there are no true cerebral symptoms, such as idiocy and epilepsy, and the bladder and bowel are almost always paralyzed. In case the lumbar cord is involved the paralysis is flaccid, with atrophy, lost knee-jerks, and even the reactions of degeneration. The history and course are also different. Infantile paralysis, due to acute anterior poliomyelitis, is usually confined to one limb; muscular atrophy occurs with lost knee-jerk and reactions of degeneration, and there are no cerebral symptoms.

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[1] For an excellent account of Little's disease see Brissaud's Leçons sur les Maladies Nerveuses Paris, 1895, p. 108.

Obstetrical paralysis, especially of the brachial plexus, could hardly be confounded with a cerebral palsy. The paralysis is flaccid, the muscles waste, the deep reflexes are lost, electrical changes are present, and cerebral symptoms are wanting.

The spastic rigidity of rickets and of tetany is distinguished by the associated symptoms of those diseases, the history, and the etiology.

VIII. HYDROCEPHALUS.

Pathology.—The lateral ventricles are enormously distended; the ependyma thickened; and the foramen of Monro or the aqueduct of Sylvius, or both, are possibly occluded. The brain may be so stretched as to be little more than a shell, the convolutions thin and flattened, and the sulci almost obliterated. The basal ganglia, the mid-brain, pons, medulla oblongata, and cerebellum are sometimes compressed and only partly developed. The choroid plexus may be thickened and congested. The bones of the skull are thin and translucent, and usually the sutures and fontanelles are widely distended, the former as much even as an inch. The essential elements, or neurons, of the cortex suffer greatly, and the optic tracts and cranial nerves may be degenerated.

Etiology. — The causation is obscure. Some observers attribute the disease to occlusion of one or other of the natural foramina, such as the foramen of Monro, the aqueduct of Sylvius, or the foramen of Magendie. Not enough attention has been paid to the state of the choroid plexus and veins of Galen. Recently much has been written by Lees, Barlow, and others about a posterior basic meningitis, which causes occlusion of the foramen of Magendie, with consequent distention of the ventricles.

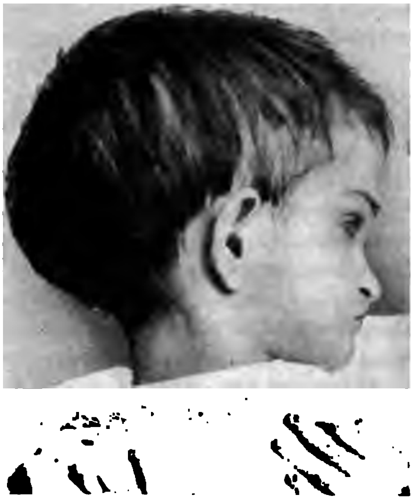

Fig. 375.—Hydrocephalus.—Lloyd.

Symptoms. — Besides the distention of the skull there are seen various defects of development of the brain and nervous system. There may be mental impairment, ranging from slight imbecility to complete idiocy. In rare cases, however, there is preserved quite a remarkable mental integrity.

The motor symptoms are often prominent; there is hemiplegia, diplegia, or monoplegia; the muscles are usually spastic, even contractured, and the deep reflexes are exaggerated. The eyes may be deflected downward, and there may be various forms of oculomotor palsy, with nystagmus. Other symptoms more or less common are convulsions, pain, as shown by the "hydrocephalic cry" (but this is more common in acute tuberculous meningitis), blindness, and incontinence. In extreme cases the child is bed-ridden from inability to hold up the enormously distended head. Occasionally hydrocephalus is associated with spina bifida.

Diagnosis.—This presents no difficulty in the advanced cases; the child's appearance is enough. In early stages, however, the diagnosis must rest on the child's evident failure properly to develop, and on the gradual enlargement of the head. Hydrocephalus sometimes begins before birth, and the skull may be greatly distended, causing grave dystocia.

IX. INTRACRANIAL ANEURISMS.

The larger aneurisms which develop on the main intracranial arteries, particularly at the base, will be discussed. The arteries usually involved are those that form the circle of Willis, and their branches, especially the middle cerebral. The internal carotid, cerebellar, and basilar arteries also are sometimes affected.

Pathology.—Diseases affecting the coats of the arteries, especially atheroma and syphilis, are the determining causes. Trauma also acts in this way. Emboli from vegetating endocarditis may be a cause. The aneurism is either fusiform or sacculated; it is likely to increase rapidly, and it eventually bursts with fatal effect.

Symptoms.—The aneurism acts by compression like a brain tumor. Some authors describe a thrill or murmur audible on the skull or over the great vessels. The patient sometimes has a subjective sense of pulsation. Headache, vertigo, vomiting, and affections of consciousness occur. In some cases the aneurism is latent until rupture occurs, while in other cases the symptoms are paroxysmal, from successive small bleedings. Aneurism of the internal carotid may compress the optic nerve or optic tract, the nerves of the eyeball, and the first division of the fifth nerve, and as it increases may even cause hemiplegia, and, if it is on the left side, aphasia. Starr observed a case of left third nerve paralysis with right hemiplegia. The optic chiasm may be compressed by aneurism of the carotid and especially of the anterior communicating artery. Various affections of the retinal and visual fields thus result, and bilateral temporal hemianopsia has been reported. Aneurism of the anterior cerebral causes symptoms similar to those of the preceding, except when it is far to the front, when it may involve no cranial nerves and cause only obscure compression symptoms in the frontal lobes. Aneurism of the middle cerebral, especially if well within the Sylvian fissure, causes hemiplegia; possibly hemianæsthesia and hemianopsia; and, on the left side, aphasia. The third nerve may be compressed. There may be loss of smell on the affected side in the case of aneurism of any one of these arteries near the olfactory nerve. Aneurism of the posterior communicating artery may involve the optic tract and the third and sixth nerves; and if it grow very large, it may compress the peduncle, causing hemiplegia. Aneurism of the posterior cerebral may compress the peduncle and the third and sixth nerves, causing hemiplegia alternans; and if it should involve the occipital lobes it might cause various affections of the visual fields. Aneurisms of the basilar and vertebral arteries cause pontile and bulbar symptoms. [illegible] striking is the compression bulbar palsy sometimes seen. If the pons alone is involved there may or may not be unilateral symptoms, such as hemiplegia alternans—paralysis of the sixth and seventh nerves on one side with opposite hemiplegia. If the medulla oblongata is involved the bulbar symptoms are dysarthria, dysphagia, paralysis of the tongue and lips, and sometimes respiratory symptoms, especially when the head is thrown forward.

Diagnosis.—Intracranial aneurism simulates a brain tumor, and the distinction between it and a neoplasm cannot always be made. A murmur, having the cardiac rhythm controlled by pressure on the carotid, is suggestive of aneurism, but even this sign is not reliable, for such a murmur has been heard in other lesions. Starr claims to have heard a loud double murmur over the Sylvian region in a case of extensive softening. Murmurs in the head have also been heard in cases of tumor (when the growth is near a large artery), in anæmia, in hydrocephalus, in exophthalmic goitre, and in several cases of loud endocardial murmurs, which I have observed. There are therefore no positive rules for diagnosis. The history and course may be the same in aneurism as in tumor of the brain. Mills calls attention to pulsating exophthalmus as a sign of aneurism of the internal carotid.

X. TUMORS OF THE BRAIN.

Under this term are included all new growths within the cranium, whether within the brain, in the membranes, or springing from the bones of the skull. These tumors are comparatively rare, but in any large neurological clinic several of them are likely to be seen in the course of a year.

Pathology.—Intracranial tumors are of various kinds. In a series of 100 cases, analyzed by Mills and Lloyd, 16 were gliomata, 15 sarcomata, 13 gummata, 13 tuberculous, 7 carcinomata, 16 unclassified, and the remainder of various forms. An area of congestion, inflammation, softening, or hemorrhage is sometimes seen about the tumor; more rarely suppuration. The cerebrospinal fluid may be increased, and in some cases the ventricles are distended, this depending on the seat of the neoplasm. The tumor may be encapsulated, especially if it be a meningeal growth, in which case it is sometimes easily shelled out. If the tumor is within the substance of the brain, as, for instance, in one of the cerebral hemispheres, this may appear swollen, and even slightly flattened and discolored from pressure. Occasionally the cranial nerves are pressed upon or stretched over the surface of tumors at the base. In some cases the new growth closely resembles brain tissue. Tumors of the brain are sometimes multiple, and in the case of carcinomata they may be metastatic.

Symptoms.—The onset of a tumor is usually insidious, and the course gradual and even slow. The symptoms are general and local.

The *general* symptoms are such as are common to all kinds of brain tumors, and indicate in a more or less distinctive way the presence of an intracranial lesion. These symptoms are headache, vertigo, vomiting, convulsions, paralysis, ataxia, sensory changes, optic neuritis, and affections of consciousness. Headache is a very common symptom of brain tumor. In the early stages it may be slight, increasing later to great intensity. Sometimes it is paroxysmal; sometimes, but rarely, it is absent (in 5 out of the 100 tabulated cases); occasionally it is localized. It is not easily controlled by drugs. Vertigo, with which we may include affections of equilibration, forced movements, and ataxia, is seen in many cases. Some of these symptoms, such as affections of equilibration and forced movements, are highly characteristic of cerebellar tumor; but vertigo is not confined to subtentorial growths. Vomiting is usually of the propulsive kind, irrespective of food in the stomach; in other words, of the type known as cerebral. It is by no means constant, but when associated with other general symptoms it is highly suggestive. It may be unaccompanied with nausea. Convulsions occur in many cases; they are either general or focal. The former are seen in practically all kinds of cases; the latter are indicative, as a rule, of irritation of the motor centres in the cortex. By focal convulsion we mean one commencing in or confined to one or a few groups of muscles. Paralysis in some form is usually present, and it may or may not be an early symptom. It is more appropriately discussed among the local symptoms, as may also be said of the sensory changes. Optic neuritis is a frequent symptom, but it occurs also in other conditions, especially in brain abscess, purulent encephalitis, and brain syphilis. In brain tumor it occurs in at least 80 per cent. according to Gowers, Bramwell, and others. From another view-point Oppenheim claims that of all cases of choked disk 90 per cent. are due to tumor of the brain. Affections of consciousness range all the way from slight stupor to profound coma. The emotions may be affected, and hysterical symptoms are sometimes seen.

The *local* symptoms are such as indicate the site of the tumor. The most important of these symptoms are the various forms of paralysis. Thus hemiplegia indicates that either the motor cortex or the descending motor tracts are involved. A monoplegia, as of the leg, arm, or face, is especially indicative of a lesion of the respective centre in the pre-Rolandic area. Aphasia points to involvement of the left cerebrum, in one or other speech centre, according to the type of the aphasia. Paralysis of the various cranial nerves, especially the third, fourth, fifth, sixth, seventh, eighth, and twelfth, is often of definite localizing value, as will be shown presently. An ataxic form of paralysis is sometimes seen in lesions of the superior parietal lobule. Affections of sensation may take the form of hemianæsthesia, hemianopsia, astereognosis, or localized anæsthesia, and will be considered with the focal diagnosis. Focal convulsions, of the type known as Jacksonian epilepsy, indicate usually a lesion in the respective motor centres. When associated with focal paralysis they are especially typical of a focal lesion in the motor area. It must be borne in mind that a convulsion may have a focal type at the beginning, passing later into a general convulsion; in such cases a focal origin of the convulsion is usually indicated, hence such fits have diagnostic value.

The *focal diagnosis* for tumors at various sites in the brain is briefly indicated as follows:

Tumors of the frontal lobe may give few if any localizing symptoms unless they involve the motor area. The mental changes are sometimes characteristic, and may consist of alterations of character, lack of power of attention, and especially retarded cerebration. If the motor area is involved, a tumor in the upper part of the pre-central gyrus, or on the mesial aspect of that region, causes paralysis of the opposite leg; in the middle part of the gyrus, paralysis of the arm; and in the lower part, paralysis of the face. On the left side a tumor involving the posterior part of the third frontal convolution causes motor aphasia.

A

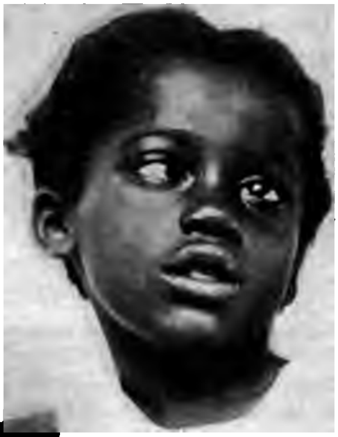

FIG. 376A.—Paralysis of the sixth nerve of both sides and of the right seventh nerve, in a case of pontile tumor.—Lloyd.

B

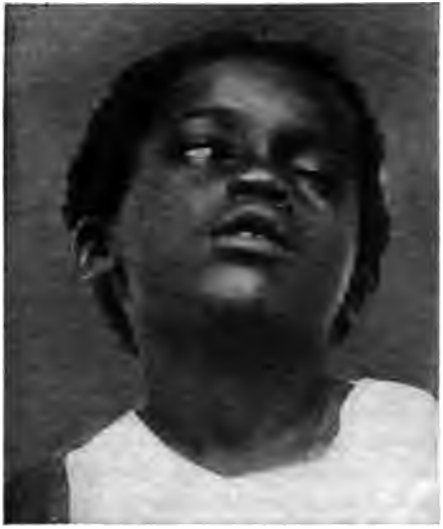

FIG. 376B.—Paralysis of the seventh nerve, right side, and of both sixth nerves. Case of pontile tumor; patient attempting to close her eyes.—Lloyd.

Tumors of the parietal lobe may also cause focal paralysis and convulsions by pressure on the pre-central gyrus; and if located on the left side they may cause motor aphasia for the same reason. Affections of the superior parietal lobule cause astereognosis and ataxic paralysis of the opposite leg or arm or both. If the posterior part of the internal capsule is involved there is hemianæsthesia, and possibly hemianopsia, on the opposite side. If the angular gyrus is involved there may be visual aphasia and word-blindness. Tumors of the occipital lobe cause hemianopsia, possibly also hemianæsthesia; and also visual aphasia, word-blindness, and object-blindness. Tumors of the temporal lobe may cause hemiplegia and hemianæsthesia by pressure on the internal capsule, if they are large enough, but the most typical symptom, if the tumor is on the left side and involves the first two temporal gyri, is word-deafness and auditory aphasia. Tumors of the mid-brain often cause a hemiplegia alternans, in which the hemiplegia is on the opposite side, while paralysis of the oculomotor nerve is on the side of the lesion—the so-called "syndrome of Weber." Tumors of the pons may also cause a hemiplegia alternans, in which with an opposite hemiplegia there is paralysis of the sixth and seventh nerves, possibly also of the fifth and eighth nerves, on the side of the tumor. Sometimes both sixth nerves are involved. These cranial nerve paralyses vary somewhat according to the site of the growth. Thus if the tumor is located high in the pons, above the decussation of the motor paths for these cranial nerves, the hemiplegia and the paralysis of the nerves are both on the opposite side. A favorite site for these tumors is in the cerebellopontile angle.

Tumors of the cerebellum give a wide variety of symptoms, the most characteristic being loss of equilibration, cerebellar ataxia (in tumor of the vermis), and forced movements (in tumor of the peduncles, especially the middle peduncle). If the tumor presses upon the mid-brain, pons, or medulla oblongata, there may be characteristic paralysis of the several cranial nerves which have their nuclei in those structures, just as in tumors of those parts, and even hemiplegia. The knee-jerks may be lost, or may even go and come, but this symptom is not always seen. Tumors of the basal ganglia cause hemiplegia, hemianæsthesia, and hemianopsia, and on the left side aphasia or paraphasia. Tumors of the membranes at the base of the brain, according to their location, cause paralysis of the various cranial nerves, especially the third, fourth, fifth, and sixth. Lloyd reported a case of total unilateral ophthalmoplegia, with anæsthesia of the first division of the fifth nerve, caused by a syphiloma just behind the orbit. Tumors of the medulla oblongata are rare; they cause paralysis of the twelfth nerve and of the motor tracts, and difficulty in swallowing and in respiration. In some cases brain tumor is latent, especially if it occupy a so-called *silent* region of the brain.

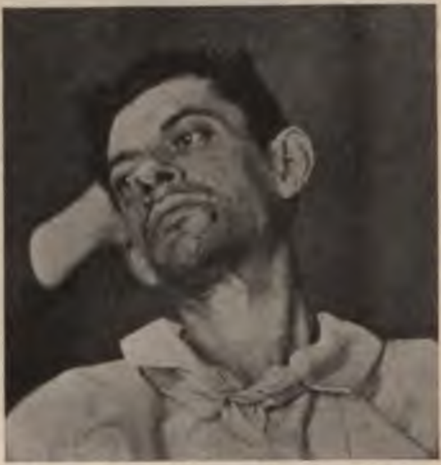

FIG. 377.—Tumor of the cerebellum, showing forced movement to one side.—Lloyd.

Diagnosis.—The above brief sketch sets forth the principles of local diagnosis; but brain tumors cause a wide variety of symptoms, both general and local, and these must be interpreted with care. A successful local diagnosis is frequently possible, and is often made.

Tumor may simulate abscess, but the history and course are usually different; there is no history of a precedent focus of suppuration, there is no evidence of sepsis, the evolution is more gradual, and as a rule the duration is longer. Hemorrhage is not likely to be mistaken for tumor; the onset is entirely different. It is sudden, the symptoms are established quickly, and the case is not progressive. Yet tumor in the motor region, causing hemiplegia, has been mistaken for a long-standing paralytic "stroke" due to hemorrhage. The history, the presence of optic neuritis and of headache in the case of tumor should prevent error. Syphilis of the brain may possibly simulate brain tumor, especially if the headache and optic neuritis are associated with focal symptoms, but the symptoms of syphilis often pursue an irregular course, quite unlike the steady progress of a tumor. Nevertheless, syphilis may cause a gummatous tumor.

In truth, tumor of the brain does not closely simulate any other lesion; the onset, the course, the duration, and the grouping of symptoms are all sufficient as a rule to prevent error. It is well, however, in this connection not to overlook those curious cases in which hysterical symptoms have masked the symptoms of brain tumor and led to error.

XI. PARASITES IN THE BRAIN.

The commonest is the Cysticercus cellulosæ, the larval form of the Tænia solium, or pork tape-worm. Occasionally the echinococcus, or hydatid, is observed.

Pathology.—The parasites exist as cysts, of the size of a millet-seed to that of a grape or even a walnut. They may be found in the substance of the brain, or beneath the membranes, or floating free in the ventricles. The last is the most common. There is usually an ependymitis and great increase of the ventricular fluid. The parasites are found in both the lateral and the fourth ventricle.

Symptoms.—The nature of the disorder may be obscure. In a case observed by Lloyd [1] in the Philadelphia Hospital the earliest symptoms were apoplectiform attacks, followed by severe headache, hemiparesis, ataxia, disturbance of equilibration, exaggerated knee-jerks, incontinence of urine and fæces, drowsiness, loss of power of attention, speech defects, trismus, and failing vision. There were no convulsions nor optic neuritis. Death occurred in coma. At the autopsy eighteen cysts, some as large as a chestnut, clear and satin-like, were found floating in the right ventricle, which was enormously distended. One small cyst was found adherent in the fourth ventricle. There was ependymitis, and the aqueduct of Sylvius was occluded.

The patient in Lloyd's case insisted that when he moved his head he could feel something rolling within it.

The symptoms are not the same in all cases; much depends upon the number, size, and location of the cysts. Convulsions are not uncommon, and affections of consciousness are frequent. Headache is a constant symptom, and choked disk is sometimes observed. A cysticercus has been seen in the eye.[2] On the whole, the symptoms are irregular.

Diagnosis.—The diagnosis is most difficult, and the chances are that the true nature of the case will not be discovered until the autopsy. This is especially so in America, where the infection is rare.

Because of the irregularity of the symptoms, with headache, drowsiness, convulsions, and various forms of paralysis, the case may easily be mistaken for one of syphilis of the brain; also for brain tumor. There are no pathognomonic signs, and in any case the diagnosis must be problematical. The subjective sense of an object rolling in the head is the most distinctive sign that we have seen noted. It is quite impossible, as a rule, to trace the origin of the infection in the character of the patient's diet. There may be no tape-worm in the bowel, for autoinfection is not common, the eggs being usually introduced from without. If cysts are found under the retina or skin or in the muscles, the presence of similar growths in the brain is rendered highly probable.

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[1] Philadelphia Med. Journal, March 19, 1898.

[2] Oliver, Ophthalmoscopy, in Keating's Cyclopædia of the Diseases of Children, vol. iv, p. 238.

XII. SYPHILIS OF THE BRAIN.

Syphilis in the spinal cord or brain begins its work, as a rule, upon the arteries. The inner and sometimes the outer coat of the vessel becomes infiltrated and thickened, as Heubner has pointed out. The inflammation then spreads to the meninges, and there results an exudative meningitis, which still further involves the blood-vessels and even invades the brain tissue and the cranial nerves.

Pathology.—There may be syphilis of the convexity, or of the base, or of both. At the base we often see the most typical picture of thickened membranes and gummatous infiltration. There is usually an associated cerebritis, especially near the surface, and deeper in there may be softening or hemorrhage from obstruction of the arteries or from rupture of their diseased walls. A common seat for this form of meningitis is between the cerebral peduncles and at the anterior perforated space, where the arteries run up to supply the interior of the brain, especially the lenticular nucleus and internal capsule. The basilar and vertebral arteries may also be involved. On the convexity there is seen sometimes a leptomeningitis or a pachymeningitis, or both. Occasionally the gummatous new growth forms a veritable tumor. It is thus understood that the syphilitic process causes damage to the brain by pressure, by inflammation, by sclerosis, by softening and hemorrhage, and by involving the cranial nerves. It also acts by elaborating secondary products—toxins.

Symptoms.—These may be exceedingly irregular, sometimes advancing quickly and then receding, or remaining for a long time stationary. From the character of the lesions it is evident that the symptoms may be multiform and show the invasion of many different parts of the brain.

Of general symptoms headache is common; it may be especially severe at night, causing insomnia. There may be vertigo and vomiting. Psychical changes are observed. There are drowsiness, stupor, confusion, dementia, even delirium and maniacal excitement. Convulsions either general or focal may occur in brain syphilis. The focal convulsions are usually indicative of a lesion at some part of the motor area. When the motor area is invaded, as by a small patch of infiltration or sclerosis, there is focal paralysis as well as focal epilepsy. The cranial nerves at the base of the brain are often involved. Optic neuritis is seen sometimes as an early symptom. Primary optic atrophy is not so [illegible]. The [illegible] is peculiarly exposed in the [illegible] of the chiasm; next to it in frequency [illegible] the [illegible] saying that syphilis [illegible] because of the involvement [illegible] results, especially in the [illegible] hemiplegia, hemianæsthesia [illegible]

[illegible] as shown by ophthalmoplegia [illegible] even [illegible] the third nerve is [illegible] the [illegible] the opposite side. [illegible] bulbar [illegible] artery is affected. [illegible] the sixth [illegible] with hemiplegia on the [illegible] side. Combined facial and trigeminal paralysis due to pontile syphilis has been noted. An apoplectic bulbar palsy, due to arterial disease, sometimes specific, has been described.

Syphilitic tumors occur in various regions of the brain, and act like other tumors.

The symptoms of brain syphilis are often controlled by specific treatment, especially in the early stages, before destruction of tissue has occurred.

In some cases there are associated spinal symptoms—cerebrospinal syphilis.

Diagnosis.—The mode of onset and progress may suggest syphilis. The symptoms are often insidious, advancing irregularly, receding, then again advancing. This is seen in syphilitic hemiplegia. There may be slight apoplectiform attacks, then slight hemiparesis, then improvement, then a more grave attack, and so on. This can readily be understood from the nature of the syphilitic meningitis and endarteritis, interfering with circulation. The third nerve palsy is also sometimes irregular; only one or two branches may be involved at first, then later the whole nerve, due to the gradual involvement of the several roots of this nerve in the interpeduncular space. The history of the case is often clear, but sometimes it is not reliable. The multiformity of symptoms is often characteristic, and the presence of spinal lesions is generally conclusive. It may be difficult to distinguish syphilis in some cases from brain tumor, and often impossible to say whether a tumor is syphilitic or not. Brain tumors, as a rule, are more focal and constant in their symptomatology, and more regularly progressive. The Wassermann test should always be made. Tuberculous meningitis has a more rapid course than syphilitic meningitis; it is not amenable to drugs, and is uniformly fatal. There is also fever and slowing of the pulse, and the disease is commonly seen in children, whereas the syphilitic form is more common in adults. A febrile reaction is sometimes, though rarely, seen in brain syphilis. The hemiplegia of syphilis cannot always be easily distinguished from hemiplegia due to other causes. If there is a clear history of syphilis, no endocardial lesion to cause embolism, and the accident occurs in a young adult; and if, especially, there be headache, involvement of one or other cranial nerve, particularly the third, and the symptoms are of rather irregular onset and course, the evidence is in favor of syphilis. The therapeutic test may, or may not, be of value.

XIII. GENERAL PARESIS.

General paresis is a degenerative disease of the brain cortex, medullary tracts, and nerves. Syphilis and over-strain, especially business worry, are probably active but not exclusive causes. The disease occurs more frequently in men than in women; and in the white than in the colored races. The French attach much importance to alcoholism. It is more common in civilized countries and among the better classes, but Krafft-Ebing's statement that it is always due to "civilization and syphilization" is probably more resonant than correct.

Pathology.—There is a destructive process in the tissues of the brain. This process shows itself in the small vessels of the membranes, leading to congestion, obstruction of the circulation and the lymph stream, morbid increase of the neuroglia, hardening and atrophy of the cortex, thickening of the membranes, minute hemorrhages, extravasation of the cerebrospinal fluid, distention of the ventricles, and impaired nutrition and destruction of the neurons in the brain cortex.[1]

Symptoms.—General paresis is usually divided into four stages.

In the first stage the prodromes appear, such as change of character, spells of irritability or even of depression, inattention to business, erratic conduct, and moral lapses. Insomnia, or its opposite, somnolence, may occur. Alcoholic and sexual excesses are common.

In the second stage the period of grandiose delusions sets in. The patient has most extravagant ideas of his wealth, of his personal importance, even of his sexual prowess. He becomes talkative, boastful, and slightly demented. Along with these mental changes appear physical changes. There is tremor of the muscles of the face, tongue, hands, and limbs; speech becomes drawling, stammering, or staccato; the handwriting is unsteady, and words are dropped or run together just as in speech. The gait is weakened and incoördinate, with increased knee-jerks as a rule. There may be myosis, or irregularity and inequality of the pupils, and sometimes the Argyll-Robertson symptom, in which the light reflex is lost while the movement on accommodation remains. There may also be optic atrophy.

In the third stage the disease assumes a more chronic aspect, and dementia is more advanced. In this stage especially we see crises of epilepsy, apoplexy, hemiplegia, or maniacal excitement. The paralytic symptoms increase.

In the fourth stage the patient is in terminal dementia, paralyzed, bedridden, almost or quite speechless, with incontinence of urine and fæces—a total wreck, until death ends the scene in exhaustion or in an epileptic or apoplectic crisis.

In rare cases arthropathies form, especially when the disease is associated with posterior sclerosis of the spinal cord.

In some cases remissions occur, of quite long duration, but the hopes raised by them are fallacious, for the disease is fatal.

Diagnosis.—This may present some difficulty in the early stages, as between this disease and disseminated syphilis of the brain. But in the latter there is not seen the characteristic psychosis, with grandiose delusions, and the disease does not present the regular evolution that is seen in general paresis. Still, the diagnosis is not always easy. The therapeutic test may help to solve the problem, but it must be borne in mind that general paretics do not bear well the heroic antisyphilitic drugging. Striking examples of cerebral lues, resembling the early stages of general paresis, sometimes make good recoveries under a judicious treatment. These are probably the kind of cases that are reported as cures of general paresis. The Argyll-Robertson pupil would point to the latter disease.

There is a pseudoparesis induced by alcohol and promoted by morphia, cocaine, etc., which closely resembl[illegible]e genuine disease, especially in the tremor and speech defects, but recovery occurs promptly on the withdrawal of the poison. The history in these cases is suggestive, and they do not present quite such a typical expansive psychosis as in general paresis; the mental condition is usually one of enfeeblement with hallucinatory delirium, which, however, may be rather grandiose.

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[1] Bevan Lewis, Mental D[illegible]dit[illegible]ley, Mental Diseases, p. 197. These two works give the best accounts o[illegible]l paresis.

The distinction between neurasthenia and the early stages of paresis is not difficult. The Wassermann test should be made.

Multiple sclerosis differs from general paresis in the history of the case, in the nystagmus and intention tremor, and in the absence of the expansive psychosis.

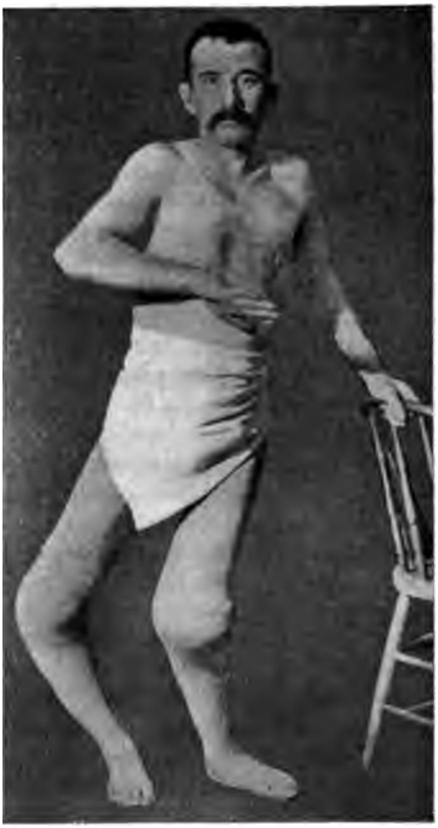

FIG. 378.—Arthropathy in general paresis.—Lloyd, Philadelphia Hospital Reports, vol. ii.

XIV. SENILE DEGENERATION.

The fundamental change in the central nervous system in old age is probably atheroma of the blood-vessels. It is a trite saying that a man's life is only as long as the life of his arteries. Some of the effects of this arterial degeneration have already been discussed in the chapters on cerebral hemorrhage and softening. There are other conditions, however, so identified with senility that they merit especial, though brief, attention. Whether or not they all depend on changes in the blood-vessels may still be an open question.

CEREBRAL SYMPTOMS.—The most conspicuous of these are undoubtedly the mental changes which occur in old age. There is loss of memory and failure of the mental powers so familiar to all. The various psychoses of the aged, such as senile melancholia, delusional insanity, maniacal episodes, and dementia, belong rather to psychiatry than to clinical medicine.

The early symptoms of atheroma of the cerebral arteries are not easily recognized. Among them are tinnitus, vertigo, throbbing or fulness in the head, transient attacks of mental confusion, slight paresis, aphasia, and headache. The throbbing or beating in the head is sometimes almost enough to suggest aneurism, especially when accompanied by headache. It may even depend in some cases on slight aneurismal dilatations. We must not overlook in these cases associated disorders in the heart and kidneys.

SENILE EPILEPSY.—The onset of epilepsy in old age is occasionally observed. It is doubtless helped on in some cases by alcoholism. The prime cause is probably degenerative change in the brain cortex. The epileptic seizure in senile patients is practically like that seen in ordinary epilepsy. It is necessary to exclude uræmia.

SENILE TREMOR.—In some old persons a very pronounced tremor is seen. It usually begins as a fine movement in the hands, most marked on exertion. Later it may spread, especially to the neck, causing a shaking of the head. In advanced stages it is even present during rest, but it disappears during sleep. It is not accompanied with the muscular rigidity and the characteristic attitude, expression, and gait of paralysis agitans, although intermediate cases are seen, and the two conditions have some points of contact.

SENILE PARAPLEGIA.—A spastic paresis of the legs, more rarely of the arms also, occurs in the aged. There may, or may not, be anæsthesia and weakness of the sphincters. The cases usually resemble primary lateral sclerosis, without loss of sensation or involvement of the bladder and bowel. This condition may depend on primary changes in the cord, especially in the lateral tracts, or it is possibly due to small foci of softening in the motor regions of the brain. These patients often have well-marked mental deterioration.

ALCOHOLISM AND DRUG HABITS IN THE AGED.—Evil habits creep on insidiously in some old people who may always have led strictly temperate lives. Bevan Lewis calls attention to the frightful impetus which the excessive use of alcohol lends to the retrograde changes which naturally occur in the brain in old age. The opium habit may be formed by the aged.

XV. ACUTE DELIRIUM.

This disease was first described by Luther Bell, an American alienist, and is sometimes called from him Bell's mania.[1] It is also called typhomania and *delirium grave.*

Pathology.—The disease is probably an acute infection, and may be caused by a variety of germs. Bacteriological studies have not led to uniform results. Berkley says that in a malady which may be caused by so many agents, a sole cause is not to be expected.

Symptoms.—The onset is usually abrupt and the course very rapid. Delirium sets in acutely and advances quickly to stupor and coma. The tongue becomes dry and brown, and sordes form. The pulse is rapid and compressible. There are muscular unsteadiness, tremor, and incoördination. The temperature rises but pursues no regular course. There is aversion to food, and the vital powers soon fail. Death may occur in a few days. Cases of longer duration are seen to follow the puerperium, and are probably due to sepsis; but in the typical cases no cause can be made out.

Diagnosis.—The diagnosis rests upon the abrupt onset, the rapid course, and the tendency to speedy death without obvious cause. Bacteriological studies should be made. Perhaps with our increasing knowledge of microbian pathology the nature of these cases will be made clear. The possibility of poisoning by alcohol, syphilis, lead, or malaria must not be ignored. Fulminating attacks of typhoid fever, measles, and scarlatina may simulate *delirium grave*, but can usually be recognized by the associated symptoms. Uræmia must also be excluded.

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[1] Bell described the disease at a meeting of the Association of Superintendents of American Asylums, in 1849.

XVI. MULTIPLE SCLEROSIS.

This disease, also called insular, or disseminated, sclerosis, is marked by foci of degeneration scattered through the brain and spinal cord.

Pathology.—The foci vary in size from that of a small bird-shot to that of a pea or a chestnut, and there may be even larger areas involved. They are different in color from, and harder in consistence than, the brain tissue. Histologically they consist of hardened connective tissue and infiltrated blood-vessels, with degenerated nerve-fibres, although many fibres are seen intact, penetrating the diseased tissue—a fact which is supposed to explain one of the chief symptoms, the intention tremor. The cause of multiple sclerosis is not known; it is not believed to be syphilis, although diffuse syphilitic lesions sometimes cause a state which clinically is not very unlike multiple sclerosis. The fact that the disease sometimes follows the infectious diseases, such as smallpox, typhoid fever, etc., does not explain its causation. The same may be said of its appearance in metal workers.

Symptoms.—The affection is one of early adult life; it rarely appears after the thirtieth year, and it is not uncommon in young women.

There are three symptoms which especially distinguish insular sclerosis—intention tremor, scanning speech, and nystagmus.

The intention tremor is an early symptom. As its name implies, it appears on voluntary motion; the arm, for instance, showing wide jerky tremors when the patient attempts to use it, as for carrying a glass of water to the lips. The motion is then so violent that often a large part of the water is spilled. The tremor is coarse, with wide amplitude and few vibrations to the second. While the patient is at rest, it is absent. The tremor extends to the face, tongue, and limbs, causing other symptoms, especially scanning speech and an unsteady gait.

The speech is usually scanning rather than staccato, although in all cases the words are uttered slowly, and sometimes with pauses between them. In a few rare cases bulbar symptoms have been seen, such as paralysis and wasting of the tongue. Pseudobulbar palsy has also been noted.

The nystagmus is usually a prominent symptom, and is most marked when the patient turns the eyeballs to one side — lateral nystagmus. Sometimes a rotary nystagmus is seen, in which the eyeballs are rolled on their axes. Even when the eyes are fixed straight ahead slight oscillations are sometimes seen.

The gait is usually spastic, and the deep reflexes are exaggerated in consequence of involvement of the lateral tracts in the insular foci at various levels. Abolition of the cremasteric reflex is claimed by Collins. The bladder and bowel are not paralyzed; if there are exceptions to this rule, they must be very rare.

In advanced stages the mental faculties may suffer, and crises of an apoplectiform kind may be seen. Optic atrophy is present in some cases.

Gowers claims that even when the optic nerve is involved in a patch of sclerosis many fibres pass through unharmed and a fair degree of vision is retained; but the visual fields are variously affected. One optic nerve may be more injured than the other. Uhthoff, who analyzed 100 cases of multiple sclerosis, found the optic nerves affected in 40. He also found paralysis in one or other of the ocular muscles in 17 of his cases. Involvement of the pupils was rare. Muscular atrophy sometimes occurs, and a slight ataxia.

Sensory symptoms are usually remarkable for their absence. Pain is sometimes felt, and various but slight modes of anæsthesia are sometimes present and are most likely to be found in the distal parts of the limbs.

The course of multiple sclerosis is chronic; remissions occur, and even slight improvement, but the disease is incurable.

Diagnosis.—The diagnosis is easily made from the association of the three cardinal symptoms. Nystagmus and scanning speech are seen in Friedreich's disease, but they are then associated with ataxia and lost knee-jerks, and the disease is usually a familial one. In disseminated syphilis the course is more rapid, the mind more involved, the evolution of symptoms is not characteristic, and scanning speech and nystagmus are not seen as a rule. General paresis shows scanning speech, tremor, and ocular changes, but the expansive psychosis, the history, the evolution, the more marked tremor of the facial muscles, all serve to distinguish it. The cremasteric reflex is likely to be preserved, but we do not insist here upon this sign as distinctive. The early diagnosis is uncertain.

Multiple sclerosis may be simulated by hysteria, in which, however, symptoms that can only be accounted for by organic lesions, such as nystagmus and optic atrophy, are never found. Cases of hysterical pseudosclerosis are usually of traumatic origin. The differential diagnosis is most important. Other hysterical stigmata are not always present. Somewhat similar cases follow exposure to mercury or lead.

XVII. DISEASES OF THE MID-BRAIN.

The mid-brain is composed in part of the cerebral peduncles, which contain the motor tracts from the cerebrum; its dorsal part consists of the corpora quadrigemina, and it is penetrated by the aqueduct of Sylvius, underneath which are located the nuclei of the third and fourth nerves. The sensory tract, or fillet, runs up just behind each peduncle.

Pathology.—Tumors are sometimes observed in this region, and more rarely hemorrhage and softening. Wernicke has described an acute destructive process located in the gray matter about the aqueduct, which he has named *superior polioencephalitis*. The meninges in the interpeduncular space are not infrequently the seat of syphilitic meningitis.

Symptoms.—Tumors of the mid-brain are usually unilateral, and cause hemiplegia alternans, in which there is an opposite hemiplegia, with or without hemianæsthesia, associated with paralysis of the third nerve on the side of the lesion. Other symptoms of tumor are usually present, such as optic neuritis, headache, vertigo, vomiting, changes in consciousness, and more rarely convulsions. A somewhat similar train of symptoms may be caused by cerebellar tumors, if these make pressure on the mid-brain; but in cerebellar tumors there is likely to be in addition some disorder of equilibration. A meningeal tumor in the interpeduncular space may paralyze both third nerves.

In Wernicke's acute superior polioencephalitis there is a destructive process in the mid-brain, and sometimes in the gray matter of the third ventricle. The floor of the aqueduct of Sylvius, hence the oculomotor nuclei, and even the peduncles, are involved. The symptoms are paralysis of the third and fourth nerves, nystagmus, optic neuritis, and rapid prostration, sometimes with ataxia, dysarthria, and even paralysis of the face and extremities. Death is common in from eight to fourteen days.

Hemorrhage and softening in the mid-brain are rare. The symptoms are those of a focal lesion, such as ophthalmoplegia of various kinds, according to the nuclei involved, hemiplegia alternans, etc.

Diagnosis.—This rests upon the grouping of symptoms as described above. The most characteristic is the hemiplegia alternans, in which the third nerve is paralyzed on the side of the lesion and the hemiplegia is on the opposite side. In Wernicke's disease the association of symptoms and the rapidly acute course, often with fatal ending, are characteristic. The disease is to be distinguished from bulbar palsy by the history and course and especially by the different cranial nerves involved. In the mid-brain lesion the eyes are paralyzed; in the bulbar lesion, the tongue and lips.

Interpeduncular syphilitic meningitis is distinguished by the headache, the third nerve palsy, the irregular course, the absence usually of hemiplegia, and the history. There may, however, be hemiplegia if either peduncle is softened by syphilitic endarteritis. Complete paralysis of both third nerves is not common; in fact, the third nerve palsy may change from time to time, and it is usually unilateral.

XVIII. NUCLEAR OPHTHALMOPLEGIA.

By this term is meant an affection in which the muscles of the eyeballs and upper lids are paralyzed by reason of disease of the nuclei of their motor nerves. The disease is often chronic and selective, for it picks out gradually the nuclei of the third and fourth nerves, which are in the mid-brain, and those of the sixth nerves, which are in the pons, some distance away. Consequently it is neither a purely mid-brain nor a purely pontile disease.

Hutchinson was one of the first to describe a pure nuclear ophthalmoplegia. Later Wernicke described an acute destructive process invading the floor of the aqueduct of Sylvius and neighboring parts in the mid-brain, which he called *superior polioencephalitis* to distinguish it from bulbar disease, which he called *inferior polioencephalitis;* but cases of this affection often present other than purely nuclear ophthalmoplegic symptoms, as, for instance, optic neuritis, nystagmus, facial paresis, dysarthria, ataxia, and even hemiplegia; in other words, it is not a purely nuclear disease. It is best to limit the description, therefore, to the nuclear disease, a sufficient number of cases of which have now been reported to entitle it to distinction as a substantive affection.

Pathology.—The disease process has some resemblance to the chronic or subacute forms of anterior poliomyelitis, inasmuch as the multipolar ganglion cells are gradually destroyed. From this form there are all grades, apparently, up to the highly acute types in which the process is more wide-spread and the case may end fatally in a few days.

Symptoms. — Brissaud has proposed a useful classification of the ophthalmoplegias as follows: The affection is *total* if all the muscles of the eyes, both exterior and interior, are involved; *partial*, if only some muscles are paralyzed; *complete*, if the paralysis in the affected muscles is absolute; and *incomplete*, if the affected muscles are not absolutely paralyzed, but only paretic. Hutchinson describes an ophthalmoplegia *externa* in which the interior muscles, that is, the iris and ciliary muscle, escape; and the opposite form is the ophthalmoplegia *interna* in which only the iris and ciliary muscle are paralyzed. This is possible, because the nuclei for the iris and ciliary body lie some distance anterior to the other nuclei. Finally, ophthalmoplegia may be either unilateral or bilateral, but the unilateral cases are never nuclear, as will be explained later. All these nuclei, except the sixth, lie underneath the aqueduct of Sylvius; those for the iris and ciliary body, however, lie somewhat farther forward, even in the walls of the third ventricle.

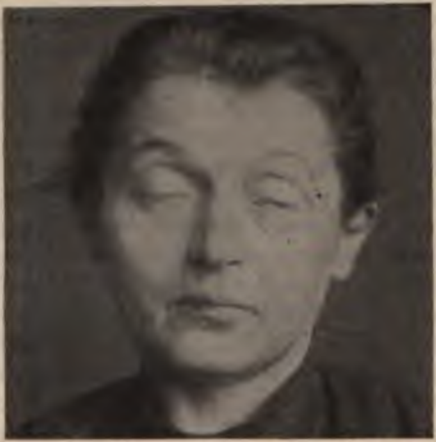

FIG. 379.—Nuclear ophthalmoplegia.—Philadelphia Hospital—Lloyd.

In a case, studied by Lloyd, the patient, a woman aged 35, noticed first external strabismus in the right eye, then ptosis, then external strabismus in the left eye, and finally, after some months, loss of all ocular movements except in the left external rectus. This patient had headache and abolished knee-jerks, but no fulgurant pains and no ataxia. There was possibly some beginning optic atrophy. No history of syphilis was obtainable.

In most of the described cases this gradual progress has been noted, significant of a slowly progressive nuclear disease. First one ocular muscle and then another, in one eye and then in the other, becomes paralyzed until all, or nearly all, are involved. Various forms of strabismus occur, until the eyeballs become motionless and ptosis is complete. It is occasionally seen in young persons, even in children, and Möbius called it then "infantile nuclear atrophy."

Diagnosis.—Syphilitic meningitis between the cerebral peduncles may involve the roots of the third nerves, and may possibly, but not probably, extend to the sixth. Headache is present, and possibly optic neuritis, but the resemblance to nuclear disease may be striking. Syphilis, however, would hardly give the picture of gradual and persistent progress, without remission, with first one muscle and then another in each eye becoming paralyzed. Syphilis of the third nerve is often unilateral. Moreover, in interpeduncular syphilis the paralysis is confined to the third nerve; the fourth and sixth escape.

Tumor of the mid-brain does not invade the nuclei alone; it usually causes paralysis of the limbs, sometimes a hemiplegia alternans, that is, paralysis of the third nerve on the side of the lesion with opposite hemiplegia; also optic neuritis, headache, and pressure symptoms. This may be true, also, of extensive syphilitic disease.

Wernicke's acute superior polioencephalitis is known by its history, its rapid course, its invasion of other than nuclear territory, and its consequent wider range of symptoms.

Nuclear ophthalmoplegia, rarely or never total, occurs in locomotor ataxia, but it is not usually an early symptom, and it is associated with true tabetic symptoms, such as ataxia, fulgurant pains, optic atrophy, lost knee-jerks, etc. The commonest form is bilateral ptosis. Some observers claim, however, that a chronic progressive ophthalmoplegia is sometimes a precursor of tabes dorsalis, combined sclerosis, multiple sclerosis, or even progressive muscular atrophy. Hence a cautious diagnosis is called for, since it may take months or even years to determine the question.

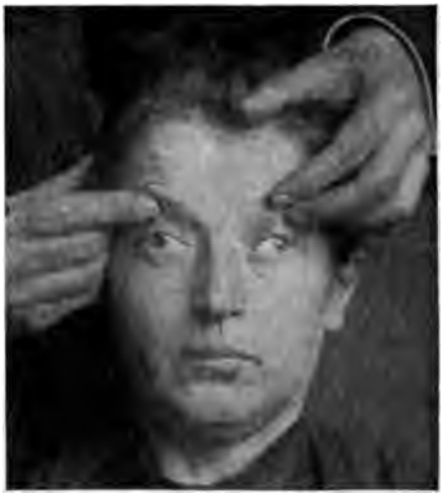

FIG. 380.—Nuclear ophthalmoplegia. Upper lids supported in order to show the position of the eyeballs.—Philadelphia Hospital—Lloyd.

Unilateral ophthalmoplegia, total and complete, is never nuclear, since nuclear disease is always bilateral. The one-sided cases are usually caused by some local lesion at the base of the brain. The first division of the fifth nerve is likely to be involved in these basilar cases; hence there is anæsthesia of the conjunctiva and brow on one side; and if the second and third divisions of the fifth nerve are involved, the anæsthesia extends over one-half of the face and tongue. These cases should not be mistaken for isolated paralysis of individual nerves, in which the loss of power is confined to the nerve affected. The third or the sixth nerve on one side is occasionally paralyzed by trauma.

PARALYSIS OF ASSOCIATED MOVEMENTS OF THE EYES.—These are the lateral, the upward, and the downward movements, and the movement of convergence. The evidence is in favor of a centre in the brain cortex for associated ocular movements. Grünbaum and Sherrington found a centre for lateral movements of the head and eyes in the frontal lobe somewhat apart from the rest of the motor area, and Ferrier has shown from many recorded instances that conjugate deviation of the head and eyes is caused by a lesion in the posterior end of the middle frontal gyrus. Perinaud, Mott, and others hold that this ocular centre is subdivided for the various associated movements; thus, one part, [illegible] the lateral, another for the upward, and still another for the downward movements. As a fact, however, it is the conjugate lateral movement which is usually affected, and this is seen in a variety of lesions, as, for instance, in large cerebral hemorrhage, and in thrombic and embolic softening. Any lesion which affects this centre directly or cuts off its underlying connections may cause conjugate lateral deviation of the head and eyes. The patient looks *toward* the side of the lesion when the paralysis is complete; but if the lesion is an irritative one, as in focal or one-sided epilepsy, he looks *away* from the lesion. The lateral deviation of the eyes that is caused by a cerebral lesion is usually temporary, and it is associated with lateral deviation of the head. It is thus distinguished from the lateral deviation of the eyes which is sometimes seen in lesions in the pons, located in the posterior longitudinal bundle, by which the nuclei of the third, fourth, and sixth nerves are joined. In these pontile cases the paralysis is not transient, it is not likely to be associated with deviation of the head, it may be accompanied with other pontile symptoms, and the deviation is sometimes, but not always, *away* from the side of the lesion.

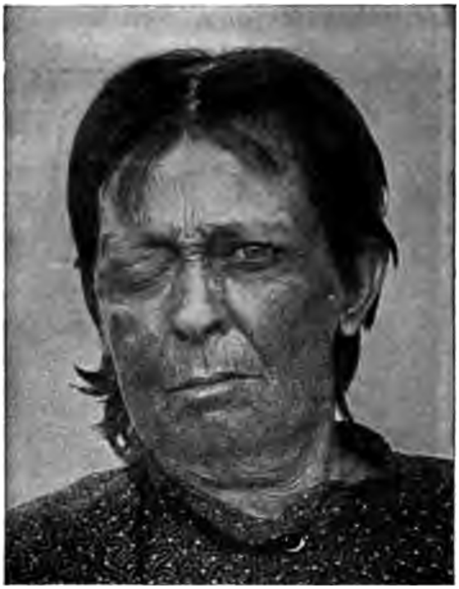
FIG. 381.—A case of unilateral ophthalmoplegia.—Lloyd.

Paralysis of the associated upward movements of the eyes is sometimes seen, and is sometimes accompanied with paralysis of convergence. The associated downward movements may be paralyzed, but this isolated paralysis is rare; it is usually associated with paralysis of the upward movements. The lesion in these cases is located on or near the floor of the aqueduct of Sylvius, and it may be a tumor, a syphilitic inflammation, or a spot of softening. It is possible, however, that paralysis of associated upward or downward movements may be caused by a lesion cutting off the cortical centres from the nuclei in the mid-brain. Wernicke has called this condition pseudo-ophthalmoplegia, and Lloyd has recently reported a case of pseudobulbar palsy, due to bilateral lesions in the lenticular nuclei, in which there was loss of power in the associated upward movement of the eyes.

Marie claims that paralysis of associated ocular movements may be caused by hysteria; but in such a case the affection would more likely be a spasm of the opposing muscles than a true paralysis. Thus a spasm of both superior recti muscles, pulling both eyes upward, would present the appearance of a paralysis of the inferior recti and the superior oblique muscles, whose function it is to pull the eyes downward. The patient would probably present other hysterical symptoms. Gilles de la Tourette thinks that hysterical paralysis of any of the eye muscles is rare, and that the real affection is a spasm or contraction of the opposing muscles. There is, for instance, an hysterical blepharospasm which may simulate a bilateral ptosis.

The muscles of accommodation (ciliary) and of convergence (internal rectus) usually act together, and in both eyes at the same time. Thus vision is accommodated for near objects. But the conjoint action of the two internal recti for convergence may be interfered with, and convergence may be paralyzed while these two muscles continue to act in other associated movements. Perlia has described a special nucleus for convergence beneath the middle line of the aqueduct of Sylvius—called Perlia's *central nucleus.* A destructive lesion at this point presumably causes isolated paralysis of convergence; that is, the internal recti muscles fail to turn the eyeballs inward in attempts at convergence, but they may act properly in associated lateral movements to the right or left.

In determining these various paralyses of associated movements it must be borne in mind that the eyeballs roll *away* from the paralyzed muscles: thus, if the upward movement is paralyzed the eyeballs roll downward, and *vice versa.* If there is right conjugate lateral deviation, the muscles paralyzed are the left external rectus and the right internal rectus.

XIX. DISEASES OF THE CEREBELLUM.

Tumor, abscess, and other focal lesions of the cerebellum have been considered under a previous heading. Besides tumor and abscess there are occasionally seen softening and terminal cysts in the cerebellum, due to occlusion of the blood-vessels, as in syphilis and atheroma. Extensive softening of one cerebellar hemisphere may occur with little or no evidence of cerebellar disease. Atrophy and sclerosis of the cerebellum are also seen in cases of arrest of development, and in the cerebellar form of hereditary ataxia.

Symptoms.—In gross lesions of the cerebellum incoördination and forced movements are often seen. There may be wide staggering and swaying, the patient standing with the legs far apart, or there may be reeling and even rotary movements. These symptoms are attributed to lesions of the middle lobe. Forced movements are supposed to depend rather on lesion of the middle cerebellar peduncles. They may be so aggravated that the patient cannot even sit upright in bed, but is forced to one side. Paralysis of the oculomotor nuclei is sometimes caused by pressure on the mid-brain; and pontile symptoms, such as paralysis of the fifth, sixth, seventh, and eighth nerves, and even hemiplegia, are also caused by gross lesions of the cerebellum pressing on the pons. Optic neuritis or atrophy is common, and headache, vomiting, and vertigo are seen just as in other intracranial affections. When one cerebral hemisphere is alone affected there may be few if any symptoms, but this is not a universal rule. The knee-jerks are variously affected. In some cases of tumor or abscess they disappear, and even return again, or they are merely exaggerated or diminished, as the case may be, or they are not the same on both sides. The functions of the cerebellum are still obscure, but they evidently are largely concerned with equilibration, as is proved by the diseases of the organ.

CEREBELLAR HEREDITARY ATAXIA is an affection nearly allied to the spinal hereditary ataxia of Friedreich, but there are well-marked differences between the two. It develops according to most authors rather later in life but has in some cases dated from a very early period, if indeed it were not congenital. In addition to ataxia there are speech defects, not mere scanning but rather an incoördinate and explosive type of speech; exaggerated knee-jerks; and optic atrophy. Nystagmus is occasionally seen. The disease may be familial, as in a remarkable series of cases reported by Sanger Brown. Lloyd is convinced that there are different types of cerebellar ataxia; that not all are necessarily familial or hereditary; and that optic atrophy is not always present. The most typical symptoms seem to be the ataxia, which is not always so extreme as in Friedreich's disease, and the incoördinate explosive speech. The knee-jerks are probably preserved or even increased in most cases. Some of these cases may be due to injury at birth. Some form of atrophy of the cerebellum has usually been found after death, but again the findings have been practically negative. The cerebellar tracts in the cord were involved in one of Brown's cases.

Diagnosis.—The diagnosis between Friedreich's ataxia and the cerebellar hereditary ataxia rests upon the difference in the speech defects, and upon the preservation or even increase of the knee-jerks, and possibly the presence of optic atrophy in the latter form. In chorea there is not a true ataxia but rather involuntary irregular movements, and an absence of the characteristic speech. Optic atrophy is not seen. The history and evolution are also different, chorea having a much more abrupt onset and a more acute course, with a tendency to recover. In insular sclerosis there is intention tremor and spastic gait, but the nystagmus and affections of speech may cause some resemblance, and the distinction should be made with care.

XX. DISEASES OF THE PONS.

The pons is well named, for it is a bridge by way of which many nerve-tracts take their course. It is also the seat of the nuclei of several important cranial nerves, namely, the fifth, sixth and seventh. The pyramidal or motor tracts from the brain pass down through the anterior parts of the pons, and the great sensory tract, known as the median and lateral fillet, passes upward through the deeper portion. The transverse fibres of the pons connect the hemispheres of the cerebellum with the opposite cerebral hemispheres, and other important cerebellar connections are probably made through the middle peduncles; and finally the cochlear nerve, or nerve of hearing, enters the lower outer part of the pons, and it, as well as the auditory tract to the posterior quadrigeminal bodies, passes through the mid-region of this great bridge.

Pathology.—Tumors, hemorrhage, softening, and meningitis are the chief lesions here, as in other regions of the brain.

Symptoms.—In such a complicated structure the symptoms vary widely; one of the most characteristic symptoms is the hemiplegia alternans, in which there is an opposite hemiplegia with paralysis of the sixth nerve causing internal strabismus, and of the seventh nerve causing facial paralysis on the side of the lesion; in some cases the fifth is also involved, also the eighth. The sixth nerves, which pass out near the median line, may both be involved even in a lesion which is mainly unilateral. The above symptom-complex points to a lesion in the lower and anterior half of the pons, and if it is a tumor it is most likely to be meningeal and located in the cerebellopontile angle. Mills has reported a case of limited softening in the lower ventral part of the pons near the median line, in which paralysis of the left sixth and paresis of the right sixth nerve were associated with left hemiplegia. There apparently was no facial palsy. The facial nerve may exhibit remarkable resisting power, as in a case observed by Lloyd, in which the seventh nerve was bent over the surface of a tumor in this region, and yet there had been no paralysis of the face. In some cases both roots of the auditory nerve are not simultaneously involved; as one of these roots subserves hearing (the cochlear nerve) and the other probably subserves equilibration (the vestibular nerve), it is well to test these two functions separately.

If the lesion is high in the pons, above the level where the motor tracts for the sixth and seventh nerves decussate, the paralysis of these nerves will be on the side opposite the lesion and on the same side as the hemiplegia.

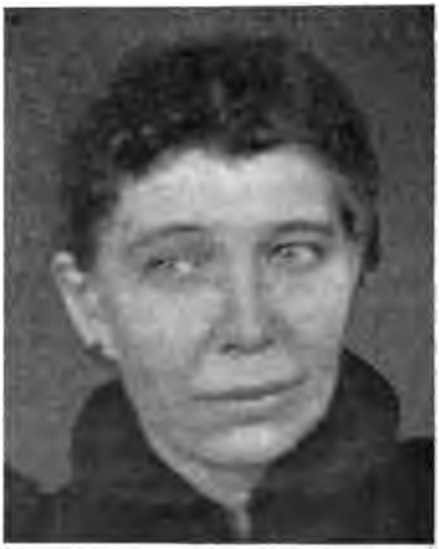

Fig. 382.—Woman with deviation of the eyes toward the right, of the head toward the left. Case of hemiplegia alternans inferioris encephalitidis pontis (crossed hemiplegia from inferior encephalitis of the pons).—Oppenheim.

Involvement of the fifth nerve causes anæsthesia of one side of the brow, face, and tongue (in whole or in part, according to the extent of the involvement), the eyeball of the affected side, and paralysis of the muscles of mastication—the temporal, masseter, and pterygoids. A neuroparalytic ophthalmia may result and totally destroy the eye.

Superficial or meningeal lesions, unless they make deep pressure, are not likely to involve the sensory tract (the fillet), which lies deeply within; nevertheless in all cases of suspected pontile lesion the limbs should be carefully tested for anæsthesia, including the tactile, the thermal, and the pain senses.

Deep lesions of the pons may cause headache, vertigo, hemiplegia, hemianæsthesia, dysarthria, paralysis of the tongue (not nuclear), convergent strabismus or even lateral deviation of the eyes, inability to swallow, and intense emotionalism, with involuntary spasmodic laughter in some cases. Rotation of the head to one side has been noted; also profuse sweating, vasomotor symptoms, and even epistaxis. Conjugate deviation of the head and eyes is said to be caused sometimes by a lesion high in the pons. Deviation of the eyes is *away* from the side of the lesion if this is in the posterior longitudinal fasciculus; and cases have been reported in which the eyes were rolled to one side and the head to the other in the hemiplegia alternans.

The symptom-complex described as pseudobulbar palsy is probably caused in some cases by a pontile lesion.

Diagnosis.—This is made from the peculiar grouping of symptoms as given above. A question may arise as to the nature of the lesion, whether it be a tumor, a hemorrhage, a softening, or a syphilitic meningitis. Tumor is usually slow in onset and gradual in its course; hemorrhage and softening abrupt in onset and not progressive; syphilitic meningitis may not be easily distinguishable from tumor, but irregularity in the onset and course of the symptoms points to specific disease. The Wassermann test should be made.

XXI. BULBAR PALSY.

In the account of progressive muscular atrophy and amyotrophic lateral sclerosis the degeneration of the ganglion cells in the anterior, or motor, horns of the spinal cord, which is characteristic of these diseases, is described. We have now to describe a disease which depends upon a similar degeneration of motor ganglion cells, but these cells are located higher in the medullary gray matter and preside over special functions; they are the motor neurons which arise in the bulb, or medulla oblongata, and especially in the nuclei of the ninth (glossopharyngeal), tenth (pneumogastric), and twelfth (hypoglossal) nerves. This disease is known as bulbar palsy, or labio-glosso-pharyngeal paralysis.

Pathology.—There is found a degeneration of the large multipolar cells in the nuclei of origin of the ninth, tenth, and twelfth nerves in the medulla, and possibly of the seventh nerve in the pons. Thus in the nucleus ambiguus, which contains the motor cell-bodies of the ninth nerve, these multipolar cells are found greatly changed; they have shrunken in size, present evidence of chromatolysis and displacement of the nuclei, and the nerve-fibrils are diminished in number. In advanced or severe cases it is evident that many cell-bodies have entirely disappeared. The same changes are found in the nucleus of the twelfth nerve, which is entirely a motor nerve. In the case of the ninth, which is a mixed nerve, the sensory ganglia (the jugular and petrous) are not involved. The diseased cells, however, are not confined to the regions just mentioned, but are found in that rather extensive mass of gray matter in the bulb from which arise motor fibres not only for the ninth but also for the tenth (pneumogastric) and even the spinal accessory. In some cases the roots of the bulbar nerves are degenerated, and occasionally some degeneration is observed in the pyramidal tracts of the cord. This sclerosis of the motor columns marks the connection of this disease with progressive muscular atrophy and amyotrophic lateral sclerosis, for that there is some relationship is evident not only from the similarity of the degeneration in the motor nuclei, but from the fact that bulbar palsy may precede or complicate either of these two diseases, especially the latter.

Symptoms.—The initial symptoms usually are disorders of speech. The articulation becomes imperfect, especially for labials and linguals, due to beginning paresis of the lips and tongue. Nasal speech occurs, and finally a very distressing dysarthria, in which the patient finds it almost impossible to make himself understood. The attempt at speech is fatiguing, and finally may be almost abandoned. Deglutition in turn becomes impaired. The patient can manage the bolus of food only with difficulty, especially in passing it back into the pharynx. There may be regurgitation of fluids through the nose, or out between the paralyzed lips, and attacks of strangling, coughing, and vomiting result. Mastication also is somewhat impaired. Phonation is altered, and becomes monotonous. Respiration may also be embarrassed. The loss of power in the lips causes inability to whistle or to show the teeth. The lower part of the face becomes immobile and expressionless; the lips are flaccid and partially open, and drooling or dribbling of saliva results. The tongue becomes so palsied that it cannot be protruded, but lies almost or quite motionless in the mouth. The paralysis is atrophic; hence all the affected muscles waste and lose tone. The tongue is flabby, wasted, and fissured. The lips are thin and lifeless. The pharyngeal reflex may be abolished, but sensation is not involved. The velum palati hangs flaccid, and the laryngoscope may reveal paralysis of the adductors of the vocal cords. As a rule, the muscles of the upper part of the face and of the eyes are not involved.

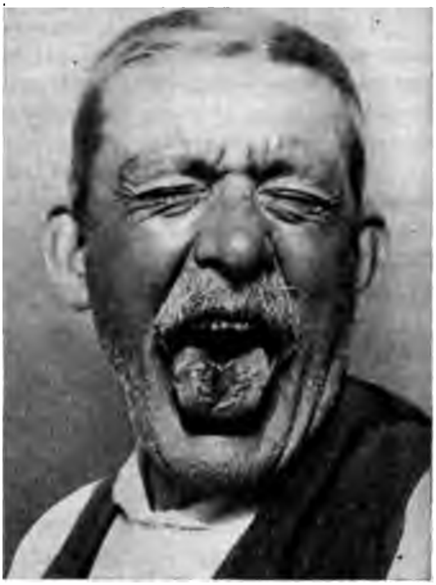

FIG. 383.—Atrophy of tongue due to partial bulbar paralysis.—Lloyd.

Occasionally there is evidence of lateral sclerosis, as exaggerated knee-jerks and some spasticity of the gait. This indicates, as already said, the kinship of this disease to amyotrophic lateral sclerosis.

The electrical reactions may be partially altered, but true reactions of degeneration are seldom seen. This is for the same reason that holds in progressive muscular atrophy; as long as any muscle fibres remain in connection with the gradually wasting nuclei they react to the current.

The onset of bulbar paralysis is usually insidious, the course is slow and chronic, and the disease is incurable. Acute cases, with rapidly developing symptoms, have been reported, but they are rare.

Diagnosis.—There is not much possibility of confusing true bulbar paralysis with any other disease. Diphtheritic paralysis has been mistaken for it, but in that disease there is no paralysis of the tongue and lips, and the history and course are different. The mistake is most likely to occur from regarding the paralysis of the velum palati and the dysphagia as evidences of an acute onset of bulbar paralysis; but the history of sore throat, the paralysis of accommodation, and the evidences of a multiple neuritis are usually sufficient to identify postdiphtheritic paralysis.

The distinction between true bulbar paralysis and myasthenia gravis presents some difficulty, but the subject is discussed in connection with the latter disease.

Organic disease, such as tumor of the pons or medulla, may simulate bulbar paralysis—may, in fact, cause a bulbar palsy—but other evidences will be present of gross organic disease, such as are described under the head of Tumors of the Brain.

There is an apoplectic bulbar palsy due to hemorrhage or vascular disease in the bulb and pons. It may simulate atrophic bulbar paralysis, but its sudden onset, often with apoplectiform symptoms, is characteristic. In some non-fatal cases there may even be a tendency for some of the earlier symptoms to improve.

An acute disease of the gray matter of the bulb, analogous to the acute anterior poliomyelitis of children, has been reported by Wernicke and others—the so-called *acute polioencephalitis inferior*. The history and nature of the attack are usually sufficient to distinguish it. The symptoms are those of bulbar palsy of rapid onset. Wernicke associates this disease with a similar affection of the nuclei in the mid-brain, causing ophthalmoplegia—the *acute polioencephalitis superior.*

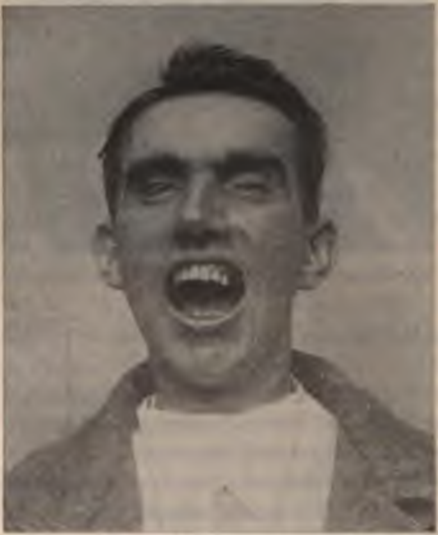

FIG. 384.—Pseudobulbar palsy, showing paralysis of the lips, tongue, and lower jaw, from lesions in the lenticular nuclei.—Lloyd.

XXII. PSEUDOBULBAR PALSY.

The term *pseudobulbar palsy* applies to a labio-glosso-pharyngeal paralysis which is of cerebral, not of nuclear, origin. To understand it we must bear in mind that there are centres in the motor cortex of the brain for the lips, the tongue, the muscles of mastication, the pharynx, and the larynx, and that these centres are connected with the nuclei of the facial, the motor branch of the fifth, the hypoglossal, the pneumogastric, and the glossopharyngeal nerves by the motor conducting paths, which run down through the internal capsule, cerebral peduncle, and pons. These nuclei are located in the pons and medulla oblongata. Hence a lesion which interrupts these motor tracts from the brain causes bulbar or pontobulbar symptoms: there is paralysis of the lips, tongue, and the muscles of mastication, of deglutition, and possibly of phonation.

Symptoms.—A few such cases have been reported, and the accompanying illustrations represent two such patients from the service of J. Hendrie

Lloyd in the Philadelphia Hospital. The nature and location of the lesions in such cases are not always clear. As the symptoms are usually bilateral, it is not easy to interpret them as due to a unilateral lesion. Sometimes the symptoms in their entirety occur only after sudden apoplectiform attacks, and the inference is that bilateral vascular lesions, such as could be caused by atheroma or syphilis, are the cause. In one of Lloyd's cases bilateral lesions were found in the lenticular nuclei. There may be hemiplegic or diplegic symptoms, not always well marked.

The muscles of the tongue and lips and of mastication and deglutition may be completely paralyzed. In one of the cases here depicted the tongue was motionless, the mouth hung open because of paralysis of the temporal and masseter muscles, the lips were paralyzed, and the patient could only swallow by thrusting the bolus of food far back into his pharynx with his finger.

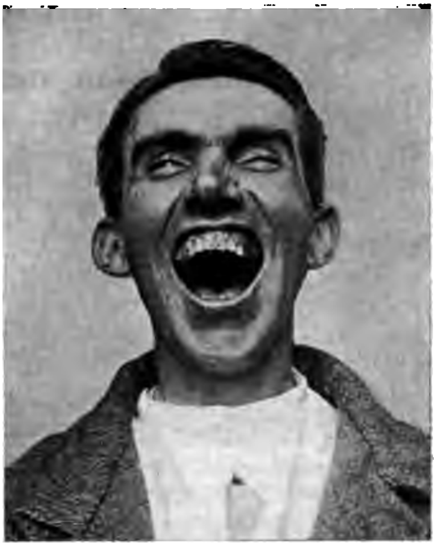
FIG. 385.—Pseudobulbar palsy; involuntary laughter.—Lloyd.

The paralysis is central, as shown by the absence of muscular atrophy, of fibrillation, and of the reactions of degeneration. A peculiar symptom is spasmodic involuntary laughter or crying. It is well shown in the cuts. Brissaud thought that this indicated lesions of the optic thalami.

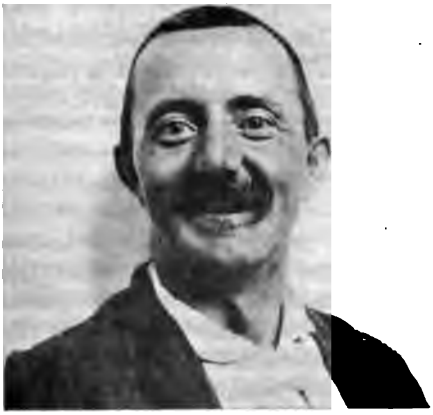
FIG. 386.- Involuntary laughter in a case of pseudobulbar palsy.—Lloyd.

Cerebral symptoms sometimes occur in these patients, such as aphasia, dysarthria, dementia, hemianopsia, etc. There is also seen in rare cases conjugate paralysis of the eyes; in one of the above cases there was paralysis of the upward movement of both eyes.

Among other symptoms rarely seen are optic neuritis or atrophy, and respiratory troubles. Anæsthesia is not commonly observed.

There has been more speculation than actual post-mortem observation about the seat of the lesion; and Oppenheim, who reviews the subject, comes to no very definite conclusion. Brissaud places the lesion, or lesions, in the posterior part of the optic thalami:[1] this was not so in Lloyd's recent case.

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[1] Leçons sur les Maladies Nerveuses, Paris, 1895, p. 446.

Diagnosis.—The disease is distinguished from true bulbar palsy by the abrupt onset, the central character of the symptoms,—the absence of atrophy, of fibrillation, and of electrical changes,—by the associated cerebral symptoms, and by the history.

DISEASES OF THE CRANIAL NERVES.

The cranial or cerebral nerves comprise twelve pairs ot symmetrically arranged nerve-trunks which are immediately connected with the brain and pass through various foramina at the base of the skull to be distributed, with the exception of the tenth pair, to the structures of the head and neck.

These pairs of nerves are numbered according to the order in which they penetrate the dura from before backward from the first to the twelfth. They have, moreover, received designations descriptive of their functions or distribution. Some of them are wholly motor; others convey impulses of special sense; while certain of them transmit impulses of common sensation and motion.

THE CRANIAL NERVES.

No.	Name.	Function.
I	Olfactory	Special sense of smell.
II	Optic	Special sense of sight.
III	Oculomotor	Motor to eye muscles and levator palpebræ superioris.
IV	Trochlear	Motor to superior oblique muscle.
V	Trigeminal	Common sensation to structures of head. Motor to muscles of mastication.
VI	Abducent	Motor to external rectus muscle.
VII	Facial	Motor to muscles of head (scalp and face) and neck (platysma). Probably secretory to submaxillary and sublingual glands. Sensory (taste) to anterior two-thirds of tongue.
VIII	Auditory,	
	(a) Cochlear division	Hearing.
	(b) Vestibular division.	Equilibration.
IX	Glossopharyngeal	Special sense of taste. Common sensation to part of tongue and to pharynx and middle ear. Motor to some muscles of pharynx.
X	Pneumogastric or vagus	Common sensation to part of tongue, pharynx, œsophagus, stomach, and respiratory organs. Motor (in conjunction with bulbar part of spinal accessory) to muscles of pharynx, œsophagus, stomach and intestine, and respiratory organs; inhibitory impulses to heart.
XI	Spinal accessory	Spinal part: Motor to sternomastoid and trapezius muscles.
XII	Hypoglossal	Motor to muscles of tongue.

Modern knowledge concerning the relative position of the cell-bodies of motor and sensory neurons renders necessary a readjustment of the former views concerning the superficial and deep origin of the cranial nerves and their course from the brain to parts outside the skull. Only the motor fibres of the cranial nerves arise from nerve-cells within the cerebrospinal axis, while the fibres which transmit sensory impulses have their origin from cell-bodies forming ganglia situated outside of the central nervous system and in the course of the nerve-trunks. The term "deep origin" as indicating cell-groups constituting nuclei within the brain and "superficial origin" as indicating the point of attachment to the surface of the brain can only be properly employed in regard to motor nerves and the fibres of motor and sensory nerves which convey motor impulses.

The cell-groups with which the terminal arborizations of the sensory fibres come into relation within the cerebral substance are not nuclei of origin but of termination—nuclei of reception. The impulses which they receive are transmitted to various parts of the brain by neurons of the second, third, or even higher order. The motor nerves then have their deep origin within the substance of the brain, their superficial origin at the point of their attachment to the surface of the brain, and their exit from the skull by the various foramina. The sensory nerves have their origin in their respective ganglia, their entrance into the skull by way of certain foramina, their points of attachment to the brain, and their nuclei of reception. Finally, the nerves of common sensation and motion, viewed from the standpoint of the direction of the impulses which they convey, whether they be afferent or efferent, contain fibres which enter the brain and fibres which make their exit by way of the respective foramina.

Every cranial nerve is directly or indirectly in relation with groups of neurons in the cerebral cortex. These groups constitute the higher cortical centres, the location of which in the case of many of the nerves has been more or less accurately determined.

I. FIRST NERVE.

The term olfactory nerve, formerly employed to designate the olfactory bulb and tract as well as the filaments, is now employed to describe the paths of conduction represented by a number of minute filaments which connect the perceptive elements situated within the Schneiderian mucous membrane with the olfactory lobe. In man the olfactory bulb and tract with its roots represent as rudimentary structures the more developed olfactory lobe of animals in which the sense of smell is keen. The true olfactory nerves, which number about twenty, are the axons of the neurons—the olfactory cells—which are situated in the olfactory area. This space is limited in extent, comprising on the outer nasal wall less than the mesial surface of the superior turbinate bone and a slightly more extended distribution upon the upper part of the nasal septum. These filaments pass upward by way of openings in the cribriform plate of the ethmoid bone and enter the olfactory bulb by its under surface.

Lesions of the nasal mucous membrane involving the olfactory area or the upper turbinate bone or the adjacent part of the septum, are attended with impairment or loss of the sense of smell. Lesions of the uncinate gyrus may also cause loss of smell upon one or both sides. The conduction path may also be destroyed in fractures of the base of the skull in the anterior fossa, involving the cribriform plate. Irritative lesions cause perversion of the sense of smell—parosmia; destructive lesions partial or complete loss—anosmia. Hallucinations of the olfactory sense may be symptomatic of hysteria, insanity, or tabes, and constitute one of the various forms of aura in epilepsy.

The sense of smell may be tested by presenting to each nostril in turn bottles containing familiar aromatic substances, as the oils of clove, peppermint, or asafœtida.

II. SECOND NERVE.

The ganglion cells among the rods and cones of the retina are the beginnings of the optic nerve; its apparent origin at the papilla is simply the point where the axons from these retinal cell-bodies, coming together, form the trunk of the nerve.

The ophthalmoscope is of special value in neurologic diagnosis because it lays bare, in the papilla, a great nerve close to the brain. Of the lesions thus directly revealed, papillitis or optic neuritis is usually a symptom of intracranial pressure or inflammation. When attended with much swelling it becomes "choked disk," since the optic foramen, unyielding, squeezes the swollen fibres. For the same reason optic neuritis is prone to pass into optic atrophy; but the latter is often primary, as in tabes, the optic nerve being, like other sensory roots, liable to degeneration in this disease.

For the consideration of vision in neurology, the retina is divided into lateral halves. The fibres from the right half-retinas (nasal half of left retina, temporal half of right) run together at the chiasm to form the right optic tract. Their arrangement is like that of the lines for driving a team of horses, the right line (as the optic tract) dividing to go to the right side of each horse's head (as to the right half of each retina) and, *mutatis mutandis*, the same applies to the left half-retinas. The partial decussation at the chiasm, then, is a device to make the two eyes, like a team of horses, act as one (binocular vision).

The optic tract passing back winds around the brain-stem (crus) to enter it dorsally after the manner of spinal sensory roots. It meets here its superior cell-bodies in three structures, the pregeminum, the pregeniculum, and the pulvinar, which constitute the "primary optic centres." From these cell-bodies, axons (the optic radiations) arise to pass into the posterior part of the internal capsule and outside the posterior horn of the lateral ventricle to the cortical optic centre in the cúneus, or, more exactly, in the region bordering the calcarine fissure. Lesion at any point in this path from the chiasm to the occipital cortex affects the half-retinas of the same side. Thus it appears that physiologically there is no cross-way in the optic path within the brain; yet just as an object touching the left side of the body is felt in the right half of the cerebrum through a crossing within the brain-stem, so an object on the left (in the left half-field of vision) is *seen* by the right half of the cerebrum through a *crossing*, not of nerve-fibres but of *rays of light within the eyeballs* (vitreous chambers); and as lesion of the right touch-path (fillet) causes left hemianæsthesia, so lesion of the right optic path, by affecting the right half-retinas, causes blindness of the left half-fields, called left hemianopsia. The varieties of hemianopsia are named by the fields which are darkened, not by the blind part of the retina. Tumor of the pituitary body by pressure at the chiasm destroying the inner fibres of each nerve from the nasal half of each retina causes temporal hemianopsia. More severe pressure may cause total blindness.

The optic tract may be pressed upon by tumor at the base. sometimes growing from the temporal lobe. There is hemianopsia, with Wernicke's sign and general symptoms of brain tumor. The primary optic centres may be the seat [illegible] tumor or they may be pressed upon by a tumor of the middle lob[illegible]is) of the cerebellum.

The optic radiations may be involved in tumor, hemorrhage, or softening at the hind part of the internal capsule, adding hemianopsia to the symptoms of capsular lesion; or one of these lesions may implicate the radiations farther back in the subcortex, when hemianopsia may exist alone; or a lesion in the angular gyrus may invade the radiations beneath, adding hemianopsia to the cortical symptoms of mind-blindness, etc.

Lesions in the vicinity of the calcarine fissure cause hemianopsia; occasionally they are bilateral, causing double hemianopsia which amounts to total blindness (amaurosis). The half-retinas are, as it were, mapped out upon the cuneus, so that, half of it being destroyed, there is blindness in a quarter of the opposite fields (quadrantic hemianopsia). Color appears to be separately represented. On the outer surface of the cerebrum, in the angular gyrus, apparently, is a higher centre for visual concepts. With lesion there the patient has mind-blindness, including word-blindness.

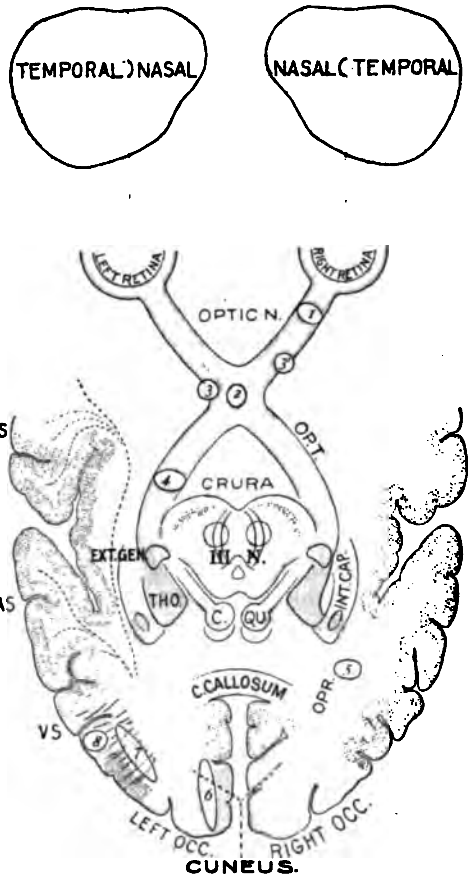

FIG. 387.—Diagram of visual system. Modified from Vialet. OP. T., optic tract; INT. CAP., internal capsule; OP. R., optic radiation; THO., optic thalamus; EXT. GEN., external geniculate body; C. QU., corpora quadrigemina; MS., motor speech centre; AS. auditory speech centre; VS., visual speech centre.

Lesions at the points indicated by the figures in the diagram cause the following morbid conditions: 1. blindness of the corresponding eye; 2, bitemporal hemianopsia; 3, nasal hemianopsia; 3, and 3', binasal hemianopsia; 4, right lateral homonymous hemianopsia with Wernicke's hemianopic pupillary inaction sign; 5, left lateral homonymous hemianopsia with normal pupillary reflexes; 6, right lateral homonymous hemianopsia with normal pupillary reflexes; 7, amblyopia (especially on the side opposite the lesion); 8, on the left side, word-blindness.

The mid-brain, receiving the great sensory eye-nerve (optic), sends back to the eye its chief motor nerve (third or motor oculi). These two nerves are the limbs of the reflex-arc through which the pupils react to light; and lesion of either may cause among other symptoms impairment of the light reflex.

Wernicke's "hemianopic pupillary inaction" is a sign of lesion at the base of the brain (see p. 373).

The third nerve nuclei just beneath the anterior gemina are connected with these bodies, or with the optic tract in front of them, by collateral fibres to complete the light reflex arc, and lesion (usually tabetic or paretic degeneration) of these collaterals may impair the light reflex alone, causing reflex iridoplegia without other eye-symptoms. Loss of the light-reaction of the iris with the preservation of the reaction in convergence and accommodation is called the Argyll-Robertson pupil. It occurs in tabes and in paresis.

OPTIC NEURITIS—PAPILLITIS.

Inflammation of the optic nerve, visible with the ophthalmoscope. When the swelling causes bulging of the nerve-head to the extent of two diopters or more it is called choked disk. An affection of the nerve back of the eyeball, causing peculiar symptoms, is called retrobulbar neuritis. Sclerosis of the nerve, seen in the papilla, is optic atrophy. Ordinarily, as the sequel of optic neuritis, it is consecutive atrophy; when a part of the degeneration in tabes, etc., it is primary; when a symptom of brain disease, like tumor, secondary optic atrophy.

Impairment of vision and of the iris-reflex to light, both in varying degree, with ophthalmoscopic changes in the disk, indicate disease of the optic nerve. The field of vision is contracted, sometimes irregularly. The diagnosis rests mainly upon ophthalmoscopic examination; the prognosis upon the cause of the condition. In general it is grave.

III. THIRD, FOURTH, AND SIXTH NERVES (MOTOR NERVES OF THE EYE).

Supplying the internal muscles of the eyeball, except the dilator fibres of the iris, and the external muscles, except the external rectus and the superior oblique, the third nerve is the most important motor nerve of the eye, whence its name, *motor oculi*.

As cortical centres control movements, not muscles or nerves, the third nerve with its opposite actions is not totally affected in cerebral palsies. When in a case of head injury one pupil is dilated and immobile, this (Hutchinson pupil) is said to be pathognomonic of extradural hemorrhage.

The internal rectus, supplied by the third nerve, when paralyzed, permits the eyeball to turn outward (divergent strabismus); there is double vision with the secondary image on the opposite side (crossed diplopia). The inferior rectus being paralyzed, the eyeball fails to move downward and to some extent outward; of the double vision, the secondary image is below. The superior rectus paralyzed, the eyeball does not move upward, nor perfectly outward; the secondary image is above. To look with this eye the head is thrown back. The inferior oblique is opposite, in action and in the effects of paralysis, to the inferior rectus. The superior oblique, supplied by a separate nerve, the fourth or trochlear, is often paralyzed; the effects are opposite to those of paralysis of the superior rectus. The fourth nucleus, under the posterior geminum, is a continuation of the third. The sixth nucleus, in the lower part of the pons, is another link in the chain of gray matter for the ocular muscles. The external rectus, supplied by the sixth or abducent nerve, is more often affected alone than any other ocular muscle. When the entire third nerve is paralyzed the eyelid droops (ptosis), the eye turns outward by the action of the sixth nerve, and slightly downward by the action of the fourth, the pupil is larger than its fellow and fails to react to light or in accommodation. Lesions of the trunk of the nerve are generally unilateral; they may affect the extra-ocular muscles, while sparing the iris-movements and accommodation. The third nerve-trunk in the orbit may be injured, as by a blow on the temple, or compressed by an orbital growth. It may be the seat of neuritis, from rheumatism, alcoholism, or diphtheria, or of degeneration in tabes. Within the skull it may be implicated in meningitis or compressed by tumor or aneurism. In its course through the crus it may be compressed by tumor, or suddenly paralyzed by hemorrhage, embolism, or thrombosis, commonly associated, by implication of the motor pathway, with hemiplegia of the opposite side (Weber's syndrome). In the cortex there is no representation of the third nerve as a whole, but of the various movements governed by it. In traumatism of the convexity on one side inducing extradural hemorrhage, the pupil of this side may be dilated and immobile (Hutchinson pupil). Lesions, as apoplexy, affecting the motor pathway within the cerebrum often cause conjugate deviation of the eyes, with the head ordinarily toward the side of the lesion. Finally, syphilitic disease, either gumma, meningitis, or neuritis, often selects the third nerve. The palsy of myasthenia gravis is often in the domain of the third nerve (recurring palsy—ophthalmic migraine).

Nuclear Ocular Palsies.—(See p. 1295.)

IV. FIFTH NERVE.

The fifth or trifacial is the great nerve of common sensation for the head. Its motor branch, for mastication, is subsidiary. The surfaces supplied by its three branches, namely, the ophthalmic and the superior and inferior maxillary nerves, are shown in the accompanying illustrations.

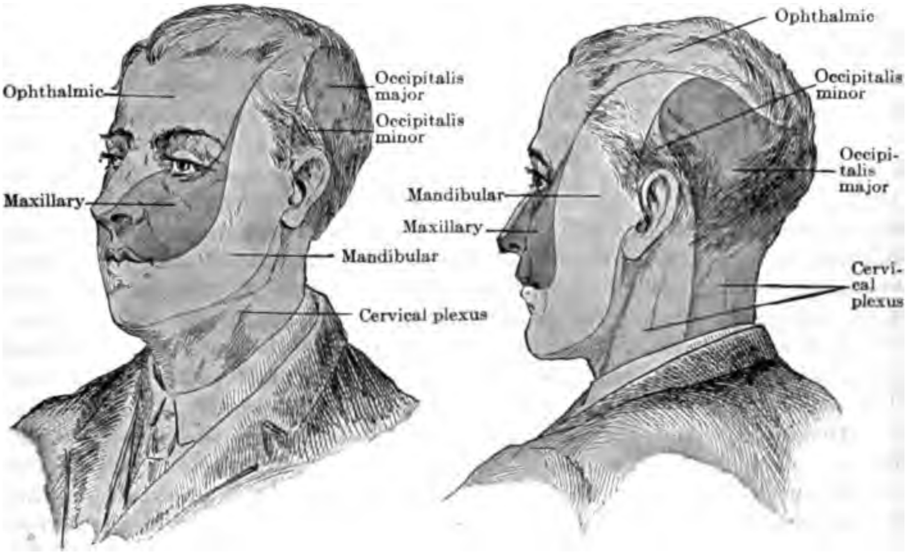

Fig. 388.—Showing distribution of cutaneous branches of trigeminal and cervical spinal nerves.—Piersol.

Entering the cranium—the first branch by the sphenoidal fissure, the second by the foramen ovale, the third by the foramen rotundum—the branches unite in the Gasserian ganglion, thence to enter the side of the pons, midway between its upper and lower borders. At this level in the back of the pons is the main nucleus of the fifth. A long column of gray matter and connecting fibres (mid-brain root) extending along the aqueduct of Sylvius forms the motor root, which leaves the pons just above the sensory root and passes under the Gasserian ganglion, and a similar chain descending at the side of the medulla conveys sensory impulses down to the cervical cord.

Diseases of the Fifth Cranial Nerve.—Branches of the fifth may be the seat of neuralgia, from cold or from dental affections; they may be damaged by wounds. Sclerosis of the Gasserian ganglion may be the cause of facial hemiatrophy; it is the usual cause of trifacial neuralgia or tic douloureux, and may be the seat of irritation, giving rise to herpes zoster of the face. Hemorrhage, tumors, or other lesions within the pons, paralyzing the fifth, cause anæsthesia of various areas of the face. Meningitis, syphilitic lesions, tumors beneath the pons often implicate the roots of the fifth.

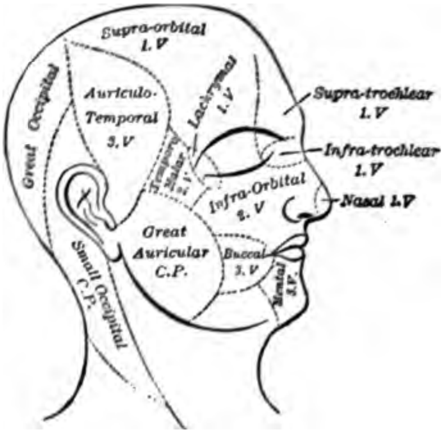

FIG. 389.—Normal distribution of the fifth nerve to the face. *1. V*, ophthalmic division; *2. V*, superior maxillary; *3. V*, inferior maxillary. The names on the different areas indicate the branches supplying them. (Flower.)—Posey and Spiller.

Symptoms.—Disease of the fifth nerve may cause first neuralgic pain, but the chief effect is anæsthesia in the distribution of one or more of its branches to the middle line of the face. A touch upon the conjunctiva is not felt, and does not excite the flow of tears. Fumes in the nostril have no effect, and on the tongue, especially its anterior two-thirds, substances whose taste-qualities are allied to touch are not recognized. The salivary secretion fails, the mucous membranes are dry, and from slight injury ulcers may form upon them, particularly over the cornea, which becomes clouded, opaque, and may perforate, leading to panophthalmia (neuroparalytic ophthalmia).

The motor portion of the fifth being paralyzed, the jaws are not closed so firmly on the affected side, and in opening deviate toward that side. The weaker action of the temporal and masseter can be felt by the fingers.

Diagnosis.—Anæsthesia of half the face, including the mucous membranes, when it exists alone points to lesion of the Gasserian ganglion or of the nerve-trunk between this and the pons. Anæsthesia corresponding to one branch of the fifth may be due to lesion at any point in the course of the branch. When the fifth nerve and the cranial nerves next in order—the sixth or seventh, fourth, or third—are affected together, the lesion is at the base of the brain involving the roots of these nerves. Anæsthesia of one side of the face and [illegible] the arm and leg of the same side (hemianæsthesia) points to lesion in the posterior third of the posterior limb of the internal capsule; but of the face on one side and of the arm and leg opposite (crossed anæsthesia) lesion in the pons, on the side of the facial anæsthesia. With the latter there may be loss of the associated movement of the eyes to this side and there may be a corresponding crossed motor paralysis.

DIFFERENTIAL DIAGNOSIS.—Hysterical hemianæsthesia is not associated with dryness of the mucous membranes; the tears flow on irritation of the conjunctiva and the special senses are affected on the anæsthetic side.

Tic Douloureux.—This is an aggravated and persistent form of neuralgia in the trigeminal nerve.

Pathology.—The disease has often been described as idiopathic, but recent observations, especially by Horsley, Rose, Putnam, Spiller, and others, have tended to show that there are degenerative or sclerotic processes in the nerve-fibres and in the Gasserian ganglion. The tendency for the disease to pass slowly but surely from one branch to the other, even after the branch first affected has been excised, seems to indicate that the process spreads from one group of neuron cells to the others in the Gasserian ganglion in somewhat the same way as the motor neurons of the anterior horns of the spinal cord are involved in progressive muscular atrophy. The essential causes of this process are obscure.

Symptoms.—The chief and usually the only symptom is pain. Lachrymation, flushing of the face, and spasmodic movements of the facial muscles are occasionally seen. Herpes has been observed in some cases, but it is doubtful whether it belongs to the disease proper.

The pain is intense, atrocious, even agonizing. It sometimes occurs in paroxysms or exacerbations, but in many cases there is more or less constant suffering. The paroxysms are usually spontaneous, but they can also be excited by trifling causes, such as movements of the face, attempts at talking or eating, or even a draught of cold air. It is characteristic of tic douloureux to begin in one division, or even in one branch, of the fifth nerve, and then to spread in time to other branches. The progress is usually slow and chronic. Many cases are operated on, but excision of the offending branch, while often giving relief for longer or shorter periods, seldom effects a radical cure, the pain returning in another branch.

Spasm of the facial muscles is seen in some cases and even constitutes a special type of the disease (the so-called convulsive or epileptiform tic), but it is not common. The pains in these cases are usually paroxysmal and severe; they occur with lightning-like quickness, and the facial muscles are thrown into twitchings and spasmodic movements. The taking of food is sometimes seriously interfered with by the pain.

Paralysis and anæsthesia are not seen in tic douloureux. Inhibition of movement is caused by the pain and the fear of pain, but neither the facial nor the masticatory muscles (the latter of which are supplied by the motor branch of the fifth nerve) are truly paralyzed. Anæsthesia in the territory of the fifth nerve is also absent as an almost universal rule; a few exceptions have been noted, but they properly raise a question whether the case is typical. Neurotrophic disorder of the eye, as seen in organic disease of the fifth nerve, is also not observed. The affection is unilateral.

Diagnosis.—The disease is unmistakable. The gradual establishment of severe pain in one branch of the fifth nerve, its progress in time to other branches, its intractability, and the facial spasms (when they occur) are easily recognized. The only doubt that may arise is with reference to the causation and pathology.

Organic lesions of the fifth nerve, such as occur from tumors, meningitis, etc., may cause pain, but usually they also cause anæsthesia of the face, brow, eye, and tongue, and paralysis of the masticatory muscles, and the pain is not always intense or strictly limited to a branch of the trigeminus. Moreover, in such cases the symptoms are seldom confined to the fifth nerve.

The term "epileptiform," as applied to the type in which facial spasms occur, is a misnomer. The disease has no relation to epilepsy.

Masticating Spasm.—Tonic spasm in the domain of the motor fifth occurs as trismus or "lockjaw" in tetanus; occasionally in tetany, in hysteria, and reflexly in dental affections, like caries of a molar. Clonic spasm, noticeable in a chill and in the epileptic convulsion, occurs rarely as an isolated affection called "chattering teeth."

V. SEVENTH NERVE.

The seventh or facial nerve, arising from the nucleus ambiguus, passes behind and over the sixth nucleus and out at the side of the pons near its lower border. With the eighth it enters the internal auditory meatus, then alone passes in the Fallopian canal close to the tympanum, and finally, through the stylomastoid foramen, emerges upon the face.

Paralysis of the facial muscles may be supranuclear or central as the result of lesion of the centre in the lower Rolandic cortex, or of the fibres from this centre passing down through the brain, commonly as a part of hemiplegia; or nuclear in consequence of lesion of the nucleus in the pons; or infranuclear from lesion of the nerve-trunk at any point. The ordinary form of facial palsy is "peripheral" from neuritis in the Fallopian canal, and is called Bell's palsy. Hemorrhage or softening in the pons, damaging one facial nucleus, may paralyze the face on that side and affect the adjacent motor pathway, the arm and leg of the other side (crossed paralysis). The seventh nerve may also be paralyzed in that rare form of tetanus known as cephalic tetanus.

At its emergence from the pons the seventh nerve may be implicated in meningitis, or compressed by a new growth, which may also involve the sixth and eighth nerves. Within the Fallopian canal the seventh nerve may be encroached upon by caries of the temporal bone from middle-ear disease. It may be damaged in operations.

Bell's Palsy.—This affection is ascribed to neuritis from exposure. The nerve swells in its bony case and is compressed. The corresponding half of the face is rapidly paralyzed.

Symptoms.—The lines of expression are smoothed out; the mouth droops on that side, and the lower eyelid sags and lets the tears run down. In "showing the teeth," the mouth and cheek are dragged toward the sound side; in looking up, the forehead does not wrinkle on the affected side; and in the attempt to close the eye, the lids remain apart.

The palate moves symmetrically and the tongue is protruded in the middle line, though by the distortion of the mouth it appears to deviate. Liquid, in drinking, or saliva runs from the corner of the mouth, and in chewing the food gathers in the cheek. The sense of taste on the front of the tongue, supplied by the chorda tympani, is impaired in some cases, as this nerve accompanies the facial within the Fallopian canal for a short distance. The reaction of degeneration occurs typically in facial palsy. Both nerve and muscle show diminishing irritability to faradism after a few days, while to galvanism the muscle contracts excessively, and in the serial order of the reaction of degeneration. Bell's palsy is rarely sudden, but usually rapid, developing in a few hours or days. It lasts ordinarily two or three months. In severe cases, after four or five months, contractures of the affected muscles deepen the lines of expression, so that the face appears normal or the sound side looks weaker.

Diagnosis.—In recent cases the condition is obvious. In older ones the muscular contracture and overaction may conceal it; but strong movements in showing the teeth or closing the eyes will show the difference of the two sides. Cerebral (supranuclear) paralysis of the facial is usually a part of hemiplegia. In this form the upper half of the face (orbicularis palpebræ, frontalis, and corrugator supercilii) regains power in a few days, through its bilateral innervation from the cortex; and even the lower half moves fairly with emotion, as in quiet smiling. In the cerebral form the supra-orbital reflex is preserved. Lesion at the base of the brain is indicated by concomitant paralysis of adjacent nerves, particularly the sixth and eighth. Deafness with facial palsy may result from tumor also involving the eighth nerve. In peripheral (nerve-trunk) palsies the entire half of the face is affected for all movements, voluntary or emotional, and the electrical reaction shows degeneration.

Prognosis.—Early return of power though slight is a good sign. Toward the end of the second week of paralysis an electrical examination gives valuable information. If at this time the faradic irritability is simply lessened, the paralysis will disappear in about two months; if lost, the outlook is bad, though some return of power is possible after several months. With the loss of faradic irritability occur the true reactions of degeneration to the galvanic current.

Facial Spasm.—As a symptom this occurs in epilepsy and chorea, in facial paralysis, in cerebral palsies as a part of athetosis, and as habit spasm. The habitual occurrence of spasm in one or several muscle-groups of the face is called *convulsive tic*. The orbicularis palpebræ and the zygomatics are its most frequent seat. Convulsive tic is a disease of later middle life (forty-five to sixty) more frequent in women. Prolonged anxiety is a factor; also, reflexly, a great variety of painful affections, as caries of a tooth.

Symptoms.—In the usual form of convulsive tic the eye is squeezed shut and the angle of the mouth drawn out and up momentarily at intervals. It is generally made worse by disturbing emotions. The spasm may be more extensive, involving other muscles of the face, mouth, neck, or arms, and especially in the platysma, which stands out on the side of the neck. In severe cases the spasm occurs in numerous quick jerks or frequently repeated contractions in the course of two or three minutes.

Convulsive tic, usually slight at first, increases gradually in the intensity and frequency of the spasm, and in the extent of the musculature involved. It is likely to continue indefinitely, but sometimes ceases after years. Intermissions of several months may happen.

Diagnosis.—Facial spasm is unmistakable. The spasm may be symptomatic of some gross disease. True convulsive tic is idiopathic. Sources of reflex irritation in the teeth, eyes, etc., must be investigated. Intracranial disease, causing facial spasm, may be tumor or other lesion of the face centre in the cortex, or of the root of the seventh nerve beneath the pons. From such a cause the affected muscles often will be found paretic, or will become paralyzed.

VI. EIGHTH NERVE.

The auditory nerve is physiologically two nerves—the cochlear for hearing, the vestibular for equilibration. From the distributions in the internal ear (the cochlea and the semicircular canals) the two parts, united as the eighth nerve, pass from the internal auditory meatus into the side of the pons. Here the two parts of the nerve, again separating, embrace the inferior cerebellar peduncle, the cochlear on its outer side, the vestibular on the inner, to connect with various nuclei in the pons and thence to seek different central goals. The cochlear fibres pass up in the lateral fillet, and by way of the postgeminum and postgeniculum reach the auditory centre in the first temporal convolution. The vestibular fibres pass to the middle lobe of the cerebellum.

Deafness.—Total deafness from birth or early childhood, depriving the child of speech, constitutes deaf-mutism. Acquired deafness frequently depends on disease of the labyrinth; but this is often secondary to middle-ear disease, particularly of the chronic catarrhal variety, or to meningitis by extension through one of the foramina. Basal fracture often enters the internal ear. The eighth nerve at its junction with the pons may be involved in meningitis, aneurism, or tumor, particularly fibroma of the nerve sheath. Degenerative disease, as tabes, may attack the eighth nerve. Pontine lesions rarely affect this nerve; but at the level of the posterior geminum, in the hinder part of the internal capsule or in the first temporal convolution, the auditory pathway may be damaged by tumor, hemorrhage, softening, etc., causing deafness of the opposite ear. Impaired hearing may be functional, as in hysteria.

Symptoms. — If no objective signs of obstruction of the external meatus or disease of the middle ear are present, deafness may be ascribed to conditions which affect the reception of sound in the labyrinth or its conduction by the auditory nerve, or to lesions involving the central auditory tract. This is especially the case when the deafness is unilateral. When the sound of a tuning-fork held against the mastoid process—bone conduction—has ceased to be heard but is again perceived when the instrument is moved to a position opposite the external meatus—aërial conduction—labyrinthine disease may be suspected. When in unilateral deafness the sound of a tuning-fork in contact with the vertex at the middle line is perceived more distinctly on the side of the deaf ear, the fault of hearing is due to the conducting apparatus; when it is heard more distinctly or only in the sound ear the deafness is caused by labyrinthine disease. In the latter condition there is an interval varying from one to several seconds between the time at which the patient ceases to hear the sound and the examiner ceases to feel the vibrations of the fork. There are no direct means by which deafness arising from lesion of the auditory nerve in its course can, in the absence of the signs of involvement of adjacent structures, be distinguished from that caused by disease of the auditory centres.

The locality of the eighth nerve-root, spoken of as the pontocerebellar angle, is a favorite seat of tumor (fibroma) which grows from the sheath of this nerve. This is recognized by its pressure-effects, paralysis of the facial and external rectus on the same side, deafness, vertigo, and incoordination, the latter partly of cerebellar origin. Deafness from a higher seat, the quadrigeminal region, or the internal capsule (posterior extremity) is usually associated with hemianopsia, and sometimes with other disturbances on the same side. Cortical deafness is likely to be of special character (word-deafness, etc.) related to aphasia. Sudden deafness indicates a vascular lesion, especially hemorrhage, most frequently in the internal ear. Hysterical deafness may be recognized by the associated symptoms.

Auditory Irritation.—Uncomfortable acuteness of hearing (hyperacusis) is ordinarily hysterical, though observed occasionally in facial palsy. Tinnitus aurium embraces simple subjective noises, as ringing, hissing, and roaring, referred either to the ear or to some part of the head. More elaborate sounds, as words seemingly spoken in the ears, in other parts of the body, or at a distance, are called auditory hallucinations. Tinnitus is a common symptom in the various diseases of the internal ear, as well as of the middle ear and external meatus. Tinnitus may arise especially in elderly persons, without definite cause. It is common in neurasthenia. In some cases it has a pulsating character, and is then referred to vasomotor disturbance in the internal ear. Head injuries, sudden loud noises, and, above all, the habitual subjection to noise (as in boiler-makers) dispose to it. Tinnitus is commonly associated with partial deafness, but may be accompanied by hyperacusis.

Diagnosis.—Irritation of the cortical centre (first temporal convolution) is a cause of hallucinations of hearing, not of simple tinnitus. Tinnitus due to irritation of the eighth nerve-trunk is known by the associated symptoms. Disease of the internal ear is the commonest cause.

Prognosis.—In a case of organic origin the prognosis is that of the primary disease. In functional disease, like neurasthenia, the symptom subsides as the patient improves. In some instances tinnitus is stubbornly persistent.

Ménière's Disease.—An affection characterized by noises in the ear, sudden attacks of vertigo with nausea and vomiting, and nervous deafness, which in many cases is progressive. The attacks are often apoplectiform, with momentary loss of consciousness.

This disease was first described by Ménière in 1861. The term should be restricted to the affection characterized by the complexus of symptoms about to be described.

Etiology.—Age plays an important part in the predisposition. The affection is very rare in early life. In a large proportion of the cases the attacks first show themselves between forty-five and fifty-five, but they may come on much later. Men suffer more frequently than women. Nothing is known of the exciting causes.

Symptoms. — The disease is paroxysmal, the attacks occurring at irregular intervals, and very often in series, several of which may take place in one day or on successive days. Such series or single attacks may be separated by intervals of weeks or even months. The attack begins suddenly with tinnitus aurium and subjective or objective vertigo of such intensity that the patient, in order to prevent himself from falling, is obliged immediately to catch some support or to sit or lie down. If loss of consciousness occurs it is momentary. Occasionally ocular symptoms accompany the attack. These consist of diplopia or nystagmus. Forced movements may occur, and in the intervals of frequent attacks there is an impairment of equilibrium, so that the patient walks with difficulty. The attack is usually of short duration. As the vertigo passes off the patient is pale, breaks into a profuse sweat, suffers from nausea, or there may be actual vomiting. As a rule, there is no disease of the middle ear. When it is present the association is accidental. The deafness, which is nervous, usually affects one ear only. It is progressive but never complete. When deafness becomes complete the vertigo ceases, the end organs of the nerve being destroyed.

Three principal theories have been suggested to account for the phenomena of Ménière's disease: 1. That the symptoms are due to lesions of the labyrinth. There is progressive degeneration of the nerve or its end organs. 2. That the disease is a vasomotor neurosis of the vessels of the labyrinth. 3. That the primary trouble consists in an affection of the centres for hearing and equilibration. Of these the first is at present generally accepted.

Diagnosis.—The direct diagnosis of Ménière's disease rests upon the paroxysmal vertigo, the apoplectiform seizure, the occurrence of tinnitus, nausea, and vomiting, and the progressive nervous deafness. The differential diagnosis between the vertigo which is so prominent a symptom and other forms of vertigo depends upon the association of the foregoing symptoms, the paroxysmal nature of the attack, and the absence of other pathological states usually attended with vertigo.

Prognosis.—This is uncertain. A small proportion of the cases terminate, after a variable duration, in complete recovery, with total loss of hearing in the affected side. More commonly the disease proves persistent and intractable, and, with periods of exacerbation and improvement for which no explanation is to be found, continues throughout life. In rare instances the symptoms are so severe that the patients become bed-ridden.

VII. NINTH NERVE.

The ninth, tenth, and eleventh nuclei form a continuous chain of gray matter, and the nerves a continuous line of fibres springing from the side of the medulla, in the order of their numbering. The ninth or glossopharyngeal, mainly sensory, supplies the back of the tongue, the soft palate, tonsils, and adjacent pharynx, with the Eustachian tube and middle ear. The muscles of the upper pharynx are probably governed by the ninth.

Tumors or meningitis affect the ninth usually in company with other nerves. Swallowing is embarrassed by lesions of the nerve-trunk or, as in glosso-labio-laryngeal paralysis, by degeneration of its nucleus.

VIII. TENTH NERVE.

The tenth nerve, termed the pneumogastric or vagus, arises in the medulla by a line of nuclei and fibres continued downward from those of the ninth. It is the chief of the "bulbar" nerves in the variety and importance of its functions, supplying motor fibres to the muscles of the pharynx, œsophagus, stomach, and intestines, and to those of the larynx, trachea, and bronchi; sensory fibres to the dura mater, external ear, pharynx, œsophagus, stomach, larynx, trachea, bronchi, and the pericardium; and spinal fibres to the heart, liver, spleen, pancreas, kidneys, suprarenal bodies, and intestinal blood-vessels. The "respiratory centre" and "cardiac centre" are thus contained in the vagus nucleus, though for these vital functions, as for vasomotor regulation and for the movements of the stomach and intestines, the sympathetic acts in connection with this nerve.

The tenth nucleus may be implicated in softening, hemorrhage, or tumor of the medulla, usually with adjacent nuclei, inducing paralytic effects in combination known as "bulbar symptoms." Degeneration of the tenth nucleus in glosso-labio-laryngeal paralysis (chronic bulbar palsy) and its inflammatory destruction in acute bulbar palsy are responsible for the impaired phonation, difficult swallowing (mainly), and embarrassed cardiac and respiratory action. In cerebral disease, particularly bilateral softening in the neighborhood of the internal capsules, these symptoms are due to the destruction of the motor fibres destined to these nuclei (pseudobulbar paralysis). The root of the vagus may be the source of these symptoms in like combination from basilar meningitis, tumor, or aneurism of the vertebral artery.

In the neck the nerve-trunk accompanying the carotid, or lower down winding over the subclavian, may be compressed by an aneurism or tumor, or damaged in operation. This nerve is involved in toxic or infectious neuritis more frequently than is ordinarily thought. The affection of the pneumogastric nucleus or trunk may be of a degree to induce irritative symptoms (slowness of the heart's action, spasm of the larynx, and vomiting), or paralytic symptoms (paralysis of the larynx, embarrassed respiration, and rapid pulse).

The inferior or recurrent laryngeal nerve, branching from the tenth at the base of the neck, winds around the great vessels—the aorta on the left, the subclavian on the right side—and ascends back of the trachea to the larynx, of which it supplies the most important muscles.

IX. ELEVENTH NERVE.

The accessory fibres of the spinal accessory join the vagus nerve, of which they form mainly the recurrent laryngeal branch. The spinal portion, composed of several motor roots of the cervical cord, forms part of the cervical plexus and supplies the sternomastoid and the upper portion of the trapezius muscle. Spasm of these muscles causes torticollis. Paralysis of the muscles supplied by the spinal accessory results from degeneration of the cervical gray matter in progressive muscular atrophy, from lesion of the trunk, in meningitis or brain tumor, and from wounds, tumors, vertebral disease, etc., in the neck. Paralysis of the sternomastoid alone may result from a wound of this muscle, severing the nerve-trunk within it. In paralysis of one spinal accessory, the head cannot be turned to the other side, the sternomastoid and the upper border of the trapezius are relaxed and in time wasted, and all movements about the shoulder, as raising the arm, are embarrassed. When this paralysis is bilateral the head falls backward or forward, according as the sternomastoid or the trapezius is more affected.

Bilateral paralysis of the spinal accessory is conspicuous in meningitis, especially the tuberculous form of childhood, and in progressive muscular atrophy. Lesion of the nerve at the base of the brain, including the accessory part (laryngeal fibres), paralyzes the vocal cords, and is likely at the same time to implicate the hypoglossal or the glossopharyngeal and paralyze the tongue or the palate.

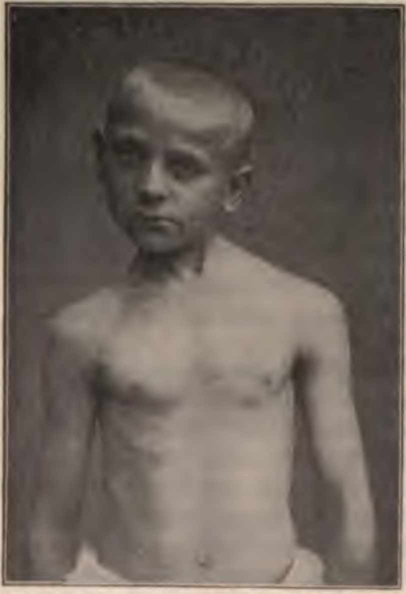

FIG. 390.—Torticollis (Jochimsthal).—Young.

Torticollis or Wry-neck—Accessory Spasm.—True torticollis is a deviation of the head due to abnormal action of the muscles supplied by this nerve. It may be a fixed deformity—congenital wry-neck—or due to spasm—spasmodic wry-neck. In congenital torticollis there is atrophy of neck muscles, principally the sternomastoid, in consequence of prenatal poliomyelitis or of injury to the muscles during labor. Contraction of the sternomastoid tilts the head toward the affected side and at the same time rotates the face toward the opposite side. The muscle stands out rigid.

Spasmodic torticollis is of the nature of *facial tic*, and like it may be either tonic or clonic. The position of the head ordinarily is governed by the sternomastoid, but in some cases there is backward tilting in consequence of contraction of the trapezius. When the affection is bilateral the trapezii draw the head backward,—retrocollic spasm,—aided by both sternomastoids, and the frontales muscles in association raise the eyebrows. Spasmodic torticollis appears usually in middle life, is more frequent in women, and has been ascribed to a variety of causes. Its source in typical cases is probably cortical. It may be ushered in by pain and stiffness about the neck, but as a rule the spasm sets in gradually. It generally centres in the sternomastoid and may be confined to it, but the trapezius of the same side and the splenius of the other are commonly associated with the sternomastoid in spasm. The head is tilted sidewise and slightly backward and twisted to the other side, more frequently the left.

In bilateral (retrocollic) spasm the face is turned upward and the eyebrows raised synchronously. Ordinarily with the tonic variety of torticollis, as it becomes intense, clonic spasms are associated. The intensity varies, and intermissions are frequent. The affection may involve various muscles of the arm or face. The affected neck muscles hypertrophy in time.

The diagnosis is obvious. The rotation of the head to one side and its slight inclination to the other side, on which the sternomastoid muscle stands out prominently, especially when this position is emphasized by clonic jerkings, cannot be mistaken. So-called rheumatic torticollis, "stiff-neck," is marked by its acute appearance, often after exposure, with lameness and tenderness of the neck muscles. "False torticollis" is an unnatural position of the head from gross disease in the neck, most frequently of vertebræ, as Pott's disease and spondylitis deformans. In these conditions the sternomastoid is prominent on the side to which the head is turned. Hysterical torticollis occurs in younger persons with other signs of hysteria. True torticollis is more common in middle life.

Congenital torticollis may be relieved by operation. Spasmodic torticollis is chronic and intractable. Often after increasing for years it becomes stationary. There are cases in which remissions are frequent and intermissions occur, sometimes lasting many months. The disease is of no consequence beyond annoyance and embarrassment. Patients sometimes complain of fatigue or pain in the affected muscles.

X. TWELFTH NERVE.

The hypoglossal nerve, governing the muscles attached to the hyoid bone, controls the movements of the tongue. Within the cranium and in the upper part of the neck it is near the pneumogastric and spinal accessory nerves, with which it often is associated in disease, and the lips have some nuclear connection of movement with the tongue. In bulbar paralysis, acute and chronic, the hypoglossal nuclei are a focus of the disease; and they occasionally are degenerated in tabes and paresis. In hemiplegia the cerebral—supranuclear—fibres for the tongue are commonly included in the lesion between the lower part of the motor cortex and the hypoglossal nucleus in the medulla. The roots in the medulla may be damaged by hemorrhage or by tumor, which usually implicates the main pathway to the opposite arm and leg; or the roots emerging from the medulla may be involved, often with the tenth and eleventh, in meningitis, syphilis, or tumor. In the upper part of the neck various gross diseases or wounds may injure the hypoglossal trunk with the spinal accessory.

Paralysis of the tongue without sensory dist
hypoglossal lesion. When this is bilateral the tong
paralyzed on one side the tongue, protruded, cu
side, speech is thick, and chewing is awkward.
wastes, shows fibrillary tremors, and is puckere
When the lesion is nuclear, as in bulbar palsy, the
when it is cerebral the tongue shows no trop
particularly with double hemiplegia, paralysis of
bulbar symptoms, constitutes "pseudobulbar pa

Paralysis of the tongue from cerebral diseas
hemiplegia. Nuclear palsy is generally bilateral
palsy," recognizable by its combination of par
particular of the tongue. The similar combinati
bulbar paralysis is not associated with atrophy n
plegic weakness. Paralysis of one-half of the to
arm and leg—a form of crossed paralysis—indicate
at the level of the hypoglossal nucleus on the sid
paralyzed and wasted. Paralysis of half the tongu
and associated by implication of the spinal acc
the palate and vocal cord on the same side (Hu
to lesion of the nerve within the skull or in the
The prognosis depends on the seat and character
it is unfavorable; the likelihood of improveme
syphilitic cases.

Spasm of the tongue is an incident of the ep
chorea. It occurs also as a rare phenomenon in

DISEASES OF THE SPINAL

I. SPINAL MENINGI

Two varieties are usually mentioned—lepto
of the pia and arachnoid, and pachymeningitis, in
Syphilis causes a meningomyelitis in which b
involved. Tuberculous meningitis of the cord i
ciated with the same affection of the brain. A
by spinal caries, and in these cases the cord as
is involved. Cerebrospinal fever is an acute
involves the membranes of both the brain and
tion called hypertrophic pachymeningitis, especia
in which the dura is much thickened and the cord
is sometimes caused by trauma, but in many c
clear. Purulent meningitis of septic origin is c
an infection extends from the membranes of the
media; or from the pelvis, as in the puerpe
infection occurs.

Pathology.—The membranes are congested
times covered with a fibrinous or purulent exuda

often implicated, and the cord itself may be involved in various degrees. The offending microbe can often be isolated after a lumbar puncture.

Symptoms.—Irritation of the nerve-roots is an early symptom; hence there are pain, stiffness of the back, opisthotonos, contractures of muscles, and even slight clonic spasms. Kernig's sign is usually present. Later in the case there may be pressure symptoms, as paralysis and anæsthesia. In very acute cases there may be chill and fever.

Pachymeningitis cervicalis hypertrophica is an affection almost *sui generis*, especially as caused by trauma. It may closely resemble syringomyelia. There is flaccid atrophic paralysis of the shoulders and arms, spastic paralysis of the lower limbs, and, in some cases, the dissociation symptom, that is, abolition of the pain and thermal senses with preservation of the tactile sense. There may also be pain and stiffness about the neck.

Diagnosis.—It is scarcely possible to distinguish a pure meningitis from a meningomyelitis; the symptoms of disease of the membranes are likely to be associated with some evidence of implication of the cord. Where symptoms of irritation predominate, as pain, stiffness, hyperæsthesia, etc., we may suspect that the membranes are the more concerned. Later, when paralysis and anæsthesia with incontinence appear, we interpret these symptoms to mean that the cord is involved. The eccentric pains, felt at points in the chest or abdomen, may suggest some deep-seated visceral disease, but the diagnosis can usually be made from the associated symptoms. The determination of syphilis as a cause of meningitis is always of first importance. The history of the case may point that way, but it is not always to be relied on, especially when it is negative. The Wassermann test is more nearly conclusive.

II. MYELITIS.

This term should be restricted to true inflammation of the spinal cord. In the past, however, it has been loosely used for a variety of lesions, such as softening and the destructive effects of trauma. When the term is properly restricted it will be found that genuine myelitis is not a common affection.

Etiology.—This disease is doubtless due in every case to some form of infection or toxæmia. The old ideas that it was caused by exposure to cold, to worry, to sexual excesses, and other such far-fetched notions are no longer credible. At most, cold can act but as a predisposing cause. It is even doubtful whether alcohol causes myelitis. Syphilis undoubtedly causes a meningomyelitis, but this is such a distinct affection that it is treated under a separate heading. Among the causes assigned are the infectious diseases, such as septic infection, varicella, gonorrhœa, and measles. It is claimed that malaria may cause it. Typhoid fever and smallpox may cause multiple neuritis, which might be mistaken for myelitis by a careless observer. In fact, these two latter diseases must be carefully distinguished. A destructive myelitis may be caused by spinal caries of tuberculous origin, but otherwise tubercle does not often attack the cord. Injury may cause extensive lesions in the spinal cord, and these may become secondarily infected, but they are not primarily inflammatory, nor do they become so in every case. Cancer of the vertebra may also cause myelitis. There are also special forms of myelitis, such as the anterior poliomyelitis, or inflammation of the anterior horns of the gray matter, but these affections are described apart. Finally, cases occur in which acute transverse softening is found post mortem, suggesting the idea of acute infection, but the cause is obscure. In fact, not a few cases of myelitis, and myelitic softening, cannot be satisfactorily accounted for.

The spinal membranes are often involved in cases of myelitis, so that in effect the condition is one of meningomyelitis, and the cause, whatever it be, may act primarily on the membranes.

Pathology.—This disease may be transverse, focal, disseminated, or diffused. In transverse myelitis the lesion implicates the whole thickness of the cord, but it may be comparatively limited in its upward and downward extent; in fact, not more than one, two, or three segments may be involved. Focal myelitis, described by some authors, is much more rare; as the name implies, the lesion is discrete; and in the disseminated variety there are more than one such lesion scattered in various places in the cord. Diffused myelitis is merely that variety in which the inflammatory process is more widely and continuously extended. The inflamed area may be variously discolored; in some cases it is congested and bright red or pink, in others rather yellowish, in others white. Its consistence varies, but it is usually softer than the normal cord, and it may be so soft as to flow out under the knife. This is the condition often called "white" or "red" or "yellow" softening. The color is merely due to the elements of the blood contained in the softened area. The tissue is usually necrotic, but it is not necessarily purulent. Pus-cells may, however, be found. The membranes may or may not be involved, congested, and thickened, and there are cellular infiltration and thickenin oı the blood-vessels.

Symptoms.—It is best to describe the symptoms of myelitis according to the level at which the lesion is located.

If the lesion is in the *cervical region*, all the functions of the cord below that point may be partly or entirely involved. There will be spastic paralysis in the lower limbs, exaggerated knee-jerks and other reflexes, ankle clonus, and Babinski's reflex; incontinence of urine and fæces, if the lesion is transverse or even extensive; anæsthesia to all modes of sensation; possibly bed-sores; in the upper limbs also spastic paralysis, unless the anterior horns of gray matter in the cervical enlargement are involved, in which case there may be, especially in chronic cases, extensive muscular atrophy, even with fibrillation, in the shoulders, arms, and hands, with flaccid paralysis. Anæsthesia is also more or less complete in the trunk, arms, and hands, and the muscles of respiration may be involved. If the lesion extends to or above the fourth segment there is danger of death from involvement of the phrenic nerve. There may be a girdle sense in the neck or upper part of the chest. As a rule, pain is not an urgent symptom, unless the membranes and nerve-roots are implicated. The pupils may be dilated or contracted, according as the oculopupillary centre is irritated or paralyzed. A total transverse lesion abolishes all functions below its level, but such a lesion in the neck is seldom compatible with prolonged life.

In the *dorsal region* the lesion causes symptoms in the trunk and lower limbs only. There is likely to be a spastic paraplegia, sometimes with contractures, exaggerated reflexes and clonus, Babinski's sign, incontinence of urine and fæces, anæsthesia to all modes of sensation, and a girdle sense about the trunk, or pain radiating through the chest or abdomen, according to the exact level of the inflammation. There may be a zone of hyperæsthesia marking the upper limits of the lesion and caused by irritation of the membranes or nerve-roots. Bed-sores of an aggravated type may form. If the lesion is totally transverse these symptoms are absolute; if, however, the lesion involves only certain structures of the cord, the symptoms may vary within wide limits. Thus, motion may be more involved than sensation; control of the bladder may not be entirely lost; the patient may be able to walk with a weak and spastic gait; and pain may not be urgent. In spinal caries the paralysis is often more motor than sensory, due to the bone lesion being located in front of the cord.

In the *lumbar region* myelitis causes an atrophic or flaccid paralysis of the lower limbs, due to the fact that the anterior horns of gray matter are involved. There is also paralysis of the bladder and rectum in grave cases, bed-sores, pain in the legs, loss of sensation in the lower trunk and lower limbs, and abolished knee-jerks. But here as elsewhere the symptoms will vary somewhat with the extent of the lesion.

The course of myelitis may be acute or exceedingly chronic. The patient may recover up to a certain point, and then remain more or less permanently crippled. Entire recovery is rare. A fatal result is not uncommon. In the myelitis of Pott's disease a good recovery is sometimes obtained.

Diagnosis.—Myelitis is to be distinguished especially from locomotor ataxia, syringomyelia, and multiple neuritis. It may also closely resemble some forms of syphilis of the cord. When it is due to trauma or spinal caries the cause is usually apparent.

From locomotor ataxia it is distinguished by the mode of onset, which is usually much more abrupt. There is absence of the fulgurant pains, the peculiar ataxic gait, the swaying with closed eyes, the abolished knee-jerks (unless in lumbar myelitis, in which case, however, there is flaccid paralysis and muscular atrophy in the lower limbs, very different from tabes). There is also absence of the Argyll-Robertson pupil, with optic atrophy; and the paralysis of the bladder and bowel is much more complete and of earlier onset than in locomotor ataxia.

In syringomyelia the symptom-complex is quite different from ordinary myelitis. There is especially the dissociation symptom, in which the temperature and pain senses are abolished without impairment of tactile sensation; also scoliosis, arthropathies, and vasomotor changes. The onset is also more gradual than in myelitis; the bladder and bowel are not so likely to be involved. Still, some cases of traumatic meningomyelitis of the cervical cord closely resemble syringomyelia, and are distinguishable only with care. The history of trauma in these cases is significant.

In multiple neuritis the symptoms are distinctly peripheral. The nerve-trunks are involved, and are often painful on pressure. The muscular masses, especially the calf, are exquisitely sensitive. The paralysis is flaccid, and the muscles waste. The reactions of degeneration may be present. The deep reflexes are abolished. The anæsthesia, while present in varying degrees, is not always seen in such extensive areas as in myelitis. The bladder and bowel, as a rule, are not involved. There may be mental symptoms—the so-called Korsakoff's psychosis—and there is a history of exposure to alcohol, to lead, to arsenic, or to diphtheria. It is often difficult to say whether or not a myelitis is due to syphilis, but in some forms of syphilis of the cord the type is quite distinct. In some cases of cord-syphilis, there may be, or may have been, some involvement of the cerebrum or cranial nerves. Lumbar puncture may throw some light on the causation and pathology of myelitis.

The constitutional reaction in myelitis at the beginning is not marked, except in the acute anterior poliomyelitis of children. There may, however, be some fever and weakness of the pulse. In advanced cases, with bed-sores and incontinence of urine, symptoms of sepsis may occur. Infection of the bladder is always a grave complication. It may be due to the use of the catheter.

III. ANTERIOR POLIOMYELITIS.

Inflammation of the anterior horns of gray matter in the spinal cord may occur as an acute or a subacute disease. The acute form is usually a disorder of childhood, although adults are not entirely exempt. In children it is called *infantile paralysis*. The subacute form is rare, and is usually seen in adults. Progressive muscular atrophy, or chronic anterior poliomyelitis, is also a disorder of adults, but its inflammatory nature may be doubted. It is a slowly progressive degeneration of the ganglion cells in the anterior horns.

The cause of acute anterior poliomyelitis has now been clearly demonstrated to be an infection or toxæmia. Infantile paralysis has been known to prevail as an epidemic, as in the instance reported by Medin, in which 44 cases occurred within a few weeks in one town in Sweden. Epidemics have been observed in the United States. A series of 126 cases occurred in Rutland, Vt., in 1894. It has prevailed epidemically in many parts of the world in the last 25 years. Among the great epidemics have been those in Norway, in Vermont, in Queensland, in Vienna, in Wisconsin, in Westphalia, and in Nebraska.[1] An epidemic, or very prevalent type, of the disease occurred in New York City in the summer and autumn of 1907, and the affection has since been more common than usual in other parts of the United States. Isolated cases sometimes follow the infectious diseases, as measles, scarlet fever, whooping-cough, and diphtheria.

We owe to the researches of Flexner and Lewis[2] and the later experiments of Flexner and Noguchi[3] the demonstration of the infectious nature of epidemic anterior poliomyelitis and its exciting cause. The microörganism consists of filterable globoid bodies measuring from 0.15 to 0.3 μ in diameter and arranged in pairs, chains or masses. Inoculation into the brain or the sciatic nerve and peritoneal cavity of monkeys is followed after an incubation period of forty-eight hours to several weeks by the clinical symptoms and pathological effects characteristic of experimental poliomyelitis.

Pathology.—Flexner says that the vascular lesions are the primary causes of the lesions of the central nervous system. In the early stage there is hyperæmia of the horns and some congestion of the cord; the ganglion cells are swollen and indistinct; the vessels are engorged, and white cells are seen migrated from them. Later the ganglion cells are atrophied or even entirely destroyed; the nerve fibrils are obliterated, and the horns are reduced in size. The disease may be located in either the cervical or lumbar enlargement. In the subacute form the lesions may be found more extensively spread; the anterior horns in many parts of the cord are involved, and in some cases the neighboring white matter is invaded.

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[1] Collins (Trans. Ass. Am. Physicians, Vol. XXV, p. 99) gives statistics of the various epidemics in tabulated form.

[2] Flexner and Lewis's articles are in vols. 53, 54 and 55 of the Journal of the Am. Med. Ass.

[3] Flexner and Noguchi. Trans. Ass. Amer. Physicians, Vol. XXVII, 1913.

Symptoms.—Infantile paralysis begins abruptly, with constitutional reaction. There is fever, sometimes moderate stupor and delirium, and convulsions may occur. Some days may elapse before the true nature of the disease is recognized, and then it is found that the child is paralyzed in one or more limbs. This paralysis is flaccid, with abolished reflexes. Pain is not prominent and may even be entirely absent; but in rare cases it may be severe in the early stages. The initial palsy may involve the whole limb, but as time passes a partial recovery takes place, until finally the paralysis is located, and remains stationary, in a few muscles or a group of muscles. There is no anæsthesia in the paralyzed part. The bladder and bowel are not involved. Infantile palsy is a common cause of club-foot, the type of deformity depending upon the group of muscles paralyzed. The reactions of degeneration are present, and the muscles do not show fibrillation. In the arm the muscles oftenest damaged are the deltoid, biceps, brachialis anticus, and supinator longus. Deformity of the hand is not common. The paralyzed limb does not grow normally, but remains partially stunted and even shortened. In the recent epidemic in New York bulbar symptoms were sometimes seen, even in mild cases in which recovery ensued. Meningeal symptoms, such as pain, rhachialgia, and rigidity, were marked, and sometimes there was photophobia. Nothing was found in the cerebrospinal fluid or in the blood to show the cause, although the disease had some resemblance to epidemic cerebrospinal meningitis. According to Harbitz and Scheel, the brain and bulb were often involved in epidemics in Norway.

Wickman has attempted to establish various types of the disease, such as the spinal poliomyelitic type, the type of ascending or descending palsy (Landry's paralysis), the bulbar and pontine type, the cerebral (encephalitic) type, the ataxic type, the polyneuritic type, the meningeal type, and abortive types. But the spinal type is the predominating one; all other forms are rare varieties.[1]

The subacute form of this disease is also a grave affection. Duchenne recognized an *ascending* and a *descending* type. In the former the disease begins in the lower extremities, and later invades the upper limbs; in the latter the course is the reverse; but some of Duchenne's cases may have been instances of Landry's disease. The paralysis is characteristic of a lesion of the anterior horn; the muscles become atrophied, and they may show fibrillation. Bramwell points out that the paralysis precedes the atrophy, that the reflexes are abolished, and the reactions of degeneration are present. In cases in which recovery occurs there may be permanent paralysis in some muscles. Sensory and bladder symptoms are wanting. Bulbar symptoms have been reported. The disease is rare, and it may not be due to the same infecting agent as the acute form. Flexner suggests that there may be more than one form of the infection.

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[1] Report of the Collective Investigation Committee, N. Y. Epidemics, pp. 50–51.

Diagnosis.—Infantile paralysis in its initial stage may be mistaken for an acute febrile infection, or infantile convulsions, and the paralysis may be overlooked for several days. After the paralysis is noted the disease is not likely to be mistaken. The flaccid palsy, the abolished reflexes, the onset of wasting, all coming on after the symptoms of an acute infection, without anæsthesia, and limited to one limb or part of one limb, are unmistakable. The disease may also resemble multiple neuritis, which is sometimes seen in children; but multiple neuritis is more wide-spread and symmetrical, pain in the affected limbs is more common, the cause may be traced in some poison, and complete recovery may occur. The subacute form in adults bears a resemblance to multiple neuritis, but the fibrillation in the muscles, and the absence of sensory symptoms and of pain on pressure over nerve-trunks and muscles, serve to distinguish it. The reactions of degeneration may not be so promptly established as in multiple neuritis. The resemblance of poliomyelitis to Landry's paralysis may be close; but the latter is a more acute affection, and the tendency to a fatal ending is more marked. Fibrillation is not seen in Landry's disease, but neither is it reported in all cases of inflammation of the anterior gray horns. The two affections have not a little in common. In myasthenia gravis there is the rapid exhaustion on exertion, and the affected muscles do not atrophy or show fibrillation. In all these three affections, namely, poliomyelitis, Landry's paralysis, and myasthenia gravis, the exact causation and pathology have points of resemblance; they seem to depend on a poisoning of the motor neurons, or, in the case of myasthenia gravis, on some affection of the muscular fibres, and the motor symptoms are not altogether dissimilar. Poliomyelitis may resemble cerebrospinal meningitis, and has been confounded with it. A striking difference is seen in the seasonal prevalence, which for epidemic poliomyelitis is midsummer, and for the other disease is late winter and early spring. Flexner points out changes in the cerebrospinal fluid in poliomyelitis which may have diagnostic significance. The fluid first contains a number of small cells hardly larger than lymphocytes, but showing a polyform nucleus, a few lymphocytes and red cells. Later the white cells increase; at the end of 72 hours a large number of mononuclear cells appear, and the fluid is opalescent. On the day of the paralysis the fluid tends to be only cloudy and contains a mixture of large and small mononuclear cells.

IV. ACUTE ASCENDING PARALYSIS.

This disease was first described by Landry in 1859, and is usually called by his name. It was for a long time depicted as a disease without a pathology, but the more refined methods of recent days have tended to make it out an affection of the peripheral motor neurons.

Pathology.—The pathology of Landry's paralysis is not established. Some observers have found changes in the ganglion cells in the anterior horns of gray matter and in the bulb as chromatolysis and swelling of the axis-cylinder, also foci of inflammation and capillary hemorrhages in the cord and medulla oblongata. Changes have been found in the peripheral nerves. The cause is unknown.

Symptoms.—The disease usually begins as a flaccid paralysis in the lower limbs, and extends upward, involving the muscles of the trunk, the arms, and finally the bulb. Anæsthesia is not present, although there may be some slight dulness or retardation of sensation. The muscles do not waste perceptibly, but in rapidly fatal cases there is not time. The electrical reactions may be preserved. There is no incontinence, as a rule, although exceptions occur. In some cases the initial symptoms are in the bulb and upper extremities—the descending type. The mind is not affected. Fever is not common. There may be hyperidrosis. The course of the disease is sometimes very rapid. Death results from exhaustion and asphyxia. A few recoveries have been claimed.

Diagnosis.—Landry's paralysis resembles a rapidly fatal multiple neuritis more than anything else, except that the sensory symptoms are almost altogether wanting, but if the pathology be proved to be a toxic affection of the motor neurons, it is rather like anterior-poliomyelitis. It is not likely to be confounded with any other disease, unless it be with myasthenia gravis, in which the history and course are different and the exhaustion symptom is marked. It is possible, however, that in myasthenia gravis there may be some involvement of the motor neurons.

V. PROGRESSIVE MUSCULAR ATROPHY.

There is a degenerative process in the spinal cord, usually chronic, which affects chiefly the anterior horns of gray matter, especially in the cervical region. This is the disease called, from its anatomy, chronic anterior poliomyelitis; and, from its clinical form, progressive muscular atrophy.

Pathology.—Chronic anterior poliomyelitis is a destructive process which invades the anterior horns of gray matter. It has the appearance under the miscroscope of being a degeneration of the motor neurons. Thus the large ganglionic cells are shrunken or even destroyed, the interlacing nerve fibrils of the anterior horns are obliterated, and these horns are smaller than normal. But there are cases in which this identical process is found, and in which is seen in addition a degeneration of the lateral, or crossed, pyramidal tracts. Where this association exists the clinical form differs, for in addition to muscular atrophy there is seen a spastic paralysis with exaggerated reflexes. This latter affection has been given a distinct name—amyotrophic lateral sclerosis—and is described in most text-books as a distinct disease. Some neurologists believe these two so-called diseases are merely different forms of the same pathological process. For the sake of convenience and to conform to custom the two affections will be described here separately. These two clinical forms represent disease of the motor neurons: in anterior poliomyelitis it is the neurons of the lower order which are affected, whereas in amyotrophic lateral sclerosis not only the neurons of the lower but also those of the upper order are involved.[1]

Symptoms.—The disease usually begins with wasting of the muscles of the hands, especially of the thenar and hypothenar groups, and the interossei. Later the muscles of the forearms become involved, then those of the upper arms and shoulders. This is called the Aran-Duchenne type. Other forms appear, as, for instance, early wasting of the deltoids, supra- and infraspinati, and biceps—the upper arm type. In some rather rare or advanced cases the muscles of the lower limbs are involved. The

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[1] R. T. Williamson: "Amyotrophic Lateral Sclerosis and Progressive Muscular Atrophy," Edinburgh Med. Journal, April, 1907, p. 304.

[illegible] may also waste. [illegible] deformities [illegible] as the claw hand, or main en griffe, in which the proximal phalanges are overextended and the distal phalanges are flexed, and the [illegible] [illegible] affected. Of the Germans, the [illegible] the [illegible] which is caused by overaction of the long extensors of the thumb, causing the metacarpal bone to be displaced backward [illegible] in the same plane as the metacarpal bones of the fingers. In the [illegible] various forms of clubfoot result. As the wasting progresses [illegible] of power [illegible] and in advanced cases the wasted arms hang powerless at the sides. In the pure forms, that is, in cases in which the lateral tracts are not involved, there is no spastic paralysis, and the reflexes [illegible] the arms or legs are not increased. In advanced cases the reflexes [illegible] affected muscles may be much diminished or even lost, but this is not invariable, for even in very much wasted muscles a sharp tap will sometimes elicit a slight response. The affected muscles are the seat of fibrillary twitchings. The complete reactions of degeneration are not present, although some modal change and partial reaction may be seen in advanced cases. There is no anaesthesia of any kind, nor are the sphincters usually affected. Scoliosis does not occur. In cases in which the neck muscles are greatly wasted there may be head-drop, but this symptom is seen more frequently in amyotrophic lateral sclerosis. As a rule, there are no bulbar or oculomotor symptoms. The disease may be very chronic, lasting for many years. Some aberrant forms are seen, but the above are the most common types. The disease, as a rule, begins in adult life. It is progressive and incurable.

Diagnosis. The distinction has already been made clear between this affection and amyotrophic lateral sclerosis. In the latter disease there is spastic paralysis in addition to the muscular atrophy, and this spasticity with exaggerated reflexes is seen in the wasted arm muscles as well as in the lower limbs, in which there may be little or no wasting. In pure cases of progressive muscular atrophy there is no real spasticity in the affected muscles and no spastic paralysis of the lower limbs. It must be acknowledged, however, that the dividing line between the two diseases is not always sharply defined. In progressive muscular atrophy increased reflexes with some spasticity of the limbs are sometimes observed. The disease may resemble syringomyelia, but is to be distinguished by the absence of the dissociation sensory syndrome, and of the trophic lesions, arthropathies, and scoliosis. From pachymeningitis of the cervical cord it is to be distinguished also by the absence of sensory symptoms and of pressure palsies of the cord. From multiple neuritis it is distinguished by its history and course, the absence of sensory symptoms, the distribution of the muscular atrophy, the absence of reactions of degeneration, the fibrillary twitchings, and the preserved or even increased reflexes. The various muscular dystrophies often resemble progressive muscular atrophy of spinal origin. They usually begin in childhood, however, and the muscles do not present fibrillary tremors, nor are the reflexes preserved. They are sometimes hereditary and more than one case may occur in a family. In some cases of myelitis there are muscular atrophies and spastic paralysis, but the onset is more rapid and the course of the disease more acute; sensory symptoms are present, and there is paralysis of the bladder and rectum. In the advanced stages of locomotor ataxia there is sometimes seen extensive muscular atrophy, but the history of the case, the fulgurant pains, the ataxia, the abolished knee-jerks, the atony of the bladder, the optic atrophy, the Argyll-Robertson pupil all serve to distinguish tabes. So, also, in Friedreich's ataxia there may be some degree of muscular atrophy in later stages of the disease, but the history and the typical symptoms, as ataxia, nystagmus, and the affection of speech, will prevent error.

VI. AMYOTROPHIC LATERAL SCLEROSIS.

Amyotrophic lateral sclerosis may be said to be a progressive muscular atrophy *plus* a lateral sclerosis. The two affections may be syndromes of one and the same pathological process, depending for their differences upon a mere difference in the distribution of the lesions in the cord. Amyotrophic lateral sclerosis was first described by Charcot, and is sometimes called by his name.

Pathology.—The lateral tracts are sclerosed, sometimes throughout their entire length in the cord, the process stopping in the medulla; and Alfred W. Campbell has traced the degeneration as far as the brain cortex, where it is seen especially in the large Betz cells in the motor region—that is, the ascending frontal convolution and paracentral lobule. The degeneration of the anterior horns in the cervical region is marked, and sometimes the gray matter in the medulla oblongata is involved.

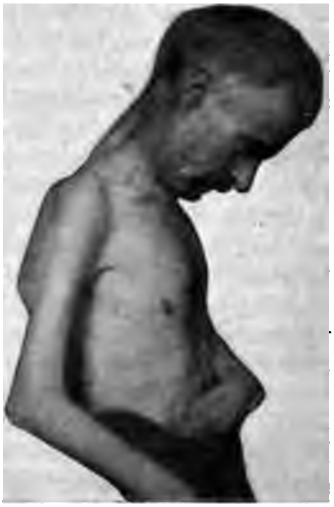

FIG. 391.—Amyotrophic lateral sclerosis, showing head-drop.—Lloyd.

Symptoms.—The disease begins as a muscular atrophy, most marked at first in the hands, but gradually including the arms, shoulders, neck, and even the trunk. In this respect its appearance is like progressive muscular atrophy. The essential muscles of the hands are much involved, and "claw hand" or "monkey hand" may develop; the arms become so powerless that they hang useless at the sides; the muscles of the neck atrophy and permit the head to fall forward on the chest, and when the patient raises the head it goes up into place with a quick jerk somewhat like the closing of a blade of a penknife; in rare cases bulbar symptoms occur, the patient having difficulty in swallowing and the voice having a nasal twang. Fibrillary twitchings are present. The muscular atrophy seldom invades the lower limbs. Along with the atrophy appear the evidences of sclerosis of the lateral tracts. Even in the wasting muscles there is hypertonus, and the reflexes are increased. The paretic arms may be slightly spastic. The biceps-jerks, triceps-jerks, and wrist-jerks are exaggerated. The spastic paresis of the lower limbs is very marked; the patient walks with a feeble but spastic gait, the feet dragging the floor. The reflexes are all exaggerated, such as the knee-jerks and ankle-clonus, and there is seen the plantar extensor reflex of the great toe—the Babinski reflex. There is no anæsthesia, nor any paralysis of the bladder or bowel as a rule.

Diagnosis.—The rules for differential diagnosis are practically the same as in the case of progressive muscular atrophy. These two diseases shade into each other and may be only variants of the same morbid process. Dejerine claims that bulbar symptoms do not occur in true progressive muscular atrophy, and that they are indicative of amyotrophic lateral sclerosis. The latter disease runs a more rapid course than the former. The increases of the deep reflexes, the spastic gait, and the paralysis of the muscles before the onset of atrophy are in favor of amyotrophic lateral sclerosis.

VII. PRIMARY LATERAL SCLEROSIS.

Some authors, as Charcot, Erb, and Dreschfeld, have described a sclerosis of the lateral tracts which appears to be primary, that is, it does not depend upon a focal lesion, and is not associated with degeneration of other structures or tracts of the cord.

Symptoms.—The symptoms are the same as those already described for amyotrophic lateral sclerosis, *minus* the muscular atrophy. There is spastic paralysis of the legs, with "clasp-knife rigidity," in which the limb, when passively extended, resists, then suddenly yields, as in the closing of the blade of a knife. The arms are sometimes involved. The deep reflexes are all exaggerated. There is no anæsthesia, nor paralysis of the bladder or bowel.

Diagnosis.—It is particularly necessary to eliminate every focal lesion from which lateral sclerosis could occur as a descending degeneration, and every disease of other structures than the lateral tracts. The affection is rare, and thus far partakes rather of the nature of a pathological curiosity. Mills has described an ascending hemiplegia which goes by his name, and which has its origin in the cord; the lateral tract of one side is especially involved. It is probably distinct from the affection here described. Spinal syphilis may simulate this disease, especially when the specific lesion is confined to the lateral aspects of the cord. It may also be simulated in the early stages of multiple sclerosis. It is to be distinguished from some degenerative brain and cord affections; as that described by Strümpell, in which spastic paralysis occurred in several members of the same family; and from spastic diplegia in children, the result of focal lesions in the brain, in which cases there is likely to be mental defect.

VIII. LOCOMOTOR ATAXIA.

This disease, also called tabes dorsalis, is characterized by a degeneration of the posterior columns of the spinal cord. It has been recognized only within comparatively recent years, and was first described clinically about 1840 by Romberg, who, however, did not recognize its morbid anatomy. Todd, of England, and Cruveilhier, of France, were among the first to associate it with disease of the posterior columns.

Pathology.—There has been much discussion as to the exact seat of the initial lesion in tabes. Vulpian, Charcot, and others held that it was primarily an inflammation of the posterior nerve-roots. Others, as Marie, Marinesco, and Wollenburg, contend that the ganglion cells in the posterior ganglia are the first involved. Leyden and Goldschneider hold that the starting-point is in the peripheral sensory nerve endings; while still others, as Nageotte, Redlich, and Obersteiner, believe that the disease begins as a meningitis, affecting especially the posterior nerve-roots. Ferrier concludes that none of these theories is satisfactory, but that the essential lesion of tabes is a dystrophy, similar to that induced by certain toxic agents, affecting the sensory protoneuron as a whole. The disease, however, is not confined to the spinal protoneuron, but may affect the optic, the sympathetic, and certain motor neurons.

When fully established locomotor ataxia shows a degeneration especially marked in Goll's columns in the cervical region, in Burdach's columns more or less marked at various levels of the cord, and in the lumbar region in the areas known as the *bandelettes externes* of Pierret. There is a rare cervical type in which the columns of Goll in the neck entirely escape. There is also optic degeneration, and in advanced cases muscular atrophy. The posterior nerve-roots are degenerated, and there is leptomeningitis. Syphilis is assigned by many as the invariable cause of tabes.

Symptoms.—Locomotor ataxia is known by an ataxic gait, loss of static equilibrium, abolished knee-jerks, crises and fulgurant pains, sensory changes, atony of the bladder, loss of sexual power, optic atrophy, and the Argyll-Robertson pupil. Other but rare symptoms are muscular atrophy, arthropathies, and trophic lesions.

The ataxic gait of tabes is its most conspicuous symptom. It is not due to paralysis but to incoördination. There may be full motor power, even an excessive use of power, but the muscles do not act in harmony with the will. The patient walks with the feet well apart; the foot is lifted high from the ground, thrown out widely, and brought down with a stamp, the heel striking the floor first. It is evident that the patient feels the unreliability of his gait, for he watches the floor, and aids his progress by the use of his eyes. Hence he walks with especial difficulty in the dark and in coming downstairs. He may not be able to walk at all with his eyes closed. The ataxia may also be marked in the arms and hands, as in touching the tip of the nose with the forefinger (with closed eyes), unbuttoning his coat, etc. It is also seen when the patient is lying down and attempts to move his legs. It is often an early symptom, but may be preceded by fulgurant pains and changes in the pupils.

The loss of static equilibrium is seen when the patient attempts to stand without support and with his feet close together; but it is much increased when he closes his eyes. He then sways violently and in some cases would even fall. This is the Romberg symptom.

The knee-jerks are abolished early in tabes. This is one of the most constant symptoms, and is the Westphal sign. The other deep reflexes are also lost.

The fulgurant pains are usually an early symptom. They are lightning-like and severe, and felt most in the lower limbs. They may prevent sleep, and are most urgent in their demand for relief. They are usually paroxysmal, and may remit for days and even weeks. Closely associated with these pains are the girdle sense and various crises. The former is felt as a band tied about the waist or abdomen, or about the chest, or even about one limb. The crises are bouts of pain felt in various parts of the body, especially in the epigastric region, but sometimes in the thorax, the larynx, or even the rectum. They may simulate some disorder of one or other internal organ. The laryngeal crises cause a sense of strangling and excite cough. According to Semon the essential cause is a paralysis of the abductors of the vocal cords, and the attacks may begin with a sense of tickling, or even pain in the throat, followed quickly by a sense of suffocation; among accessory symptoms are dizziness, mental confusion, and even loss of consciousness with convulsive movements, but the attacks are in no sense epileptic. Involuntary passage of urine and fæces is occasionally present.

There are various disorders of sensation. Tactile anæsthesia is not always present, or it may be present in only limited areas; in other cases, especially in the advanced stages, it may be extensive. There may be paræsthesia, or altered sense. Thus, the patient may have abnormal feelings in the soles of the feet, causing him to feel as though he were walking on some soft substance, as velvet or mud. There may also be numbness, or formication, or a sense of cold. Alteration of the thermal sense, however, is not usually marked. Analgesia is common: the patient has lost the sense of pain, particularly in the legs; pinching or sticking with a pin is not felt as pain. There is often loss of muscular sense and sense of position; also of the sense of pressure and sense of active and passive motion; and some writers attach great importance to these changes in deep sensibility as the fundamental cause of the ataxia.

Some loss of power in the bladder is often an early symptom; at first there is difficulty in extrusion, later there may be retention or even incontinence. Loss of sexual power is not uncommon; occasionally, in the early stages, there is sexual excitement.

Optic atrophy is frequent in tabes, but its exact frequency is a subject of some debate. Gowers, in 70 cases of posterior sclérosis, found only 9 with this condition; Voight in 52 cases found 9; and Erb in 56 cases found 7. Optic atrophy may appear early, sometimes before the onset of ataxia. It is primary; that is, it is not dependent on a preceding neuritis. It may progress to complete blindness, but its progress is often slow. Changes in the pupils are likely to be early symptoms of tabes. The sympathetic reflex from irritating the skin of the neck is often lost. There may be myosis, sometimes extreme; the pupils are contracted. Sometimes they are unequal, and even irregular in outline. Later in the disease they may be widely dilated. But the commonest change is the Argyll-Robertson pupil.

The exterior muscles also of the eyes are sometimes involved. Thus, there may be ptosis of one or of both upper lids. Other paralyses of the third nerve and of the sixth are also seen, but are not so common.

Among the rarer symptoms of tabes are arthropathies. These may affect the knee, ankle, hip, elbow, or shoulder. A very typical form is that seen in the knee. The joint is the seat of a painless swelling; there is denudation of the articular surfaces, grating, effusion of fluid, osteophytes, and deformity. The joint is relaxed, allowing the knee to be over-extended or bent backward. The whole leg may be enlarged and brawny. In the shoulder and hip an atrophic form of arthropathy is seen; there is preternatural mobility. Sometimes painless fractures occur. A condition known as hypotonia exists: the joints may allow a much wider range of motion than normal. Thus, when the patient lies upon his back, the whole lower limb, straightened at the knee, may be so extended that, in extreme cases, the foot may even rest alongside the neck. A trophic characteristic lesion is the *mal perforans*, or perforating ulcer. This forms on the ball of the foot or great toe; it is deep, painless, and obstinate in healing. Muscular atrophy is seen in some advanced cases of posterior sclerosis, and may be extreme. It is probably dependent on involvement of the anterior nerve-roots or anterior horns, or on a peripheral neuritis.

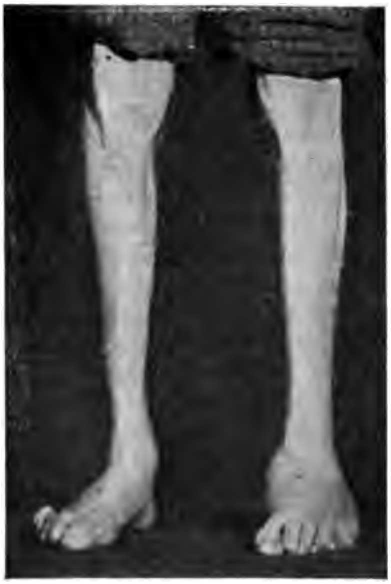

FIG. 392.—Arthropathy of the left ankle-joint in locomotor ataxia.—Lloyd.

The course of tabes is usually chronic. It is a disease of long duration, often extending over many years. In the advanced stages the patient is unable to leave his chair, or even his bed, the victim of painful crises, partially or entirely blind, with incontinence of urine and possibly with one or more arthropathies. There is a *sensory type* of tabes. This is marked by early optic atrophy, proceeding to complete blindness, associated with severe lancinating pains and crises, with lost knee-jerks, but without impairment of gait. This form may persist for many years, finally developing ataxia. Buzzard reported a case which preserved this type for fifteen years, and Gowers mentions one in which optic atrophy had existed for twenty years before the onset of incoördination. This type is peculiarly liable to arthropathies. Locomotor ataxia sometimes coexists with general paresis. It may precede that disease, as is the more common way, or in some cases it may follow it.

There is a juvenile tabes, which is the result of hereditary syphilis. The disease, however, is usually one of adult life, the initial symptoms generally showing themselves between the ages of 30 and 40 years. It is not limited to any race or country, but is seen wherever syphilis abounds; and the statement that locomotor ataxia does not occur in the negro race, is erroneous. It is not so common among women in any race as among men. It has been seen in both husband and wife.

Diagnosis.—Locomotor ataxia is to be distinguished from multiple neuritis by the fulgurant pains, the crises, the pupillary changes, the bladder symptoms, and the absence of true paralysis with atrophy and the reactions of degeneration. There is a *pseudotabes* due to multiple neuritis, in which ataxia is marked, but the history of the case, the flaccid paralysis, with atrophy and electrical changes, the painful nerve-trunks and muscles, as well as the absence of the optic atrophy and pupillary changes, serve to distinguish it. Muscular atrophy with loss of power may occur in advanced stages of tabes, but the other tabetic symptoms and the history of the case should prevent error. From syringomyelia, in which there sometimes occurs an ataxic type. tabes is distinguished by the absence of the dissociation syndrome, of the scoliosis, of the spastic paralysis in the legs with exaggerated knee-jerks, and of the muscular atrophy in the shoulders and arms. Arthropathies occur in both diseases. Optic atrophy is seldom if ever seen in syringomyelia.

Progressive muscular atrophy and amyotrophic lateral sclerosis are hardly to be confounded with tabes. The muscular atrophy, often with preserved or even exaggerated reflexes, and the absence of ataxia, crises, optic atrophy, and the Argyll-Robertson pupil, are sufficient to distinguish the one from the other. From multiple sclerosis tabes is distinguished by the ataxia, which is not the same as the intention tremor of the former disease, in which the movement is jerky, tremulous, and very marked only on voluntary motion. Moreover, in multiple sclerosis there is usually a spastic gait with exaggerated reflexes, and an absence of fulgurant pains and crises; nystagmus and scanning speech are observed; optic atrophy may be present, but only rarely; the Argyll-Robertson pupil is not seen. The painful crises of tabes, occurring in the chest or abdomen, may simulate disease of some internal organ, as angina pectoris or gastric ulcer, or some affection of the bowel. The resemblance is only superficial, and the coexistence of other tabetic symptoms points to the correct diagnosis. The laryngeal crises sometimes simulate laryngismus stridulus, especially in the crowing inspiration at the end of the attack, and even epilepsy, when consciousness is lost and spasmodic movements occur; but the diagnosis is to be made from the associated tabetic symptoms in the pupils, the gait, and the reflexes. In various forms of myelitis there may be a girdle sense, and in inflammation of the lumbar cord loss of the knee-jerks; but usually the knee-jerks are exaggerated, and the optic and pupillary symptoms are wanting. There is spastic paralysis in the legs, sometimes incontinence of urine and fæces early in the case, and the general history is different. Crises and fulgurant pains are wanting. Anæsthesia, clearly delimited at its upper margin, is often present. In the meningomyelitis of syphilis we sometimes see an ataxic paraplegia, in which the incoördination is very similar to that of tabes; but it is associated with a spastic state of the lower limbs and exaggerated knee-jerks.

Tabes may coexist with, or lead up to, general paresis, but the peculiar mental symptoms serve to distinguish the latter disease.

Lymphocytosis is found in locomotor ataxia and in general paresis, and does not diminish with anti-syphilitic treatment, and therefore is useful for differentiating those diseases from cases of pseudo-tabes and pseudo-paresis. It also occurs in sleeping sickness, in tubercular meningitis, in Landry's paralysis, in pernicious anæmia, and in herpes zoster, but Mott thinks it is strong presumptive evidence, and when combined with other facts is an invaluable sign of syphilitic and parasyphilitic affections. He says that spirochætes have not been demonstrated in the fluid.[1]

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[1] Power and Murphy's Syst. of Syphilis, vol. IV, p. 137.

IX. ATAXIC PARAPLEGIA.

This is a syndrome caused by a combined sclerosis of the lateral and posterior columns of the spinal cord.

Pathology.—There is a posterior sclerosis very much as in locomotor ataxia, and in addition a sclerosis of the lateral columns of the cord, but more especially of the crossed pyramidal tracts. Occasionally the direct cerebellar tracts are also involved; but it is rather rare for other parts of the anterolateral columns to be invaded, as, for instance, Gowers's tracts or the direct pyramidal tracts. The disease seems to be a system disease, confining itself to certain definite tracts, and not a diffused myelitis. There is a form of syphilitic meningomyelitis, however, which closely resembles combined sclerosis both clinically and anatomically: in this form, the inflammation is located in the lateral and posterior aspects of the cord; but the membranes are involved first and the posterior and lateral columns are affected secondarily.

Symptoms.—Because of its morbid anatomy it is easy to understand that ataxic paraplegia partakes of the nature both of locomotor ataxia and spastic or primary lateral sclerosis. This idea, however, requires some qualification, for those two diseases cause some contrary symptoms, such as lost knee-jerks by the one and exaggerated knee-jerks by the other, and the two symptoms cannot coexist in the same person. As usually seen, the share of the disease contributed by the posterior sclerosis is the ataxia. There may be some other tabetic symptoms, as lancinating pains, sensory changes, and optic atrophy, but they are rare. Except for the ataxia the disease takes its form largely from the sclerosis of the lateral tracts. There is a spastic gait, with exaggerated reflexes, and these with the ataxia produce a rather confusing picture.

There is a type of the disease in which the tabetic symptoms predominate, and there is then seen the ataxia with lost knee-jerks, fulgurant pains, and bladder weakness, combined with some loss of power due to the lateral sclerosis. According to Oppenheim this predominance of the ataxia and other symptoms of posterior sclerosis is likely to appear in the more advanced stages.

Some authors claim that the muscle tonus is lowered, or at least not increased. This may be so in cases in which the posterior columns are the more involved; but in many cases, and especially in advanced stages, there is increased tonus along with the spastic paraplegia. The type of the case, whether more ataxic or more paraplegic and spastic, will depend upon which region of the cord is more affected.

Diagnosis.—The disease is not likely to be mistaken for any other. The combination of ataxia with a spastic gait is distinctive. Multiple sclerosis may be simulated by ataxic paraplegia, but in multiple sclerosis there is not a true ataxia, but rather an intention tremor, with nystagmus and speech defects. From some forms of spinal syphilis the distinction is not always easy. Diffused myelitis may also resemble combined sclerosis, but the symptoms are not so characteristic of a system disease. The ataxia closely resembles that of tabes dorsalis, but the spastic paraplegia, with exaggerated knee-jerks, marks the difference.

X. HEREDITARY ATAXIA.

This affection, also called Friedreich's disease, is a family rather than an hereditary disorder. It is often seen in several brothers or sisters, but is seldom directly hereditary. It is due to degeneration of several tracts of the spinal cord, especially the posterior columns, but the lateral tracts, and sometimes the direct cerebellar tracts and Clarke's columns, may also be involved. The affection of the posterior columns seems to give the disease most of its individuality.

Symptoms.—The patient has an ataxic or staggering gait, in which he keeps his feet far apart and sways his body violently. The gait is not identical with that of locomotor ataxia; the stamping is not so marked, the swaying of the body is more conspicuous, and the progression has something in it that even suggests a cerebellar lesion. There may also be violent ataxic movements while sitting or even reclining. The swaying is not particularly increased by closing the eyes. Later there are ataxic movements in the arms. The knee-jerks are abolished. Anæsthesia is not present, unless in the advanced stages, and then not always. True paralysis of the legs occurs also in advanced stages, and there may even be marked muscular atrophy. Scoliosis sometimes occurs and a form of club-foot with characteristic over-extension of the great toe. In some cases a Babinski reflex has been seen. The fulgurant pains and crises of true tabes are not seen, nor is there paralysis of the bladder. Especially characteristic are the nystagmus and speech defects. The former is usually of the lateral variety. The speech is slow, labored, staccato, or scanning. Optic atrophy and ophthalmoplegias do not occur. The disease begins in childhood, as a rule, although in a few cases it has not appeared until early adult life. It is an incurable affection, and steadily, but sometimes slowly, progressive.

Diagnosis. — From locomotor ataxia Friedreich's ataxia is distinguished by its early onset, its family association, its freedom from crises and bladder atony, its exemption from optic atrophy and ophthalmoplegias, its nystagmus, and its speech defects.

Multiple sclerosis may simulate this disease, but it is rare in early childhood. It presents spastic paralysis with exaggerated knee-jerks, intention tremor, sometimes an optic atrophy, and it is not seen as a family affection.

In cerebellar ataxia there may be exaggerated knee-jerks, optic atrophy, ataxic speech, and a more distinct cerebellar gait than in Friedreich's disease. Still these two affections have much in common.

The ataxic movements in this disease may simulate chorea and various kinds of tremors; but the association with the other symptoms is usually enough to prevent error. These movements have not the rhythm of tremors, and they are not of the involuntary nature of choreic jerkings. In chorea the movements persist while the patient is at rest; in Friedreich's disease they are seen only during voluntary motion.

XI. SYRINGOMYELIA.

This term is applied to a process in the cord which results in the formation of a cavity. This process is a gliomatosis, or proliferation of a gliomatous tissue which breaks down in the centre. It is not a mere dilatation of the central canal of the cord, for this canal may not be included in the cavity, as was first pointed out by Simon in 1875. Dilatation of the central canal may result from other conditions, and is then properly called hydromyelia.

Pathology. — The overgrowth of gliomatous tissue is usually most marked in the gray matter. When this tissue breaks down, the resulting cavity is of greater or less extent. It may extend across the cord almost symmetrically on either side; in other cases it tends to follow one or other horn of gray matter. The resulting injury to the structures of the cord may be extensive. The anterior horns are involved, the lateral tracts below the lesion are degenerated, and the posterior horns may be almost cut off from the rest of the cord; but the posterior columns may largely escape. The location of the central canal is usually marked by a collection of ependymal cells on the anterior border of the cavity. The walls of the cavity are sometimes lined with a sort of membrane formed from gliomatous tissue. The lesion may be largely located in the cervical region, the cavity extending upward and downward in various shapes and to various levels. In a few cases the dorsal cord is most involved; in others, the lumbar cord. The cord at the seat of lesion is often flattened and ribbon-like.

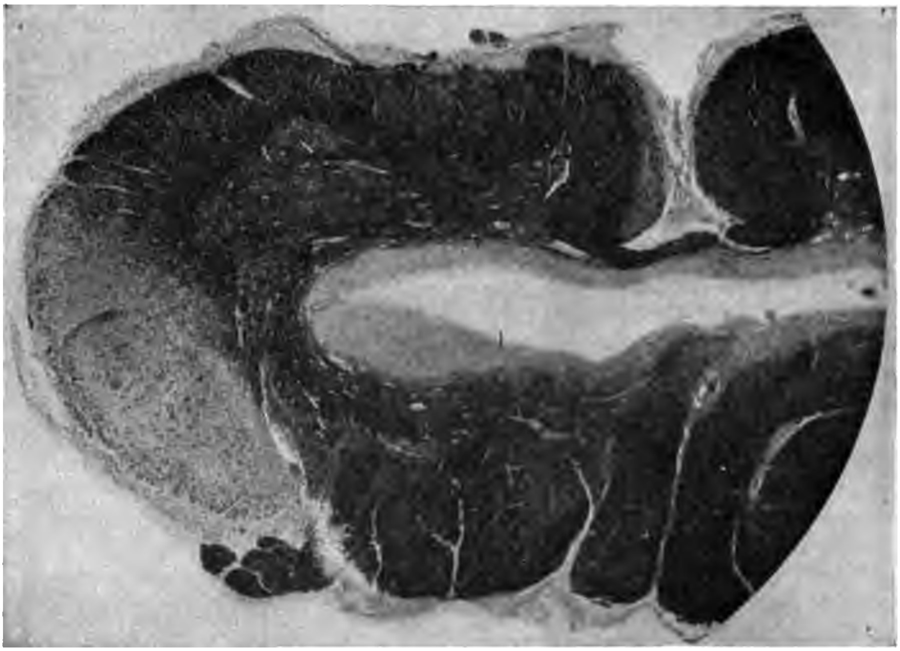

FIG. 393.—Syringomyelia in the cervical region.—Lloyd.

Symptoms. — There is degeneration of the anterior horns. If the cavity is in the cervical cord, there results extensive muscular atrophy of the shoulders and arms with a flaccid paralysis and fibrillary tremors. Occasionally, however, the type of paralysis in the wasted limb is spastic, and the reactions of degeneration are wanting. The degeneration of the lateral tracts causes a spastic paralysis of the lower limbs, with exaggerated knee-jerks. The paralysis in rare instances is hemiplegic, and in some cases ataxia has been seen. The sensory symptoms are most characteristic: they constitute the so-called *dissociation* syndrome. There is loss of the sense of heat, cold, and pain, with preservation of the tactile sense. The location of this syndrome depends upon the seat and extent of the lesion: it may be most marked on the trunk, but it is also seen on the extremities. It may also be more marked on one side, and it occurs in areas of various extent. Occasionally some areas of tactile anæsthesia are also found. Trophic lesions occur. Maculæ appear on the skin of the legs; the toe-nails are enlarged and thickened, with transverse ridges. In the type known as Morvan's disease there are painless destructive lesions of the fingers. Scoliosis is not uncommon; and occasionally an arthropathy of one or other joint is seen just as in locomotor ataxia. Among the rarer symptoms are paralysis of the muscles of respiration, bulbar and oculomotor symptoms, paralysis of the vocal cords, nystagmus, painless fractures, and various skin eruptions, such as urticaria and pemphigus. Bladder symptoms are usually not present.

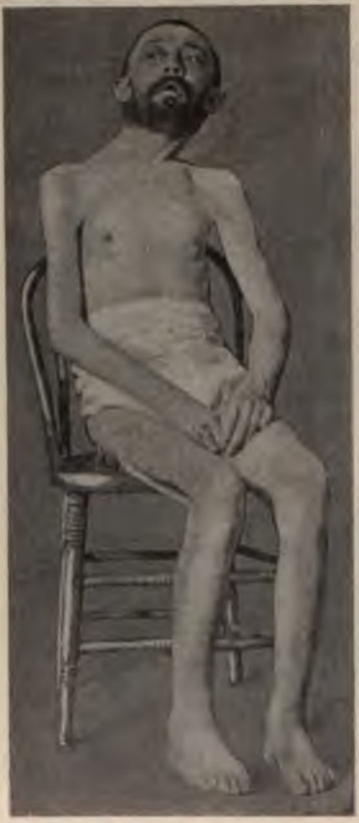

FIG. 394. — Syringomyelia, showing scoliosis, muscular atrophy of the shoulder, and arthropathy of the ankle. — Lloyd.

Diagnosis. — Syringomyelia is to be distinguished especially from progressive muscular atrophy and amyotrophic lateral sclerosis. To both diseases it bears a resemblance because of the muscular atrophy of the shoulders and arms and the spastic paralysis in the legs; but it differs from both in its dissociation sensory syndrome and in its trophic lesions. The disease known as pachymeningitis hypertrophica of the cervical cord sometimes bears a striking resemblance to syringomyelia, even in its sensory symptoms. There may be the same loss of sense for pain, heat, and cold, with preservation of tactile sense; muscular atrophy of the shoulders and arms; and spastic paralysis of the legs; but in the former disease there is often a history of trauma, and in some cases the stiffness and deformity following upon fracture of the vertebræ are seen.

Morvan's disease is a form of syringomyelia in which there are painless destructive whitlows of the fingers (the *panaris analgesique* of French writers). Morvan claimed that the disease is distinguished, however, from syringomyelia, by the predominance of trophic lesions and the loss of tactile sense; but there may be the same muscular atrophy, scoliosis, and even arthropathy, and the two affections are probably closely allied. This type so much resembles some forms of leprosy that Zambaco and others have even claimed that syringomyelia is a form of leprosy—a curious instance of confusing a resemblance with an identity. There is no central gliomatosis in leprosy, and there are many distinctions, too numerous to mention here, which are described under that disease. The anæsthetic form of leprosy is dependent on a neuritis, not a cord lesion.

Charcot pointed out that one of the trophic lesions of syringomyelia is an enlargement of the hand closely resembling acromegalia. In his case the change was limited to one hand, and was symptomatic of gliomatosis. In some cases the paralysis is largely unilateral, resembling hemiplegia of cerebral origin; but the other cord symptoms distinguish it.

Dejerine contends that Friedreich's ataxia is due to a gliomatous change in the cord; and in a series of 12 autopsies in Griffith's collection of cases cavities were found in three. Doubtless in rare cases in which the posterior columns are much involved and ataxia results, the resemblance of syringomyelia to hereditary ataxia is manifest, but the sensory and trophic lesions of the one, and the speech defects and nystagmus of the other, serve to distinguish them. Thus in syringomyelia the knee-jerks are exaggerated, except in the rare tabetic form; in Friedreich's ataxia they are abolished, while in the latter there are speech defects which are not seen in the former, as well as a familial history in many cases. Nystagmus is most rare in syringomyelia, while it is common in Friedreich's disease. Finally, the dissociation symptom, muscular atrophy, arthropathies and other trophic lesions are all suggestive of syringomyelia; which disease, moreover, rarely begins in early youth.

In the rare cases in which ataxia occurs, especially if there should be a spinal arthropathy and lost knee-jerks, the resemblance to locomotor ataxia may be striking, but the sensory symptoms, the muscular atrophy, the scoliosis, and the other trophic lesions would establish the diagnosis. The knee-jerks are not always lost in the ataxic form.

XII. SYPHILIS OF THE SPINAL CORD AND MEMBRANES.

Syphilis may confine its ravages entirely to the spinal cord and membranes, or it may affect both the spinal contents and the brain. The diffused cerebrospinal syphilis is usually more conspicuous for its brain symptoms, but evidences of involvement of the cord can often be found on close inspection.

Pathology.—The essential lesion of syphilis of the nervous system is an initial endarteritis. The membranes, however, are soon involved, and the resulting meningitis then spreads to the contiguous nervous structures. It is highly characteristic of the syphilitic inflammation to become exudative; there then results a thickening of the membranes, a plastic exudate, and very often a gummatous neoplasm. In some forms the resulting meningomyelitis presents but little exudation and practically no gumma. Thickening and even obstruction of blood-vessels may occur, leading to various degrees of necrosis or softening. In the spinal cord the lesion may be largely confined to the membranes, especially the pia, with only a limited area of peripheral myelitis underneath. This condition is especially seen about the lateral or posterolateral columns. In other cases the disease process is more diffused, without reference to the various tracts and systems of the cord, thus causing various bizarre combinations, and symptoms of disseminated myelitis.

Symptoms.—Because of its irregular distribution and various degrees, the syphilitic process gives rise to manifold and irregular symptoms. In some cases there is merely a meningomyelitis, more or less circumscribed, with some resulting softening. The symptoms are then practically the same as have already been described under the head of myelitis. In the more diffused or disseminated form, the symptoms are irregular. Irritation of the nerve-roots is common, with resulting pains, girdle sense, and stiff back. There may be spastic paraplegia, impairment of the nerve supply to the bladder, and various forms of anæsthesia. Sometimes there is muscular atrophy, and even monoplegia. As a rule, spinal syphilis does not cause a so-called "system-disease." Exceptions to this rule occur, however, especially in the form described by Erb and sometimes called by his name. In Erb's paralysis the lesion is a meningomyelitis of the lateral aspects of the cord. There results a spastic paralysis of the legs, with exaggerated knee-jerks, low muscle tension, that is, without contractures; with weakness of the bladder, and usually no involvement of sensation. In some cases, however, the lesion includes the posterior aspects of the cord, and there is then added an ataxia, possibly with some alterations of sensation. This condition closely resembles ataxic paraplegia.

Another form of spinal syphilis is seen in the gummatous tumor. This may be located at almost any level of the cord, or sometimes in the cauda equina. Its symptomatology will depend upon its location and extent, the same as in any other tumor of the spinal cord.

The clinical picture of spinal syphilis varies; there may be recessions and improvement; at other times, an irregular advance with remissions. Pure "system-diseases" are rare, except in Erb's type; and the affection may especially change under specific treatment. In some cases there may be cerebral symptoms, and involvement of one or more cranial nerves.

Syphilis is now believed by many neurologists to be the sole cause of locomotor ataxia. The lesion in tabes, however, does not always present the type of an exudative meningomyelitis, but it is rather a parenchymatous change, a dystrophy confined to the sensory protoneuron. Nevertheless there are some pathologists, as Nageotte and others, who believe that even in locomotor ataxia the initial lesion is a meningitis affecting the posterior nerve-roots. However that may be, tabes dorsalis is usually described as a distinct disease.

Diagnosis.—From ordinary myelitis it is not always easy to distinguish syphilis of the spinal cord; in fact, the syphilitic lesion may consist largely of a meningomyelitis. Syphilis is likely to cause an irregular distribution of symptoms, but this fact is not of as much diagnostic importance as some writers contend. There are cases of myelitis, or meningomyelitis, with or without softening, in which it is not possible to make a differential diagnosis. The problem is simplified in cases in which there is a clear history of syphilitic infection. In doubtful cases the Wassermann test may be determinative. In cases in which there is some associated cerebral syphilis the diagnosis is more evident.

The same difficulty occurs in cases of Erb's palsy, for this affection closely resembles primary lateral sclerosis (the so-called primary spastic paraplegia), except that in the former the bladder is often involved, slight sensory changes occur, and the muscle tone is less increased. In Erb's palsy there may also be irritative symptoms, as pain and the girdle sense, due to involvement of the posterior nerve-roots. This is particularly so in cases in which the lesion spreads to and upon the posterior columns. We then see a condition of ataxic paraplegia, which is practically indistinguishable from the combined sclerosis which some authors describe as a system disease of non-syphilitic origin. These fine problems in diagnosis cannot always be satisfactorily solved, but in all such cases the history of syphilis in the patient should be carefully sought; and whether this is found or not, the antisyphilitic treatment should be given a fair trial. The Wassermann test may throw light on these cases.

Some authors attempt to make a distinction between locomotor ataxia and syphilitic leptomeningitis extending to the posterior columns. In the latter disease there are sensory symptoms and ataxia closely resembling these symptoms in tabes, but the other distinctive tabetic symptoms, such as optic atrophy, Argyll-Robertson pupil, crises, and arthropathies, are wanting.

XIII. TUMORS OF THE SPINAL CORD.

We include here not only tumors of the cord proper but also tumors of the membranes, for the distinction between them is not clinically possible. We also include tumors of the vertebræ, for although these are sometimes distinguishable at the bedside from intraspinal growths, their clinical features are similar. Tumors of the spinal cord proper are rare; those springing from the membranes are the more common. In 50 cases collected by Mills and Lloyd, the largest number were sarcomata, gliomata, or gummata. In nearly one-half of these cases the tumor was in the cervical region; the dorsal region was involved next in frequency, and then the lumbar region. The cauda equina was also involved in a few cases.

Symptoms.—In most cases the symptoms indicate irritation early of the nerve-roots and membranes, and later pressure on the cord. Hence pain is often an early or initial symptom; it may be intense, neuralgic, persisting for a long time in one region, and may radiate or be located far from the cord (eccentric pain). It may be associated with hyperæsthesia, hyperalgesia, paræsthesia, or even anæsthesia, and there may be a subjective sense of numbness in some localized part, and even a girdle sense. With the pain there may be some stiffness of the spine, and contracture of some or other muscle groups. As the case progresses the symptoms of pressure show themselves; there is paralysis of one or other limb or group of muscles, and anæsthesia is more marked and more extensive. There may be anæsthesia dolorosa, that is, absence of sensation to objective tests, with the presence of pain in the affected part. Paralysis of the bladder and bowel may eventually come on. In fact, we see the symptoms of either a partially transverse or even (in advanced cases) a totally transverse lesion. The distribution of these symptoms will, of course, vary according to the seat of the lesion. In a very advanced stage there will be total paralysis below the seat of lesion, contractures, deviation of the spine, anæsthesia, incontinence, bed-sores, cystitis, alteration in the reflexes, dyspnœa, tachycardia or bradycardia, in cervical cases dysphagia, and even bulbar and ophthalmic symptoms. In some cases there is pain on pressure or palpation over the site of the tumor, and pain on twisting or bending the spine.

Diagnosis.—The mode of onset is often suggestive. The initial symptoms are likely to be irritative, hence pain. This pain may be eccentric and localized in the distribution of one or of a few nerves. Paresis begins also as a localized symptom; it may at first be more marked in one limb or even in one group of muscles. Later the symptoms are more suggestive of compression of the cord, as anæsthesia, paraplegia, incontinence, and bed-sores. In some cases the symptoms are markedly unilateral, causing the so-called Brown-Séquard paralysis, or an approach to it.

From syphilis of the cord it is not always possible to distinguish tumor. In fact syphilis may cause a gummatous tumor. The mode of onset is more suggestive of a neoplasm. The Wassermann test should always be made. The progress of the disease in tumor is rather more persistent and hopeless than in most cases of syphilitic meningomyelitis.

From myelitis and acute softening the distinction is often to be made by the more abrupt onset of these affections. The symptoms of a transverse lesion are much sooner established than in tumor; and the initial pain, so marked in case of neoplasm, may be wanting. Unilateral symptoms are not common in myelitis and softening. In hemorrhage the onset is sudden, often caused by trauma, and the disease reaches its acme in a short time. In spinal caries the bone lesion can usually be detected by inspection. The X-ray may demonstrate the lesion. In very early cases, before deformity appears, it may be possible to elicit pain by jarring the spine, and there may be stiffness of the back, and pain on passive movements of the trunk.

In traumatism the case can usually be distinguished by the history. Still, it is well to recall that tumor may follow trauma. Aneurism of the aorta may erode the spine and cause symptoms, first of irritation and later of pressure. It is only to be detected by exploration and by the methods of physical diagnosis. Cancer of the vertebræ usually causes very urgent symptoms, especially of initial pain, and later of pressure on the cord or nerve-roots. The nature of the lesion may remain for some time obscure; or there may be the history of precedent cancer. The symptoms soon become extremely aggravated, and the patient becomes cachectic.

In neuritis there is soreness of the nerve-trunks, with paralysis of motion or of sensation, or of both, in areas supplied by the individual nerves. In early stages motion is more likely to be impaired than sensation. Muscular atrophy occurs. Compression symptoms, and involvement of the bladder and rectum, are wanting. Still, the distinction is not always easy between neuritis and intraspinal tumor, especially if the new growth affects chiefly the nerve-roots, as in the cauda equina.

The eccentric or localized pain of tumor, especially in the early stage, may closely simulate a mere neuralgia, or even a disease of some internal organ, as in the chest or abdomen. The differential diagnosis must depend on the association of the symptoms; for instance, of other symptoms of cord-lesion, on the one hand, or of disease of the suspected viscus, on the other.

The local or regional diagnosis is to be made as in cases of myelitis or trauma.

XIV. INJURIES TO THE SPINAL CORD.

The spinal cord is subject to injury by blows, falls, crushings, stab-wounds, and gun-shot wounds. The commonest injuries are those which also cause fracture and dislocation of the vertebræ. It is not essential, however, that there should be a fracture or dislocation of vertebræ, as fatal injury has been done to the cord by falls, without visible injury to the bones. Gun-shot wounds are common, and a few instances are on record of the cord being injured or partly severed by a stab with a knife or stiletto. The commonest seat of injuries to the cord is in the neck; next in the dorsal region. The lumbar region, being more massive and better protected by large muscles, is not so often involved. It has been supposed in some cases that a vertebra may be partly dislocated and then spring back into place, thus causing a crushing of the cord. Fracture is not always associated with dislocation, nor *vice versa*.

The lesion in most of these cases is severe. The cord is either partly disintegrated, or entirely so in its transverse diameter. It is softened and necrosed, and may be the seat of hemorrhage. There may also be hemorrhage within the spinal membranes. In long-standing cases in which there have been attempts at repair, there is much scar tissue, together with degeneration of various tracts in the cord.

Symptoms.—The symptoms depend upon the seat of the lesion.

Injury to the *cervical region* causes a characteristic symptom-group, which varies according to the extent of the injury. There is a spastic paralysis of the legs, more or less complete, with paralysis of the bladder and rectum. The knee-jerks are, as a rule, increased, although in totally transverse lesions the knee-jerks may be abolished at first. Later, if the patient survives, they may return and become exaggerated. This is also true of the other deep reflexes. Contractures of the leg muscles often supervene, and bed-sores may form very rapidly about the buttocks or on the sacrum. The arms are totally or only partly paralyzed, according as the cervical enlargement and the roots of the brachial plexus are or are not totally involved. In some cases the arms lie paralyzed and

flaccid at the side; in other cases some powe
power of flexion of the arm at the elbow, and
Wasting of the muscles of the shoulders, arms, a
in if the cervical enlargement is injured. Se
abolished below the line of injury. If the lesio

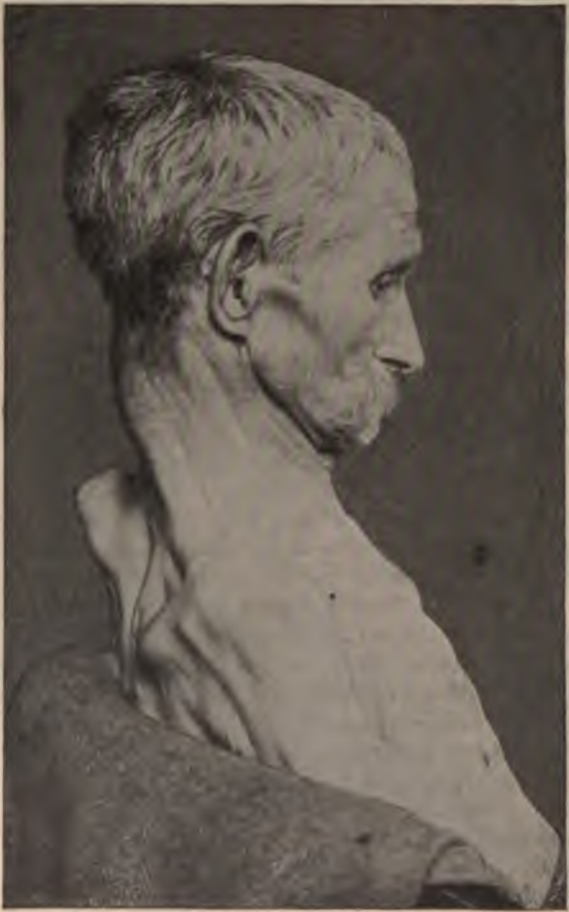

FIG. 395.—Trauma of the cervical region of the spinal cord, simulating syringomyelia.—Lloyd.

above except those in the upper extremities.
present gives a valuable clue to the uppermost li
may be a zone of hyperæsthesia at the extre
irritation of the nerve-roots; and pain may ra
at this level for the same reason.

Injury to the *lumbar region* also causes para
and rectum, but if the lumbar enlargement is inv
legs is flaccid, with abolished knee-jerks, wasting
trical changes. Anæsthesia may be present on
perineum, thighs, and legs. Pain in the legs ma

A unilateral lesion of the cord may cause the so-called Brown-Séquard syndrome: there is paralysis of motion on the side of the lesion and loss of sensation on the opposite side, but this sensory loss is in the temperature and pain sense rather than in the tactile sense. If, however, the posterior column of one side is affected there is tactile anæsthesia also, but it is on the side of the lesion.

Diagnosis.—To determine the exact seat of the lesion the practitioner should study the uppermost limits of the anæsthesia, and the muscle-groups involved, and compare them with a chart and table of the spinal segments. In this way an exact local diagnosis may be reached (pp. 313–314).

The history of the case is usually sufficient to establish the clinical diagnosis. It is not always possible to say whether the lesion is a mere hemorrhage in the substance of the cord, or in the membranes, or whether it is a crush of the cord. Practically the distinction is not of much importance so far as the welfare of the patient is concerned. It is more important to ascertain, if possible, whether the cord is merely affected by pressure, or whether it is actually crushed, especially when surgical intervention is contemplated. But this is seldom possible before operation. The reason why laminectomy is so seldom beneficial in these cases is that the cord has been damaged beyond the power of surgery to repair. Clinically these cases closely resemble myelitis; and in some of them secondary inflammation may be present. Abolition of the knee-jerks may indicate a total transverse lesion and is usually of grave import.

XV. HEMORRHAGE IN THE SPINAL CORD AND MEMBRANES.

Hemorrhage in the spinal cord, or hæmatomyelia, is usually the result of injury. It may be associated with a crush of the cord due to violence, or with fracture or dislocation of the vertebræ. In a few cases of injury, however, the hemorrhage may be the only lesion. Thus cases have been reported of great violence, as a fall down a long flight of stairs, in which the bones were uninjured, and even the membranes escaped, and yet a small and rapidly fatal hemorrhage was found in the substance of the cord. The hemorrhage may be in the gray matter, or close to it, and may cause extensive destruction of the spinal medulla. It may be so small as not to be apparent until the cord is sectioned, or it may cause an appearance of pallor or slight swelling on the surface. In some cases the blood breaks through to the surface and is present in the meninges. Minute capillary hemorrhages may be the starting-point for necrotic soft-

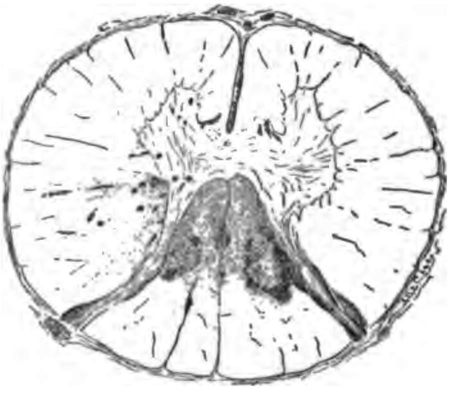

FIG. 396.—Hæmatomyelia.—Lloyd.

ening. Van Gieson has found long slender columns of necrosis in the cord, causing narrow cavities, which he thinks may be due to traumatic hemorrhage. They extend for long distances both above and below the main lesion. Meningeal hemorrhage may also occur from trauma.

Spontaneous, or non-traumatic, hemorrhage in the spinal cord is rare. The primary lesion is probably vascular, due to some weakness or disease of the blood-vessels. It is remarkable that such vascular lesions, which are so common in the brain, are apparently so uncommon in the spinal cord. It has been doubted by competent pathologists whether arteriosclerosis, as well as thrombosis and embolism, occurs in the cord; but there is not sufficient ground for dogmatic statements in the negative. It seems more probable that some cases of hemorrhage and hemorrhagic softening in the cord may be due to such lesions.

Symptoms.—The symptoms come on rapidly, even suddenly, and may cause profound shock; but even in the traumatic cases this rule is not absolute, for hours may elapse before all the symptoms are established. This is probably due to the fact that a small hemorrhage, once started, continues for some time to progress, with gradually increasing effect. Especially in meningeal hemorrhage this gradual onset is seen, and perhaps more so in the lower part of the spinal canal, where the cauda equina is involved. Many hours have been known to elapse before the paralysis was complete. In such a case presumably the blood gradually settles and clots in the lower part of the spinal canal. There may be complete paralysis of all the muscles below the level of the lesion. If the lesion is in the neck, the muscles of the chest may be paralyzed. The respiration is then diaphragmatic and irregular.

All modes of sensation may be absolutely lost below the level of the lesion. These symptoms, however, vary. Some hours may elapse before they are fully established, and later, even in unfavorable cases, sensation has been known to return in part. Thus the patient may have a return of tactile sense in some limited area; or of pain sense, or thermic sense, or sense of position, one or all. The bladder and rectum may be paralyzed, and priapism may occur.

The reflexes, especially the knee-jerks, may be retained and soon become exaggerated. If the lesion is totally transverse, the knee-jerks are likely to be abolished, although they may return in time. Occasionally they are not lost until after some days. Total abolition of the knee-jerks, therefore, in a hemorrhage *above* the lumbar enlargement is not a favorable sign, for it indicates an extensive lesion. If the hemorrhage is *in* the lumbar enlargement the knee-jerks will probably also be abolished, but this is from interference with the reflex centres in the cord; hence it is not necessarily so ominous a symptom. The ciliary reflex from irritation of the skin of the neck may be absent in cases of hemorrhage in the cervical region; hence there may be spastic myosis. If this centre is only irritated there is symptomatic mydriasis in one or both eyes.

Pain may be an urgent symptom. In Kindred's case, in which the hemorrhage was in the fourth dorsal segment, there was an initial agonizing pain simulating angina pectoris. Pain may persist, and it may even be felt far below the level of the lesion, if this is not totally transverse; this probably because free blood escapes in the meninges and causes pressure and irritation at lower levels. The girdle or "cincture" feeling is present in some cases.

In brief, the symptoms of hæmatomyelia, whether traumatic or non-traumatic, are those of a complete or almost complete transverse lesion of the cord. Abolition of the knee-jerks may be seen. The localizing symptoms depend upon the exact level at which the lesion occurs, and they have already been described under the head of myelitis.

The course of severe cases is usually rapid. In Kindred's case of spontaneous hemorrhage death came in six hours. If the lesion is totally transverse, with abolished knee-jerks and rapidly forming bed-sores, the prognosis is highly unfavorable. If the lesion is high in the neck death may come from paralysis of the phrenic nerve.

High temperature and excessive sweating are seen toward the end in fatal cases. In a case recorded by Lloyd moist skin was noted above, and dry skin below, the line of anæsthesia toward the end.

In hemorrhage in the membranes, when the cord itself is not involved and not much pressed upon, the outlook is more favorable, although permanent crippling in some form is not unusual. Pain is often a prominent symptom, and the anæsthesia and pain may follow individual nerve-trunks. In the legs, if the cauda equina is involved, there is a flaccid paralysis with muscular atrophy and abolished reflexes, somewhat like a multiple neuritis, but with paralysis of the bladder and bowel.

Diagnosis.—In traumatic cases the diagnosis is not difficult, except to distinguish hemorrhage from crush, and this is often not possible, nor is it of great practical importance. The history of the case points unerringly to a grave lesion of the cord. It is important to distinguish fracture, and this can be done only by a careful surgical examination, aided by the X-rays. Hæmatomyelia is not likely to be mistaken for a disease of any internal organ, and yet in dorsal hemorrhage the initial symptoms have simulated angina pectoris. But the speedy onset of paralysis, anæsthesia, priapism, and incontinence would distinguish the cord lesion. So, too, of pain from lumbar hemorrhage simulating disease of abdominal viscera. The supreme difficulty in non-traumatic cases is to distinguish hæmatomyelia from transverse myelitis, acute white softening, and pachymeningitis. The sudden onset and rapid course serve better than all other symptoms to distinguish hemorrhage from any of these conditions; but the diagnosis in non-traumatic cases may remain obscure. The extreme rarity of spontaneous hæmatomyelia must not be overlooked in coming to a conclusion. Softening and hemorrhage being due often to the same causes, and being part of the same pathological process, a dogmatic diagnosis between them may sometimes not be practicable.

The exact seat, or level, of the lesion is to be determined as in injury or myelitis.

XVI. SOFTENING OF THE SPINAL CORD.

Acute softening of the cord is often confused with myelitis, and indeed in some cases it may be impossible to distinguish between them at the bedside. The subject has already been discussed under the head of myelitis. The latter term properly indicates an inflammation, and this in turn is due to some infection; whereas softening may presumably be caused by a vascular lesion, such as embolus or thrombus. Nevertheless, softening is sometimes secondary to inflammation; and it also results from trauma. Weiss has recorded a case of softening of the cord occurring suddenly in a boy who had mitral disease. Gowers refers to a similar case. The inference is that cardiac emboli were the causes of the softening. The symptoms are so similar to those which occur in myelitis that it is not necessary to describe them again. The onset of the disease, however, may be more abrupt.

XVII. THE CAISSON DISEASE.

The caisson is a large compartment, inverted, in which a constant supply of compressed air is maintained in order to counteract the pressure of the water from without, thus allowing men to work upon the foundations of piers. The caisson disease is an affection, largely of the central nervous system, and especially of the spinal cord, caused by working in the compressed air of these compartments. The highest pressure attained at the St. Louis bridge was 50 lbs. to the square inch, the normal pressure of the atmosphere being 15 lbs. By some observers the mischief is attributed to the emerging from the compressed air while the system is overheated and fatigued with the hard labor.

Pathology. — Van Rensselaer studied the cord in a case of this disease and found extensive changes. A disseminated necrotic area was found in the dorsal region, with ascending degeneration in the columns of Goll and in the direct cerebellar tract, and descending degeneration in the pyramidal tracts, respectively above and below the mid-dorsal lesion. The necrosis seemed to be confined to the white substance, the gray matter not being involved. Possibly with the more refined methods now used, this entire exemption of the cells of the gray matter might not be found. No hemorrhages were seen. The fanciful theory that air-bubbles or air-emboli cause the softening need not be discussed here. Brooks has recently studied the blood-pressure in 75 workmen before, during, and after working in the caisson, and found no marked change in arterial pressure.

Symptoms.—The initial symptoms do not appear until after the workman emerges into the outer air. Some minutes, even hours, may elapse. There is usually severe epigastric pain, with vomiting, then very severe pain in the back and lumbar region, along the spine, and shooting down the legs. Smith described these pains as of a tearing character and intolerable. Anæsthesia shows itself promptly, often of the type known as "anæsthesia dolorosa," and advances with the motor symptoms. The paralysis is usually in the form of a paraplegia, although in some cases the arms also have been involved. The bladder and rectum are commonly paralyzed. Bed-sores form.

Brain symptoms are seen in the more severe cases, and especially in fatal cases. There are headache and vertigo, and in fatal cases unconsciousness supervenes before death. Pelton describes a comatose type, which may appear suddenly, with cyanosis, and is usually fatal. The duration in mild cases is from a few hours to six or eight days. In severe cases death may result in a few days.

The viscera are not, as a rule, seriously involved. There may be bronchial irritation and cough. The urine has usually a high specific gravity. Perforation of the ear drum and catarrh of the middle ear have been observed.

Diagnosis. — The import of severe spinal symptoms, such as paraplegia, anæsthesia, and incontinence, in a caisson worker soon after quitting work is unmistakable. The disease, as described by most observers, is of spinal or central origin, as proved also by such autopsies as the one made by Van Rensselaer, and is not a mere peripheral neuritis, due to carbonic acid poisoning, as others have contended. Still, it is not unlikely that the peripheral neurons may be found involved in some cases. An affection similar in every way to the caisson disease occurs in divers.

XVIII. AFFECTIONS OF THE CAUDA EQUINA.

The cauda equina is the leash of nerve-roots lying in the lower end of the spinal canal. These are the nerve-roots that come off from the lumbar and sacral segments. The spinal cord ends about opposite the second lumbar vertebra; consequently the nerve-roots from its lower segments have to traverse comparatively long distances before uniting to form the lower spinal nerves at or about their respective foramina of exit from the spinal canal. These prolonged nerve-roots are the cauda equina.

Pathology.—The cauda equina may be the seat of injury or disease. The injuries are similar to those that cause damage to the cord itself. Thus there may be fracture or dislocation of the vertebræ, rupture of the membranes, or traumatic hemorrhage, sometimes with secondary inflammation. Fractures and dislocations of the lower parts of the spine are not so common as in the dorsal and cervical regions, for the parts are massive and protected by large muscles; nevertheless they sometimes occur. But a traumatic meningeal hemorrhage may settle from still higher levels and press upon the strands of the cauda equina. Septic meningitis may occur in the lower part of the spinal canal, sometimes during the puerperium, or from other causes, such as a sloughing bedsore. Syphilitic meningitis may also occur, and syphilitic tumor, or gumma, is not so very uncommon. Other varieties of tumor may also be seen. It is well to bear in mind that although the cauda equina, being composed of nerve-fibres, is anatomically a part of the peripheral nervous system, nevertheless, being contained within the spinal canal and enclosed within the spinal meninges, it has practically some points of resemblance to central structures.

Symptoms.—Pain is a common symptom in meningitis or neuritis of the cauda equina, and is due to involvement of the sensory roots. It may be felt in the lower part of the abdomen or pelvis, or it may be transmitted to the legs and feet. It is sometimes lancinating or neuralgic, or burning as in neuritis, and may be associated with glossy skin

XIX. SPINA BIFIDA.

Spina bifida is a developmental defect, caused by failure of the vertebral arches (which grow from the mesoblastic somites in the embryo) to coalesce behind the spinal cord and between it and the skin of the back. Several varieties of deformity result according to the tissues involved (see p. 16).

Spina bifida is sometimes associated with other developmental defects, especially hydrocephalus.

This condition usually presents no difficulty in diagnosis, although the defect is not always detected at once in the new-born child. The small tumor on the back tends to grow, and in time may become very large. The important point is to determine the variety of the bifid spine. In meningomyelocele the spinal cord and nerves are involved, and paraplegic symptoms are always present. This distinction is important, especially from a surgical stand-point, for any operation for the excision or obliteration of the sac in this variety, even if successful, must inevitably leave the child a more or less hopeless cripple. In simple meningocele the results may be better, as the cord is not involved.

The tumor is usually in the lumbar, lumbosacral, or sacral region. It is infrequent in the cervical and upper dorsal regions.

Spina bifida requires to be distinguished from certain other embryological defects, such as congenital tumors in the sacrococcygeal region, dermoid cysts, and teratomata, but these fall within the purview of surgery.

DISEASES OF THE SPINAL NERVES.

I. MULTIPLE NEURITIS.

This disease, also called peripheral neuritis or polyneuritis, is, as its name indicates, an inflammation, more or less wide-spread, of the nerves.

Etiology. — Multiple neuritis is caused by a great variety of poisons, the most common being alcohol, lead, arsenic, mercury, and some of the infectious diseases, especially diphtheria. Cases also occur after typhoid fever, and more rarely after smallpox; also in diabetes. Beriberi is a form of multiple neuritis, the exact causation of which is still undetermined. Among the rarer causes of polyneuritis are carbon monoxide and carbon bisulphide. Phosphorus may also cause neuritis, and in ergotism there is involvement of the sensory and motor nerves. The anæsthetic form of leprosy is due to a neuritis.

Pathology. — There are two forms usually described — the parenchymatous and the interstitial. Whatever may be the cause of multiple neuritis, its essential morbid anatomy is much the same, and sometimes the cases partake of both forms. There are segmentation of the myelin, proliferation of the nuclei of the sheath of Schwann, and destruction of the axis-cylinder. In many cases there are also some overgrowth of the connective tissue and alterations in the blood-vessels. In the purely parenchymatous form there is destruction of the neuron, even including its cell-body; but according to Berkley, in experimental poisoning with alcohol, the earliest changes appear to be in the blood-vessels of the nervous system. The seat of the most active changes is usually in the periphery of the nerves. As the cord is approached the disease process diminishes. At the present time, however, the tendency is to find some evidence of degeneration even in the cord, due doubtless to the peripheral neurons being implicated as far as their course in the spinal medulla—as, for instance, in the posterior columns.

Symptoms.—The most common form of multiple neuritis is that which is caused by alcohol. This may be taken as a type, although some of the other forms, notably that caused by diphtheria, differ from it in certain particulars, as will be pointed out.

In alcoholic neuritis the earliest symptom is usually pain. This pain is felt especially in the nerve-trunks and in the muscular masses, such as the calves of the legs. It is commonly of an intense burning character, and is so urgent that for a while it may mask the other symptoms. When fully developed this pain is quite unmistakable; it is increased by pressure and by handling the parts; and the patient, especially if a woman, will cry out and even weep when the limbs, and particularly the legs, are handled. The toes and soles of the feet may be exquisitely sensitive, and sometimes exhibit some erythema. This is the state known as [illegible]. The pain is increased on voluntary motion, and is usually worse in the legs and feet; but it may not be absent in the upper limbs. Pain is not present in all cases. Other sensory symptoms are paræsthesia and various grades of anæsthesia. The thermal sense may be affected in alcoholic cases, and the electrical sensibility may be impaired, especially to weak currents; but strong currents, particularly if they cause muscular contraction, are most painful.

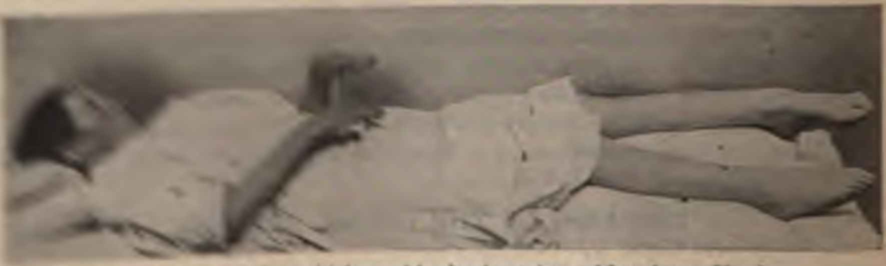

FIG. [illegible].—Alcoholic multiple neuritis, showing wrist- and foot-drop.—Lloyd.

Of motor symptoms the commonest are cramps, tremors, paralysis, contractures, and ataxia. Cramps occur early, but they are not seen in all cases. Tremors are occasionally seen in the weakened muscles, and are especially common in the alcoholic cases. Paralysis is the most important motor symptom. This is of the flaccid or peripheral type; the muscles waste and become flabby, and complete reactions of degeneration appear. The distribution of this paralysis is characteristic; it invades all four limbs, and is most marked in the extensor muscles; hence there are wrist-drop and foot-drop. It is also most marked in the distal muscles; that is, in the muscles furthest from the trunk; hence the forearms and hands, and legs and feet, are more paralyzed than the upper arms and thighs. The trunk muscles, however, do not escape, and the external muscles of respiration may be seriously involved. There is also tachycardia. The bladder and bowel are not paralyzed, as a rule, although a few doubtful cases are recorded. In the alcoholic cases the cranial nerves, except the vagus, are not often affected. Paralysis of the abducens nerve and optic neuritis have been observed.

Muscular contractures are not uncommon in advanced cases; they are most marked in the flexor muscles, which are the least paralyzed. These contractures hold the limbs, especially the legs, in a semiflexed position; they are hard to overcome, and greatly retard recovery.

Ataxia is seen occasionally, giving rise to *pseudotabes*. The incoördination is not unlike that of locomotor ataxia. It occurs in the alcoholic cases and in those caused by lead.

The reflexes are usually abolished. This is true especially of the deep, or tendon, reflexes. The knee-jerks, as a rule, are lost early; in some cases, however, they may persist much diminished. Some observers claim that the deep reflexes are exaggerated; but such exceptions are extremely rare, and they are difficult to explain except on the theory of irritation of the sensory neurons. The superficial, or skin, reflexes are not so promptly or uniformly lost, and in cases in which there is marked hyperæsthesia they may even be very active; but in advanced cases, particularly when there is anæsthesia, these reflexes are abolished.

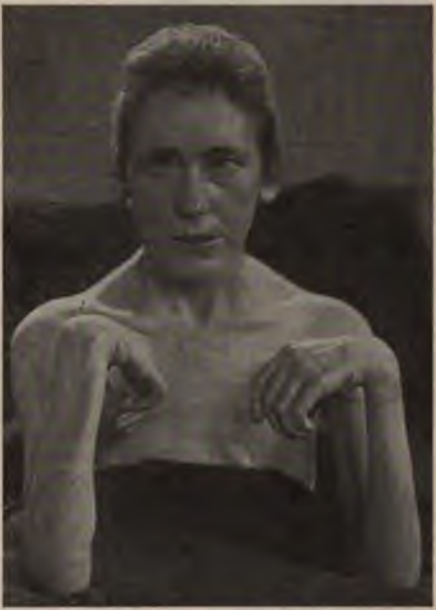

FIG. 398.—Alcoholic polyneuritis, with marked muscular atrophy and wrist-drop.—Singleton Smith.

Of trophic lesions the commonest is atrophy of the muscles. Œdema of the paralyzed legs is sometimes seen, and occasionally erythema, as of the soles of the feet. Ulcers and skin lesions are very uncommon; but trophic bed-sores, especially about the malleoli, have been observed in the post-typhoid cases. Glossy skin may be entirely absent. Profuse weating is seen in some cases.

In severe cases the weakness of the heart muscle may be the determining ause of death. Paralysis of the external respiratory muscles constitutes n additional source of danger. The phrenic nerve is not often involved.

In some of the alcoholic cases a characteristic psychosis occurs. It characterized by a wandering delirium, with hallucinations of sight and earing, confusion of identity of time, place, and persons, and a tendncy to fabulation. Atypical cases of alcoholic multiple neuritis occur. ccasionally, the patient has little if any pain, while the motor paralysis may be extreme. This *motor type* may or may not be associated with great ataxia, and constitutes a form of *pseudotabes.* It does not follow, however, that in all cases of pseudotabes there is an absence of pain. Another rather rare form is that in which the paralysis is confined to the lower limbs. The usual pain and sore muscles in the calves are present, but there is an absence of bladder and rectal troubles, such as occur in cord lesions. The knee-jerks are lost.

In *lead neuritis* the clinical picture is somewhat different. The distribution of the paralysis is usually much less extensive; as, for instance, in the cases in which the extensors of the forearms are alone involved. In some rare cases, however, there is a more wide-spread paralysis, and the upper arms, shoulders, and even the lower limbs are affected. There is a form of lead palsy closely resembling progressive muscular atrophy. The pseudotabes may also occur, but as a rule sensation is not much involved in lead cases, and pain is rarely observed.

Postdiphtheritic paralysis (q. v.—diphtheria).

The two most specialized forms of multiple neuritis are *beriberi* and the *anæsthetic variety of leprosy* (q. v.—beriberi; leprosy).

In *diabetes mellitus* neuralgic pains of a severe type sometimes occur, and occasionally anæsthesia, especially of the legs, and along with this may occur some paralysis and atrophy of the muscles, with lost knee-jerks.

The forms of multiple neuritis due to arsenic, mercury, typhoid fever, smallpox, and most other infectious diseases show nothing very distinctive.

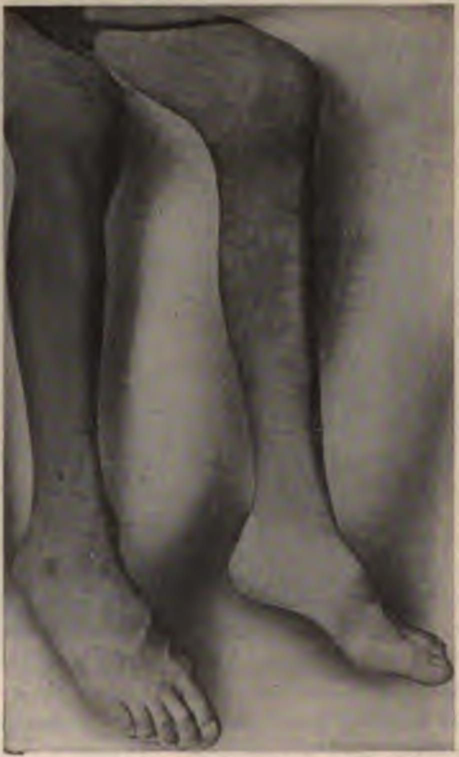

FIG. 399.—Peripheral neuritis, with foot-drop of right side, after enteric fever.—Pennsylvania Hospital.

Diagnosis.—The disease with which multiple neuritis is most likely to be confused is *locomotor ataxia,* and this is true especially of the ataxic type of polyneuritis. But polyneuritis differs from tabes in its mode of onset, which is usually much more brusque; in its history, which usually points to the poison or infection which causes it; in the pain, which is constant, burning, and neuralgic, and much increased by pressure, while in tabes the pains are lancinating, paroxysmal, and not affected by pressure, unless sometimes, indeed, they are relieved by it; in the paralysis and muscular atrophy, which are late phenomena in tabes and often absent even late in the disease; in the reactions of degeneration; in the gait, which is high-stepping in neuritis, with foot-drop, the toe grazing the ground (the so-called "turkey-gobbler walk"), due to paralysis of the extensor muscles, while in tabes it is incoördinate, the foot being flung far out and the heel striking the ground first—a distinction which is seen even in pseudotabes of neuritic origin. Moreover, in locomotor ataxia there are the true tabetic phenomena, such as optic atrophy, Argyll-Robertson pupil, atony of the bladder, sexual impotence, and arthropathies, which are not seen in polyneuritis, although in the form due to lead, optic neuritis and atrophy sometimes occur. In both diseases the knee-jerks are abolished, and the loss of static equilibrium may be seen in the pseudotabes as well as in the true tabes. Sensory changes are somewhat similar in the two diseases, except that deep sensibility (the sense of position, of pressure, and of voluntary and passive motion) is more likely to be abolished in tabes, while the superficial sensibility (tactile sense especially) is preserved. But these modes of sensation are sometimes affected also in multiple neuritis.

Various forms of *myelitis* simulate multiple neuritis. In the subacute and chronic anterior poliomyelitis there is a flaccid paralysis with muscular atrophy, but pain is not conspicuous and may be entirely wanting, and fibrillation of the muscles is seen. Sometimes the tendon reflexes are not entirely lost. The reactions of degeneration are not complete; and finally the history and evolution are different. Transverse myelitis is marked by spastic paraplegia, with exaggerated knee-jerks, and incontinence. When the lumbar enlargement is involved there may be flaccid paralysis, but the bladder and bowel are paralyzed, and the symptoms are confined to the lower limbs.

In the *myopathies* the evolution of the disease is extremely slow; pain is not conspicuous, or it is even wanting, anæsthesia is usually absent, and the history of the case is different. The neuritic form of the myopathies has more in common with neuritis, and may even depend upon a slow neuritis, but the course is extremely slow, the disease is sometimes familial, and the history is different. Some of these forms begin in childhood, whereas multiple neuritis is usually, but not always, a disease of adult life. A few cases of alcoholic multiple neuritis have been reported in children.

Hysteria may simulate multiple neuritis, but only superficially, and more particularly in chronic cases, with anorexia and wasting and contractures of muscles. But there are no true reactions of degeneration; the tendon reflexes are preserved or even increased; hysterical stigmata are present; and the history is characteristic.

II. THE CERVICAL NERVES AND CERVICAL PLEXUS.

The cervical plexus is formed from the four upper cervical nerves. These nerves after issuing from the spinal canal divide into anterior and posterior branches; it is from the anterior branches alone that the cervical plexus is formed. The posterior branches are largely sensory, and the largest nerve arising from these is the great occipital nerve. The most important branch of the plexus proper is the phrenic nerve.

Pathology.—This group of cervical nerves r eases about the base of the skull and the upper ce these especially are spinal caries, and, very rarely, and dislocation of these vertebræ occasionally affections also may be seated in these nerve branc

Symptoms. — Neuralgia of the great occipi tributed to the occiput as high as the vertex, There are painful points, especially where the trapezius muscle, and over the boss of the pari usually paroxysmal. This nerve may also be ir the atlas, as in a case reported by Lloyd, in whi in the distribution of the great occipital (see Fig. by the fact that the nerve is a branch of the sec issues from the spine between the atlas and the a new growths there is not only pain but also stiff and this may also be seen in meningitis in the the brain. In some cases of torticollis it is pr motor branches of the plexus are involved.

Diagnosis.—Care is required especially to det lesion, as new growths, spinal caries, fracture, ba

III. THE PHRENIC NE

This nerve supplies the diaphragm. It ari fourth cervical nerves, and therefore has its ce and fourth cervical segments of the spinal cord. I also from the fifth cervical. In the neck it is de between the subclavian artery and subclavian ve under surface of the diaphragm.

Pathology.—Owing to its deep situation in phrenic nerve is not often injured. It may be invol of the neck and in surgical operations; also in ca and in cervical meningitis. In injuries to the cerv or dislocation of the vertebræ, this nerve is so centres of origin. This complication is then the in In some of the cases in which the lesion is low and the external respiratory muscles are paraly the phrenic nerve, the respiration being entire nerve is said also to be involved sometimes in a and in diphtheritic paralysis. It may also be pre

Symptoms.—Paralysis of both phrenic nerve ysis of the diaphragm. Respiration is entirely by muscles. Dyspnœa may or may not be present monia are among the risks in these cases.

Diagnosis.—The symptom may be overlooke phrenic nerve is affected. On close inspection, h latter case that the movement of the diaphragm impaired, and when both nerves are paralyzed the to descend is evident. Compensatory action of muscles causes exaggeration of the costal type of

IV. THE BRACHIAL PLEXUS.

The brachial plexus is formed from the anterior branches of the last four cervical nerves and of the first dorsal nerve. It begins close to the vertebræ and extends to just beneath the clavicle, where it gives origin to its main nerve-trunks. These trunks go to form the circumflex, musculospiral, musculocutaneous, median, ulnar, and internal cutaneous nerves. Hence the brachial plexus supplies with motor and sensory filaments the shoulder and upper limb. These various nerve-trunks, however, take origin from the brachial plexus in such a way that none of them is derived entirely from any one spinal nerve-root.

Pathology.—The brachial plexus may be the seat of severe neuralgia, trauma, inflammation, and neoplasm. Neuralgic affections are often of obscure nature; they may be caused by rheumatism or gout, possibly also by exposure and by slight grades of inflammation. Tumors are rare; aneurism sometimes acts as a cause. Trauma may cause extensive lesions. Fracture of the clavicle and dislocation of the head of the humerus, especially the subcoracoid form, may cause injury to the plexus. Spinal caries may cause irritation of the nerve-roots.

Symptoms.—In neuralgia usually the sensory fibres alone are involved. Painful points are present, as where the nerve-trunks are most exposed or most easily subjected to pressure; hence, on the side of the vertebræ, in the axilla, and on the musculospiral nerve in the upper arm, and on the ulnar and radial nerves in the forearm. If inflammation is present there may be areas of paræsthesia and anæsthesia, and vasomotor and trophic lesions. In these cases the motor fibres also suffer, paralysis resulting. The pain is sometimes intense.

In traumatic cases the pain may be very severe, and various forms of paralysis result according as the lesion is total or partial; also glossy skin and other trophic lesions, such as muscular atrophy; also contractures in old-standing cases. In organic lesions of the plexus from whatever cause there may be complete or almost complete brachial monoplegia. All voluntary movements are lost when the lesion is a total one, and the arm then hangs inert at the side. The shoulder may be kept slightly elevated by over-action of the trapezius muscle. The paralysis is flaccid, with reactions of degeneration, and the tendon-reflexes are lost. The pain may be severe, paroxysmal, ill-defined, and may interfere with sleep. In total lesion the anæsthesia involves the hand and forearm and the outer aspect of the upper arm. The tip of the shoulder is not involved, as it is supplied by the cervical plexus, and the inner aspect of the upper arm also escapes, as it is supplied by the first three dorsal nerves. The upper arm type, according to Erb, is caused by injury to the fifth and sixth cervical roots; the lower arm type by injury to the seventh and eighth cervical and dorsal roots. The upper arm type shows paralysis of the deltoid, biceps, brachialis anticus, supinator longus, and possibly the infraspinatus, supraspinatus, and serratus magnus. The lower arm type is characterized by paralysis of the muscles of the lower arm and hand.

Diagnosis. — The diagnosis is easy, and is made from the peculiar distribution of the symptoms. Cases vary according to the extent and completeness of the lesion. The cause in pure brachial neuralgia may be obscure; and careful search should be made for organic lesions, such as neuromata, aneurism, and vertebral disease.

V. THE ANTERIOR THORACIC NERVES.

These nerves are not of great clinical importance except as pointers in some cases of injury to the brachial plexus. They are two in number. The external nerve is a branch from the outer cord of this plexus and supplies the pectoralis major muscle; the internal nerve arises from the inner cord and supplies both the pectoralis major and minor muscles. They are consequently likely to be involved in a lesion high in the plexus, but they escape in a lesion as low as the retroclavicular portion of that system of nerves. Paralysis of the two pectoral muscles impairs the use of the arm; but the incapacity to the patient is not as great as might be supposed. Forced adduction of the arm across the chest is affected.

There are a few cases on record of congenital absence of the two pectoral muscles on one side. Whether this anomaly depends on primary defect of the two nerves, is not determined. There is surprisingly little embarrassment to the patient, and in one case the individual was a laboring man who had found no inconvenience from his defect.

VI. THE POSTERIOR THORACIC NERVE.

The posterior, or long, thoracic nerve (the external respiratory nerve of Bell) arises from the fifth and sixth cervical nerves. It passes through the middle scalene muscle in the neck, and runs upon the outer surface of the serratus magnus muscle, which it supplies with a filament at each of its digitations. It supplies no other muscle.

Pathology. — This nerve is most exposed to injury at and about the point where it penetrates the middle scalenus muscle in the neck. Thus it has been injured by carrying heavy weights on the shoulder; possibly also by mere muscular exertion, acting directly through the middle scalene muscle. Cases have occurred thus in parturition. It may also be injured by blows upon the chest and by axillary wounds. The nerve is probably also involved in some cases of alcoholic multiple neuritis in which disease paralysis of the external respiratory muscles is not uncommon. Rare cases have been seen to follow typhoid fever and diphtheria.

Symptoms.—Paralysis of the serratus magnus muscle is the only result of injury to this nerve. This paralysis causes an excessive mobility of the scapula, which stands out from the chest wall, giving the patient a peculiar "winged" appearance. This is best shown when the patient elevates the arm: the posterior edge of the scapula then flies out from the chest wall and rotates its inferior angle upward and outward, while the acromion descends. The movement of the arm is thus somewhat embarrassed, as it is deprived of the basis of support normally given by the shoulder-blade. The chest wall on the affected side is not properly expanded in inspiration. In traumatic cases pain may be felt in the neck. The affection is generally unilateral, but bilateral cases have been reported.

Diagnosis.—The paralysis is easily recognized by the peculiar "winging" of the scapula.

VII. THE CIRCUMFLEX NERVE.

This nerve supplies the deltoid and teres minor muscles, and gives sensation to the skin over the lower and posterior parts of the deltoid. It arises from the posterior trunk of the brachial plexus in common with the musculospiral nerve, and in order to reach its destination it winds around the neck of the humerus—a fact of great clinical significance. It enters the deltoid from the under surface.

Pathology.—The commonest cause of injury to the circumflex nerve is dislocation of the head of the humerus. The nerve may also be injured in fractures of the neck of the humerus. A rare instance is reported by Raymond of injury to both circumflex nerves by pressure during sleep, the patient lying on his back with the arms elevated and the hands clasped behind the neck. One or both nerves may be injured also during surgical anæsthesia. This nerve is not usually the seat of spontaneous or primary neuritis, although it may be affected in lead poisoning and in diabetes. It lies too deep to be often injured by external causes. In cases of arthritis, rheumatic or otherwise, of the shoulder-joint there may be some wasting and paralysis of the deltoid muscle.

Symptoms.—Paralysis of the deltoid muscle is shown by inability to lift the arm from the side and to elevate and hold it above the head. Loss of power in the teres minor causes inability to rotate the head of the humerus outward. As these two muscles take part in various combined movements with the muscles of the upper arm and shoulder, their paralysis is very disabling. The anæsthesia, occupying but a small area, may readily be overlooked, and in fact cannot in some cases be found even by careful search.

Diagnosis. — Paralysis of the deltoid and teres minor muscles is unmistakable from the peculiar disablement of the arm. The greatest risk of error is in cases of dislocation of the shoulder-joint, when the practitioner omits to prepare the patient's mind for the possibility of this complication. A week or more may elapse before the paralysis is recognized, especially if the shoulder has been kept bandaged and pain has prevented movement in the joint. The paralysis is of the flaccid type, and the deltoid muscle may waste rapidly and give the reactions of degeneration. Cases have been recorded in which recovery did not take place for fully a year.

Arthritis causes immobility of the shoulder-joint, but the joint is usually ankylosed and the scapula moves with the humerus. In some of these cases there is more or less atrophy and loss of power in the deltoid muscle.

VIII. THE MUSCULOSPIRAL NERVE.

This nerve is the largest branch of the brachial plexus. It winds around the humerus from within, behind, to the outer side of the arm in a spiral groove beneath the triceps muscle. It divides, near the external condyle, into two terminal branches, the radial and the posterior interosseous nerves. In the upper arm its main trunk supplies branches to the triceps, anconeus, extensor carpi radialis longior, supinator longus, and in part to the brachialis anticus. The interosseous branch, below the elbow, supplies the extensor muscles of the wrist, thumb, and fingers. The musculospiral nerve, and its main branch, the radial, supply sensation to the posterior aspect of the arm, the anterior aspect of the lower part of the arm, and the back part of the forearm, hand, and fingers, except possibly the extreme tips of the thumb and first three fingers, which, according to Richelot, are supplied by the median nerve.

Pathology. — The musculospiral nerve may be injured in various ways. It is exposed to pressure during sleep, when the patient lies with his head upon his arm; this is particularly so in the sleep of alcoholic drunkenness. The nerve is injured occasionally by the pressure of the head of a crutch. Bilateral palsy may be caused in this way. It is also liable to gun-shot and other wounds, but these are rare. The posterior interosseous branch in the forearm is particularly vulnerable to lead poisoning, the well-known wrist-drop resulting. The musculospiral nerve is also injured sometimes in fracture of the humerus, in dislocation of the elbow, and even by sudden and violent muscular action of the arm.

Symptoms.—These depend upon the seat of the injury. If the main trunk is injured high in the upper arm all the muscles supplied by the nerve are paralyzed, including the triceps. This is the case usually in the crutch palsy. In the case of pressure during sleep the triceps usually escapes, and then the paralysis is only in the supinator longus and the extensors of the wrist and fingers; occasionally, however, the supinator longus escapes. When the paralysis is complete the patient cannot supinate the hand, nor extend the hand at the wrist, nor extend the fingers. The resulting wrist-drop is characteristic. A curious result is seen in diminished power of flexion of the hand and fingers, not that the flexors are truly paralyzed, but according to a physiological law that when the antagonistic muscles are paralyzed the protagonists lose some of their power apparently from failure of a basis of support. This is shown if the hand is passively over-extended, for then the power of the grip is much strengthened. The loss of power of supination excites an effort of compensation in the patient, in which he rotates the humerus outward and presses the arm strongly against the side. The basal phalanges cannot be extended, but the other phalanges, being extended by the interossei, which are supplied by the ulnar, can be extended rather weakly, but best if the basal phalanges be extended passively. As a rule, however, in wrist-drop the fingers are flexed and the thumb turned in and depressed. The paralysis of the supinator longus and brachialis anticus causes some loss of power of flexion of the forearm on the arm, but the latter muscle is not much involved, as it does not receive its whole supply from this nerve. In lead palsy, as a rule, the supinator longus escapes and may stand out conspicuously among the wasted muscles. The extensor of the metacarpal bone of the thumb may also escape. The muscles most involved in lead paralysis are the extensors of the wrist and fingers.

Sensation may be not much impaired in pressure cases and in lead palsy. When present from a total transverse lesion, anæsthesia is distributed about as follows: When the lesion is high in the upper arm the loss of sensation is located on the posterior part of the arm, the anterior aspect of the lower part of the arm, the back of the forearm, especially on the radial side, and the back of the hand and fingers, except the tips of the thumb and the first three fingers. When the lesion is lower, as in pressure cases, loss of sensation, if present, is only seen below the elbow. As in all peripheral palsies there may be marked atrophy of the muscles, lost tendon reflexes, and reactions of degeneration.

Diagnosis.—The paralysis is so characteristic that a mistake is hardly possible. Lead palsy is bilateral, although occasionally it is worse on one side than the other; it is to be distinguished from the extensor paralysis of alcoholic multiple neuritis by the history; by the pains and the wider extent of the paralysis in alcoholic cases; and by the escape of the supinator longus in lead cases. Pressure cases and traumatic cases generally are unilateral. The history is usually clear.

IX. THE MEDIAN NERVE.

The median is properly called the fellow of the ulnar nerve, as both are flexors of the wrist, hand, and fingers. It arises from the brachial plexus and passes down by the side of the brachial artery. All its branches are given off in the forearm and hand. It supplies all the muscles on the front of the forearm except the flexor carpi ulnaris and the inner half of the flexor profundus digitorum, which are supplied by the ulnar. In the hand it supplies the abductor, the opponens, and the short flexor of the thumb; also the first two lumbricales. Its functions therefore are largely flexor and pronator. It also gives sensation to the radial side of the palm and to the palmar surfaces of the thumb, fore and middle fingers, and the radial side of the ring finger. According to some observers it also supplies the dorsal aspect of the tips of these fingers and the thumb.

Pathology.—The median nerve may be injured in many ways. It is not often involved by pressure during sleep, nor is it injured as often as the ulnar.

Symptoms.—One of the most characteristic symptoms of paralysis of the median nerve is inability to pronate the forearm; this cannot be accomplished beyond the mid-position, and the patient supplements the attempt by rotating the humerus inward. Another characteristic symptom is inability to oppose the thumb to the tips of the fingers; the thumb, in fact, is much hampered in many of its movements, including flexion and abduction. Still other symptoms are impaired flexion of the wrist, which is then done entirely through the ulnar nerve, with consequently marked deviation of the hand toward the ulnar side; and loss of flexion of the phalanges, except the distal phalanges of the ring and little fingers, which are supplied through the ulnar. The unopposed extensor action of the interossei muscles may cause a subluxation of the joints between the second and third phalanges. Anæsthesia is present on the radial side of the palm, the palmar surfaces of the thumb, fore and middle fingers, the radial side of the ring finger, and the dorsal tips of these fingers and thumb.

Trophic lesions are very common in injuries of the median nerve.

Diagnosis.—This is usually clear from the history of the case and the characteristic distribution of the symptoms. The loss of power of complete pronation, the awkward flexion of the wrist with deviation of the hand to the ulnar side, the disablement of the thumb, and the loss of power of flexion of the fingers, together with the classical anæsthesia, are determinative. The muscular atrophy and reactions of degeneration help to distinguish the case from one of cerebral origin. The trophic lesions, if present, are especially significant.

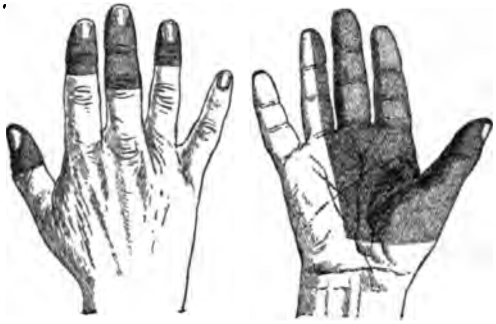

FIG. 400.—Distribution of anæsthesia in paralysis of the median nerve.

X. THE ULNAR NERVE.

The ulnar nerve is one of the two flexor nerves of the wrist, hand, and fingers. It arises from the brachial plexus and passes down the inner side of the upper arm in close proximity to the brachial artery. At the elbow it passes behind the internal condyle, where it can readily be felt in most persons, and where pressure upon it causes a tingling sensation. It supplies the flexor carpi ulnaris, part of the flexor profundus digitorum. the muscles of the little finger, the interosseous muscles, two of the lumbricales, the adductor pollicis, and one head of the flexor brevis pollicis. It gives sensation to the ulnar side of the forearm and hand, the little finger, and the ulnar side of the ring finger. By reason of its distribution this nerve presides over the ulnar flexion of the wrist, the flexion in part of the fingers, the adduction and abduction and other finer movements of the fingers, also extension of the second and third phalanges and flexion of the first phalanges, and the movements of the thumb toward the palm.

Pathology.—This nerve is much exposed to injury, as by wounds, pressure, dislocation, and fractures. It may also be involved in tumors and malignant growths, and in surgical operations. Pressure may occur during sleep, especially during alcoholic sleep and the unconsciousness caused by ether or chloroform; also during parturition.

Symptoms. — Paralysis of the ulnar nerve causes a characteristic disability. Flexion of the wrist is impaired, and on attempts at flexion there is a deviation of the hand toward the radial side. Flexion of the little and ring finger is especially impaired, and the little finger is almost entirely paralyzed. Adduction of the thumb is lost, hence the patient's inability to grasp objects. Flexion of the first phalanges and extension of the last two phalanges are impaired, but the loss of flexion is less in the first two fingers which are supplied in part by the median nerve. The finer movements of the fingers are abolished. In long-standing cases a characteristic deformity results which is called the "main-en-griffe"; the first phalanges become over-extended, much more so than is possible by voluntary power, and the last two phalanges are strongly flexed. This deformity, however, is not so marked as in cases of anterior poliomyelitis, because the lumbricales of the first two fingers are not involved in ulnar paralysis, being supplied by the median nerve. Muscular atrophy is usually marked; the interosseous spaces are hollowed out, the palm is wasted, and the hypothenar eminence is atrophied. Anæsthesia involves the ulnar side of the arm for some distance above the wrist, also the ulnar side of the hand on both the palmar and dorsal aspects, the whole of the little finger, and the ulnar side of the ring finger.

Diagnosis.—This is comparatively easy, because of the characteristic disablement and deformity. Some care may be necessary in cases due to pressure, as during sleep, or parturition, or surgical anæsthesia. The history of the case, however, is usually clear, and the resulting paralysis is unmistakable. In a case occurring during the delirium of typhoid fever, the paralysis was not detected until the patient's mind became clear and he himself called attention to the loss of power. In irritative injuries, such as gun-shot wounds, trophic lesions may result, such as glossy skin and œdema, and these may be accompanied with burning pain or "causalgia."

XI. THE INTERCOSTAL NERVES.

These nerves are branches of the twelve dorsal nerves. They are separate from each other in the sense that they do not form a plexus. Each nerve runs forward in an intercostal space, and for part of its course it is in close proximity to the pleura.

The intercostal nerves may be variously affected, and they sometimes furnish important indications for diagnosis. This is especially so in diseases of the spinal cord. Thus a spinal meningitis or a spinal tumor may irritate the roots of one or more dorsal nerves, and this may point to the location of the lesion. It may also simulate an intercostal neuralgia. The same is true of spinal caries. In all such organic lesions the diagnosis is to be made by a study of other attendant symptoms. Aneurism of the thoracic aorta may, by irritating a dorsal nerve-root, simulate disease of the spinal cord. Herpes zoster frequently attacks one of the dorsal roots, its point of attack being probably the posterior ganglion, and the pain and characteristic eruption follow closely the distribution of the nerve affected. Intercostal neuralgia is to be distinguished from pleurisy. The affection known as mastodynia, or neuralgia of the breast, is often a puzzling affection, and is commonly seen in neurotic or hysterical women. Fracture of the ribs is usually attended with severe pain in the intercostal nerves, and the diagnosis may require great care, especially where the history of trauma is obscure.

XII. THE LUMBAR PLEXUS.

This plexus is formed from the twelfth dorsal and the first four lumbar nerves. The fifth lumbar nerve does not enter into this formation, but after receiving a branch from the fourth, goes, as the lumbosacral cord, to help form the sacral plexus deep in the true pelvis. This formation of the lumbosacral cord has not a little clinical importance, as will be shown later. The principal nerves arising from the lumbar plexus are the iliohypogastric, the ilio-inguinal, the external and internal inguinal, the anterior crural, and the obturator. The iliohypogastric nerve supplies sensation to the skin of the gluteal and hypogastric regions. It is of little clinical importance. The ilio-inguinal nerve is both motor and sensory. It supplies the internal oblique muscle. It passes out at the external abdominal ring and is distributed to the skin of the scrotum in the male and of the labium in the female, and to the upper inner part of the thigh. The genitocrural nerve is of clinical significance as supplying the cremasteric muscle. It might be involved in psoas abscess, as it penetrates the psoas muscle. The external inguinal or external cutaneous nerve is the sensory nerve of the outer part of the thigh as far down as the knee. Its posterior branch supplies the outer posterior part of the thigh as far as the middle third. The anterior crural and obturator nerves, because of their importance, are described under separate headings. From the nature of their origin and relations, however, they are often involved in lesions of the lumbar plexus.

Pathology.—The lumbar plexus is less frequently the seat of injuries and disease than either the brachial or sacral plexus. Its anatomical position, deep within the body, explains this comparative exemption. The lumbar plexus, being merely a continuation of the nerves which have their origins just within the spinal canal, is closely associated with these parts in its pathology. Falls and crushing injuries may possibly involve the lumbar plexus as well as the intraspinal nerve-roots. Tumors within the body may also cause pressure or irritation. Lumbar and psoas abscess may variously affect these nerve-cords. Finally, parturition may do injury to the anterior crural and the obturator nerves.

Symptoms. — These vary widely according to the particular nerve-cords involved. A good method of diagnosis is carefully to study the motor, sensory, and reflex phenomena *seriatim*, and then by grouping these, to arrive at a conclusion as to what parts of the plexus are involved.

The pain caused by these lesions may be most misleading. Lydston has reported a case of acute lumbar abscess which, by irritating some strands of the lumbar plexus, simulated nephritic colic; the pain was in the iliolumbar region. Psoas abscess may cause pain and paralysis in the anterior crural nerve, which passes through the psoas muscle. It may also involve the genitocrural nerve. Referred or distal pain is sometimes a puzzling symptom from this cause, such as pain on the inner side of the knee and down the inner side of the leg following the course and distribution of the anterior crural and its long saphenous branch. The knee pain due to hip-joint disease is another example, probably due to irritation of the obturator nerve.

The motor and reflex symptoms depend upon the nerves implicated. If the anterior crural is involved the knee-jerk is lost and the extensor muscles of the thigh and leg are paralyzed. The cremasteric reflex may be lost by involvement of the genitocrural nerve. Paralysis of the obturator causes loss of power of adduction in the thigh. Paralysis of the bowel and bladder is not caused by involvement of the lumbar plexus.

Diagnocis.—This has been indicated in the foregoing description of the pathology and symptoms. It is most important to search for gross local lesions, such as abscess, tumor, and vertebral disease. Injuries to the plexus are rare; they sometimes occur as results of child-birth.

XIII. THE ANTERIOR CRURAL NERVE.

This nerve arises from the lumbar plexus and passes out of the pelvis beneath Poupart's ligament on the outer side of the femoral artery. It supplies the muscles on the anterior of the thigh, which act as extensors of the leg; within the pelvis it passes through the psoas muscle, and supplies the iliacus. It gives sensation to the front and inner surfaces of the thigh, and by its main branch, the long saphenous, to the inner side of the leg and foot as far sometimes as the great toe.

Pathology.—The anterior crural may be involved in psoas abscess, as the nerve passes through the fibres of the psoas muscle. Other deep-seated abscesses in the pelvis might also affect it, but lumbar abscess is more likely to affect other and higher branches of the lumbar plexus. Injury to the anterior crural nerve is rare. Fullerton reports the case of a dwarf in which pressure during labor caused transient injury to this nerve; and it may also be hurt in some forms of dislocation of the hip, but only rarely; probably also by a wrench of the psoas muscle.

Symptoms.—There is paralysis of the quadriceps extensor muscle with consequent inability to extend the leg. This paralysis is flaccid, with atrophy, and the knee-jerk is abolished. If the iliacus muscle is involved within the pelvis, there is inability to flex the thigh, which is characteristically shown by the patient lifting the thigh with his hands when asked to cross the lame leg over the other. This movement also is embarrassed by the paralysis of the sartorius. Pain may be present and is experienced from Poupart's ligament to the inner side of the knee, and thence down the inner side of the leg even to the foot and great toe. In psoas abscess this pain is sometimes distal and is relieved by flexing the thigh on the pelvis with the knee kept bent. Anæsthesia involves the anterior and internal surfaces of the thigh and the inner surface of the leg and foot following the course of the long saphenous branch.

Diagnosis. — Psoas abscess may simulate crural neuralgia. The paralysis of the nerve is readily determined by the characteristic loss of power in the iliacus, sartorius, pectineus, and quadriceps extensor muscles, with lost knee-jerk, and by the distribution of the anæsthesia and pain. Irritation of the roots of this nerve might be caused by a tumor in or near the spine, or by disease of the lumbar vertebræ. It may also be paralyzed by aneurism of the femoral artery. Diabetes has been known to cause paralysis and neuralgia in both crural nerves.

XIV. THE OBTURATOR NERVE.

This nerve, after rising from the second, third, and fourth lumbar nerves, and penetrating the inner fibres of the psoas muscle, runs along the inner and lateral wall of the pelvis to the obturator foramen, by which it emerges. It supplies the obturator externus and adductors of the thigh, the articulations of the hip and knee, and the skin of the inner side of the thigh, and, possibly by its communication with the long saphenous nerve, of the leg also.

Pathology. — Because of its deep position injury to the obturator nerve is rare. Some obstetricians believe that it is sometimes involved in puerperal cases, as by the forceps, or the child's head, or in pelvic inflammation. The pain in the knee in the early stages of hip-joint disease is usually ascribed to irritation of the obturator nerve, which sends filaments to both joints; but some orthopædists explain it as due to muscular spasm. Obturator hernia and pelvic tumors may compress the nerve, and it may be irritated by psoas abscess.

Symptoms.—Paralysis of the obturator nerve interferes with crossing the affected leg over its fellow, and with outward rotation of the thigh. The anæsthesia is on the inner side of the thigh and perhaps of the leg, but it may not be marked.

Diagnosis.—This is indicated in the account of the symptoms. The knee pain in hip-joint disease has led to error. In an obscure case it is well to bear in mind the possibility also of a psoas abscess.

XV. THE SACRAL PLEXUS.

The sacral plexus lies deep in the pelvis. It is formed from the lumbosacral cord, which arises from the fourth and fifth lumbar segments, and from the three upper sacral nerves and a part of the fourth sacral nerve. It gives origin to four main nerves, the superior gluteal, pudic, small sciatic, and great sciatic.

Pathology.—The sacral plexus may be injured by tumors within the pelvis and diseases of the womb, ovaries, or rectum; by pressure during labor; by wounds; and it may be the seat of neuritis.

Symptoms.—These vary widely, according to the particular nerve-cords involved.

Pain is a common symptom, and may be felt about the buttock, hip, perineum, and even down the thigh and in the leg, foot, and toes, following the course of the great or small sciatic nerves. Paralysis of various muscles is also present, and this paralysis may even present certain types, according to the nerves involved. The bladder and bowel may be paralyzed. For a proper understanding of these cases it is best to describe the various types of the affection.

One of the commonest and most important is the *peroneal type*. This is particularly likely to occur after child-birth. After instrumental delivery, or even after normal labor in rare instances, the patient has pain in the foot and toes; this is soon followed by loss of power in the extensor muscles of these parts, such as the peronei, the tibialis anticus, and the long extensor of the toes. The patient in walking has foot-drop and lifts the foot high, the toes dragging along the floor. The paralysis is flaccid, the muscles atrophy, and complete reactions of degeneration are seen. The knee-jerks are not abolished. Anæsthesia may be present on the outer part of the leg and on the dorsum of the foot and toes. The explanation of this affection lies in the fact, that the external popliteal or peroneal nerve is a continuation of the lumbosacral cord in the pelvis, and that this lumbosacral cord is particularly exposed to injury by pressure where it runs over the brim of the true pelvis in passing down to the sacral plexus. In these cases there may also be some loss of power in the gluteus medius muscle, which derives its motor supply from a branch of the lumbosacral cord. In most of these puerperal cases the affection is unilateral; occasionally, however, there is a bilateral paralysis, presenting a rather different type.

The form of paralysis varies. All depends upon the particular nerves involved. Occasionally the great sciatic nerve alone is impaired by some intrapelvic lesion, and the case then presents the picture of an ordinary sciatica. A small ovarian tumor has been known to cause pain and loss of power in the buttock. In some of the post-partum cases pain is the chief or only symptom.

Diagnosis. — It is of first importance that the practitioner should determine the cause. When this has once been done the nature of the case usually becomes clear.

The chief difficulty is to distinguish these cases from affections of the cauda equina. The history, however, usually points to an intrapelvic lesion, as in the puerperal and gynæcological cases, and an expert investigation *per vaginam* or *per rectum* will generally detect a local cause if it is present. The examination by the rectum is especially important, as by this means the nerve-trunks can be palpated. Affections of the cauda equina are usually bilateral; while those of the sacral plexus are often unilateral; but this latter rule is not absolute.

Hysteria may simulate these affections, but this psychoneurosis is to be recognized by the mental state, history, and peculiar hysterical stigmata.

Cancer of the rectum, as well as ovarian tumor, has been mistaken for sciatica or some form of pelvic neuralgia.

XVI. THE SCIATIC NERVES.

The great sciatic arises from the sacral plexus and passes out of the pelvis by the sacrosciatic foramen, where it is readily accessible to pressure between the great trochanter and the tuberosity of the ischium. It supplies in the thigh the hamstring muscles—the biceps femoris, the semimembranosus, and semitendinosus—and divides just above the popliteal space into two main branches, the internal popliteal and external popliteal (or peroneal) nerves. These two branches supply all the muscles below the knee (both extensor and flexor) and give sensation to most of the leg and foot, the parts not supplied by it being a strip along the inner side of the leg which is supplied by the long saphenous nerve, and the upper part of the calf which is supplied by the small sciatic.

The small sciatic also arises from the sacral plexus and passes out of the pelvis alongside of the great sciatic; it supplies the gluteus maximus muscle (through the inferior genital nerve), and a well-defined area of the skin which includes the buttock, the perineum, and a strip running down the back of the thigh to and including the upper part of the calf.

Pathology.—The great sciatic nerve may be the seat of wounds, inflammation, and tumors. It is sometimes injured in dislocations of the hip, also in forceps deliveries, and by pelvic tumors and even masses of fæces. Inflammation of the nerve constitutes the disease known as "sciatica." Opportunities to examine the nerve are rare in sciatica, but in a few instances on record the nerve-trunk has been found swollen and congested. In the operation for stretching, a similar state has been found. Gout, rheumatism, syphilis, alcoholism, and lead have all been assigned as causes of sciatica. Exposure to cold, especially after great or prolonged muscular effort or a wetting, may cause the attack. Fournier said that it may follow gonorrhœa.

Symptoms.—In organic lesions, such as injury or tumor, the symptoms are characteristic. Pain is an early symptom, sometimes with twitching of the muscles and drawing up of the leg. If the nerve is severed or in any way completely paralyzed, there is loss of power in the hamstring muscles and in all the muscles below the knee, with anæsthesia on the outer side of the leg, dorsum of the foot, the toes, the sole, and a portion of the inner and back part of the leg. The regions that are exempt are a narrow strip on the inner side of the leg, supplied by the long saphenous nerve, and the upper part of the calf, supplied by the small sciatic. The muscles atrophy, and reactions of degeneration are present. The knee-jerk may not be lost. Trophic ulcers sometimes occur.

Pain is the common and sometimes the only symptom in sciatica. This pain is severe and neuralgic in type, especially felt about the upper and back part of the thigh. It may also extend down along the course of the nerve and be particularly intense in the calf of the leg and even in the foot and toes. Any unusual motion in the leg may aggravate it, as suddenly bending the thigh. In such cases the forcible action of the pyriformis muscle probably causes pressure on the hypersensitive nerve-trunk at its exit from the pelvis. In the early stages the pain may be paroxysmal; later it is constant, dull, and aching, or even severe and shooting. Sensitive points are found at the exit from the pelvis, behind the knee, at the head of the fibula, and on the back of the foot. The trunk of the nerve in the thigh is usually extremely sensitive to pressure. Firm pressure between the great trochanter and the tuberosity of the ischium is usually most painful.

Paræsthesia is sometimes observed, as burning and tingling in the areas of distribution of the sensory nerve endings. Anæsthesia is not common, and when present may escape detection if not carefully sought for.

Motor involvement is not common in sciatica. There is usually no true paralysis of muscles, no atrophy, or reactions of degeneration. The knee-jerk, as a rule, is not affected. The Achilles-tendon jerk may be lost. Inhibition of motion is not uncommon, but this is due to pain. The gait is affected for, the same reason, and there may be slight scoliosis. The muscles may become flabby and wasted from disuse. Trophic lesions are rare. Herpes may occur.

The small sciatic nerve may be injured along with the great sciatic at the point where they emerge together from the pelvis. Instances in which it is injured alone must be very rare. Its paralysis causes loss of power in the gluteus maximus muscle, which is shown in inability to rise from a sitting position. This paralysis, however, is due to involvement of the inferior gluteal nerve, which, according to Gray, is closely associated with, rather than a branch of, the small sciatic. Anæsthesia is present on the buttock, the perineum, and the back part of the thigh and popliteal space as far as the upper third of the calf.

Diagnosis.—If there is some organic cause, as injury or tumor, the history of the case and a careful physical examination usually reveal it, and, with the characteristic motor and sensory symptoms, disclose the nature of the case. It is said that flat-foot may cause sciatic pain.

It is easy, as a rule, to recognize sciatica. Pelvic abscesses, disease of the uterus and ovaries, large fecal accumulations, and especially cancer of the rectum have been mistaken for sciatica. Bone disease in the lumbosacral spine may simulate sciatica by irritating the nerve-roots. Hip-joint disease is to be distinguished by the pain on moving the joint, restriction of motion, alteration in the length and position of the limb, tilting of the pelvis, and the other classical symptoms. Obscure cases always call for a rectal examination or vaginal examination. Appendicitis may cause deep-seated pains in the pelvis, but is not likely to be mistaken for sciatica. In sciatica the most distinctive symptom is the pain on pressure on the nerve-trunk. A sign pointed out by Lasègue is of diagnostic value—when the patient lies on his back and the extended limb is elevated, pain is not felt until an angle of about 90° is reached, and this pain is then relieved by bending the leg at the knee. The disease is unilateral in the vast majority of cases, bilateral sciatica being usually symptomatic of organic disease, such as myelitis, tumor, spinal caries, or affections of the cauda equina. Still, cases of genuine bilateral sciatica have been seen, especially in constitutional diseases, as gout and diabetes.

XVII. THE INTERNAL POPLITEAL NERVE.

This nerve is a continuation of the great sciatic. It supplies the flexors of the foot and toes (the calf muscles and muscles of the sole), and gives sensation to the outer part of the back of the leg, to the sole, and in part to the toes.

Pathology.—The internal popliteal, or posterior tibial nerve as it is called in its extension down into the leg, is so deeply situated that it is but seldom injured. It shares, however, in some diseases with other nerves, especially such affections as cause muscular atrophy and club-foot. In many of these cases, however, the seat of the lesion is in the anterior gray matter of the cord rather than in the nerve itself.

Symptoms.—Paralysis of the flexor muscles of the foot—the soleus, gastrocnemius, and plantaris—which are supplied by this nerve, causes the form of club-foot known as valgus, sometimes associated with calcaneus. Thus flexion of the foot is impaired, and the unopposed peroneus longus pulls the outer edge of the foot upward. The patient tends to walk on the inner edge of his foot and on the heel, and he cannot lift himself on his toes. When the nerve is injured, loss of sensation is found on the lower part of the back of the leg and on the sole.

Diagnosis.—The diagnosis is easily made from the characteristic distribution of the paralysis and anæsthesia.

XVIII. THE PERONEAL NERVE.

The peroneal or external popliteal nerve is in appearance a branch of the great sciatic arising in or just above the popliteal space, but according to some observers its fibres are really derived from the lumbosacral cord in the pelvis, as is proved by the occasional high division of the great sciatic within the pelvis, in which cases the peroneal is seen to be a continuation of one of these branches, which itself is a continuation of the lumbosacral cord. It passes into the leg behind the head of the fibula, where it may easily be palpated and where it is especially exposed to injury. It supplies the extensor muscles of the foot and toes, and gives sensation to the lower and outer part of the leg, the inner and outer parts of the ankle and foot, the dorsum of the foot, the inner side of the great toe, and the adjoining sides of the other toes.

Pathology.—This nerve is sometimes paralyzed by trauma acting upon it near the head of the fibula. It may also be involved in fracture of the fibula. The peroneal is particularly liable to involvement in cases of alcoholic multiple neuritis in association with other nerves; and in the polyneuritis following typhoid fever this nerve, as well as the ulnar, is apt to be conspicuously affected. There is also a form of muscular atrophy—the so-called Charcot-Marie-Tooth type—in which the muscles supplied by the peroneal nerve suffer especially. The disease is likely to begin in these muscles, but later the hands and forearms become affected. It probably depends upon a peripheral neuritis, and is sometimes a familial affection. Thus Ormerod observed three cases of this disease following measles in one family. The distal muscles are more affected than the proximal. In puerperal palsy the fibres going to form the peroneal nerve may suffer from pressure of the child's head on the lumbosacral cord and sacral plexus. Among rare causes of peroneal palsy are the application of an Esmarch tourniquet, and the pressure of stilts. It is a curious fact that for some obscure reason potato-pickers and others who work in a stooping position sometimes get paralysis of this nerve (Oppenheim).

Symptoms.—There is loss of power of extension of the foot and toes, with consequent foot-drop in walking. The first phalanges of the toes may be flexed by contracture of the interossei. The anæsthesia is on the outer side of the lower leg and ankle, the dorsum of the foot, and the toes. According to some observers the inner side of the ankle is anæsthetic.

Diagnosis.—The diagnosis is easily made from the characteristic distribution of the symptoms. The paralysis of this nerve is distinguished from that of its main trunk, the sciatic, by the escape of the parts supplied by the internal popliteal, and of the hamstring muscles.

GENERAL NERVOUS DISEASES.

I. CHOREA; SYDENHAM'S CHOREA.

St. Vitus's Dance.

A disease of children and young adults, characterized by continuous, irregular, involuntary muscular contractions and psychical derangements.

This substantive affection has nothing in common with a number of other diseases unfortunately described as chorea or choreiform, except abnormal muscular movements.

Etiology. — PREDISPOSING INFLUENCES. — The disease appears in successive generations in certain families. The readily transmitted neurotic constitution plays a more important rôle. The nervous, excitable children of nervous parents are especially liable to chorea. Chorea is particularly a disease of childhood and adolescence. It is rare before the fifth year and after the fifteenth. The cases in early adult life almost always occur in women. The disease occurs about three times as often in females as in males. Chorea is more common among the children of the poor, but is frequently observed among those living in affluence. It is rare among negroes. The disease is relatively frequent among bright, intelligent school children, especially girls, between ten and fifteen, who are encouraged by their teachers to unreasonable and unnatural application to study. Anæmia and general poor health often precede the attack, but, on the other hand, it frequently occurs in well-nourished children. The disease frequently develops in chlorotic girls. *Scarlet fever* is sometimes followed by chorea. This sequence also occurs as regards whooping-cough and other diseases of childhood; but there is no satisfactory evidence of a causal relation between these infections and chorea. In older persons chorea has been observed after gonorrhœa and sepsis. Chorea frequently develops shortly after an attack of acute rheumatism, and in a group of cases the arthritis is so mild and the choreic symptoms are so prominent as to justify a doubt as to whether or not rheumatic fever has actually been present. In other cases there is a history of rheumatic fever months or years before the development of the chorea. Under either of these conditions the evidences of endocarditis or chronic valvular disease may or may not be present. The endocardial murmurs present in a case of chorea may be due to actual valvular lesions, to irregular action of the heart, or to anæmia. A woman suffering from chorea may become pregnant; and the disease may develop during pregnancy or after parturition. It has been observed in repeated pregnancies. It begins more frequently in the early than in the later months and is often of the severe type—chorea insaniens. About twenty per cent. of the cases in pregnant women terminate in death. The causal relation of pregnancy is shown by the fact that the chorea ceases upon the occurrence of abortion or miscarriage, or delivery at full term.

THE EXCITING CAUSES. — In the present state of knowledge it must be assumed that various pathogenic agencies act as the immediate exciting cause of chorea. Among these are: emotional shock, especially fright; mental shock, particularly in young women; and some conditions associated with rheumatic fever and acute or chronic endocarditis, the nature of which is not understood. The attack has followed a slight injury or a surgical operation and has been attributed to reflex causes, as intestinal worms or genital irritation. It has been stated, but not substantiated, that ocular defects may cause the disease. Finally, the disease frequently develops in the absence of any noticeable cause.

Hypotheses of Chorea.—The view that it is a pure neurosis appears to be widely accepted. The embolic view has some basis of support in the experimental chorea produced in animals by the injection of indifferent substances in fine particles. Cases occur in the absence of endocarditis and without embolism, and with endocarditis but without embolism. That chorea is an infectious disease is an opinion which has the support of many observers. The fact that the attack in an important proportion of the cases directly follows fright or other profound emotional disturbance, and the prominent psychical derangements militate against this view.

Symptoms.—Two forms may be recognized: (1) the ordinary form in which the symptoms are of variable intensity, and (2) the maniacal form—chorea insaniens.

(1) THE ORDINARY FORM.—The onset of the disease is insidious. The child gradually becomes awkward, clumsy, and restless. He cannot sit still. There is a marked change in disposition. He appears to be careless and indifferent, and upon correction has spells of crying or becomes sullen. In the course of some days the characteristic involuntary movements begin. In the mild cases only one hand or the side of the face is affected—hemichorea; in the more severe cases both sides are involved, but one side to a greater degree than the other. In a well-marked case, the child cannot remain quiet, but is in continual motion. The face twitches, the arms are abducted, adducted, rotated, the hands extended, the fingers separated and at once withdrawn and flexed; the head and trunk are rotated and alternately flexed and extended. The gait may be disordered so that progression is unnatural and difficult. These and other movements are rapidly repeated in the most bizarre and disorderly manner, and often with convulsive suddenness. They are manifestly purposeless and constantly vary in extent and direction—"insanity of the muscles." The hands and arms are most affected, the face next, and to a less extent the trunk and lower extremities. The tongue is protruded and withdrawn with a jerking movement; words are uttered with an irregular, jerking cadence, and in grave cases the patient often does not talk at all for hours or even days together. The diaphragm may be involved, a condition which is manifest by irregular, spasmodic breathing. Attempts at voluntary movements increase the involuntary twitchings. There may be transient loss of power in a limb or the entire side. This is usually a mere paresis, but in certain cases it is marked—paralytic chorea.

Emotional influences increase the muscular movements. The consciousness of being under observation almost always intensifies them. As a rule they cease during sleep.

Sensory derangements are not common. In certain cases of hemichorea there is tenderness on pressure and spontaneous pain. Tender points in the line of nerve trunks are rare.

Psychical derangements occur in a majority of the cases. They are usually moderate; sometimes intense. The antecedent neurotic element is to be considered. Irritability, peevishness, and weakness of memory are present. These symptoms often increase, especially in adults, until a condition of mania is established. There is no distinct line of separation between the ordinary forms and those which are most severe.

(2) CHOREA INSANIENS.—This most intense form does not often occur in children, but is not very rare in young women, and especially in pregnancy. The muscular movements are excessive and continuous. The patient cannot remain standing or lie in bed. There is an uncontrollable jactitation, movement of the eyes and lips, inability to pause to take food or for any purpose, associated with a veritable psychosis with mental confusion, maniacal excitement, and hallucinatory delirium. After a time the patient falls into a state of profound exhaustion, with apathy or melancholia. The temperature may rise to 102°–104° F. (38°–40° C.) and in fatal cases hyperpyrexia has been observed.

The muscles do not waste, their electric excitability is not affected, and the deep reflexes remain normal.

Ocular Phenomena.—The pupils are often dilated, but the light reflex is retained. The muscular twitchings may give rise to transient strabismus. A concentric contraction of the visual field has been observed. The ophthalmoscopic findings are mostly negative, though a few cases of optic neuritis have been reported.

Course of the Attack.—The duration of the disease in children varies usually between six and twelve weeks. There is a remarkable tendency to recurrence, which often takes place in the spring of the year. Two or three attacks are common; as many as five or six have been noted. The tendency to recovery appears to be spontaneous. The chorea of adults runs a longer course. A chronic intermittent form has been described.

Diagnosis.—DIRECT.—This is, as a rule, made without difficulty. It rests upon the character of the muscular movements, their distribution, the age of the patient, the psychical phenomena, the frequent coincidence of rheumatic arthritis, endocarditis, or chronic valvular disease, and the tendency to spontaneous recovery. The absence of the signs of organic disease of the nervous system is of diagnostic importance.

DIFFERENTIAL.—There are several affections occurring in children which present superficial resemblances to Sydenham's chorea.

Multiple Cerebral Sclerosis.—Weakness, incoördination, tremor, and ataxia are characteristic. The gait is spastic paretic; the course chronic, with little tendency to improvement. The reflexes are increased and the intelligence is impaired. Has been described as chorea spastica.

Certain Disorders of Motility Associated with Hemiplegia.—Posthemiplegic hemichorea is a term used to designate the involuntary movements on one side of the body which manifest themselves in one muscular group after another and give rise to coarse trembling and awkwardness. A prehemiplegic chorea may occur but is much more rare.

Athetosis or hemiathetosis consists in slow involuntary movements chiefly affecting the muscles of the fingers and toes. There are movements of flexion and extension, adduction and abduction, which occur with occasional interruptions both during the waking hours and in sleep, or only upon attempts to use the member, or under excitement. The fingers may be over-extended and spread apart while the hand remains flexed or some fingers may be flexed and others extended at the same moment.

Hereditary Ataxia; Friedreich's Disease.—The chronic nature of the affection, its occurrence in several members of a family, the ataxia, tremor, scoliosis, and talipes, together with the nystagmus, the scanning speech, and the slow, irregular muscular movements, render the recognition of this disease a comparatively easy matter.

Hysteria.—The movements of so-called hysterical chorea are usually rhythmical and wholly different from those of acute chorea. The stigmata of hysteria are present and the psychical derangements are more marked than in the chorea of childhood. The two affections are sometimes associated.

Prognosis.—In children the outlook is good. Most of the cases terminate in complete recovery. Death may result from exhaustion in the severest cases. The mortality is from 2 to 3 per cent. Chorea in adults and especially chorea insaniens, is a much more serious affection. In the chorea of pregnancy the death-rate is about 25 per cent. Rapid loss of flesh, delirium, and a rise of temperature are ominous symptoms.

THE CHOREIFORM AFFECTIONS.

Several affections characterized by irregular, involuntary muscular movements are described as chorea. This nosological error is extremely unfortunate and misleading, since these diseases not only have no etiological and but little clinical relationship among themselves, but also none whatever to Sydenham's chorea.

Chorea Major; Epidemic Chorea.—It is a matter of history that at times of religious excitement in the Middle Ages there were extensive popular outbreaks marked by great excitement, gesticulations, and dancing. Under such circumstances pilgrimages were made in the Rhine provinces to the shrine of Saint Vitus at Zebern. This martyr as a saint of succor was invoked for protection against sudden death and against many diseases and distempers, notably chorea, which thus came to be called Saint Vitus's dance. Limited outbreaks of a similar character occurred in the nineteenth century and in this country in Kentucky. The fantastic pilgrimages of the Doukhobors in Manitoba are of this nature. These folk uprisings under the stimulus of religious fervor are hysterical manifestations and have nothing to do with Sydenham's chorea.

Habit Chorea; Habit Spasm; The Tics.—These affections have been misnamed chorea. They have no relationship with Sydenham's chorea.

Momentary grimaces or twitchings of bundles of facial muscles are not uncommon in otherwise healthy adults and are of no clinical significance.

Tic Convulsive; Gilles de la Tourette's Disease.—A psychosis characterized by involuntary violent muscular movements affecting certain muscle groups, as the facial and brachial, or generalized; explosive utter ances, which may be inarticulate, sometimes resembling the bark of a dog, or the repetition of words (echolalia), accompanied by involuntary movements, or the repetition of obscene words (coprolalia), or the spasmodic and involuntary imitation of movements (echokinesis), and in many of the cases by curious mental impulses or fixed ideas, as the impulse to touch certain objects (*folie de toucher*), or the obsession of names (onomatomania), or the insane habit of counting with worriment about numbers (arithmomania).

The affection, as a rule, begins in childhood about the time of the second dentition and affects neurotic individuals. The outlook is not favorable, but some of the cases recover. Allied to tic convulsive is the affection known as—

Saltatory Spasm; Static Reflex Spasm of Bamberger; Palmus.—This is probably not an independent disease but in some cases a form of tic; in others a manifestation of hysteria; and again a manifestation of increase in the skin- and deep-reflexes. The spasms do not occur when the patient is at rest or in the recumbent posture. When he touches the floor with his feet he begins to hop, jump, and dance about as the result of clonic convulsive contractions of the muscles of the legs and feet, and in particular of the muscles of the calves of the legs. It occurs in both sexes and at any age; in many cases without obvious cause; in others after emotional disturbance or in the convalescence from an infection. It has been observed in dancers as an occupation neurosis. The prognosis is favorable; recovery usually takes place in the course of a few months. The condition occasionally persists for years.

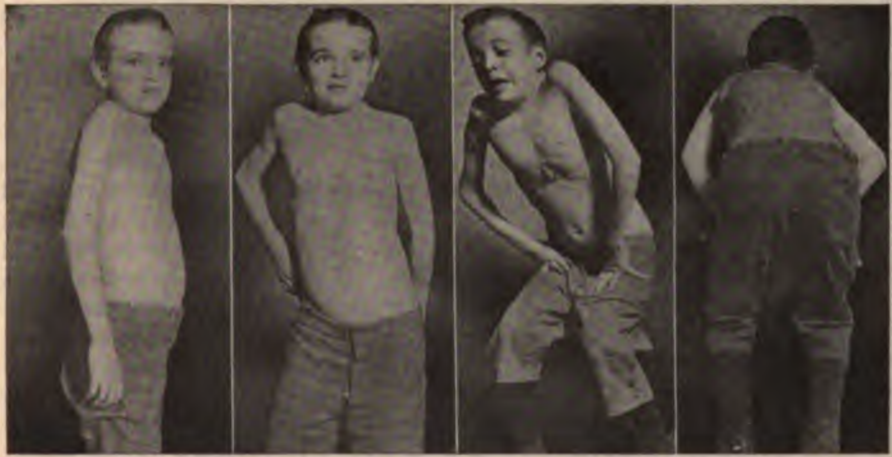

FIG. 401.—Gilles de la Tourette's disease in four phases.—Pennsylvania Hospital.

Jumpers.—This form of saltatory spasm has been observed as a ocal or family neurosis in Maine and parts of Canada. It is characterized y sudden jumping, with outcries, echolalia, and echokinesis. A similar ffection has been observed in parts of Siberia—myriachit—and in Java, vhere it is known as latah.

Chronic Progressive Chorea; Hereditary
Disease.—An affection of early middle life, mo
terized by irregular muscular movements, disor
tability and mental weakness gradually leading t
tion was first described by Huntingdon, of Lor
it in families in whom it has occurred for four c
part played by heredity is a conspicuous feature,
observed in which this etiological factor was al
alike suffer. The onset is usually between the thi
and is insidious, without apparent exciting cause.
followed profound and depressing emotion.

The symptoms are motor and psychical. Th
sist of involuntary, purposeless movements, ma
degree in limited muscle-groups, as the hands o
creasing in force and extent until all the volunta
These movements are disorderly and irregular, of
abrupt than in chorea, and cause almost continu
ulations, increased by excitement and interru
At first and to some extent throughout the cours
capable of some degree of temporary control by
presently recur again with renewed violence. In
is much impaired. The body is inclined forwar
the movement of the legs is irregular, uncertai
arrested for a moment after a few steps. Mu
until toward the end, when palsies may occur
usually increased. Sensation and the special sen

Psychical phenomena are irritability, excit
Suicide is not uncommon. As the disease adva
apathy and progressive dementia. Speech is slo
tinct, the words being slurred and ill-pronounced

The prognosis is invariably unfavorable.
Its duration varies from ten to twenty or thirty
is usually caused by some intercurrent disease or

II. EPILEPSY.

A disease of the nervous system characteriz
sciousness, with or without convulsions, recurr
In very rare instances consciousness is not wholl

The following phases occur, independently o

1. *Grand Mal.*—Loss of consciousness with
first tonic, then clonic.

2. *Petit Mal.*—Momentary loss of conscious

3. *Status Epilepticus.*—Convulsive attacks r
consciousness not being regained in the intervals

4. *The Psychical Epileptic Equivalent.*—Out
mental symptoms, as automatism, take the place

5. *Jacksonian Epilepsy.*—The convulsive att
muscle group and may be unilateral and unatten
ness; later consciousness may be lost and the co

Etiology. — PREDISPOSING INFLUENCES. — Age plays an important rôle. In a large proportion of the cases the first attack occurs in early childhood; in the majority before the twentieth year. Many but by no means all of the cases occurring in adult life are symptomatic of a local lesion. In rare instances the disease begins in old persons. Sex is without influence. In children males appear to be slightly more liable; in adults there are more cases among males than females. Direct inheritance is comparatively infrequent, but the children of neurotic families, in which neuralgias, palsies, hysteria, and insanity have occurred, are more liable to become epileptic than the descendants of healthy stock. Paternal or maternal intemperance, especially when associated with syphilis or insanity, is frequently found in the anamnesis. Epileptic convulsions are not very uncommon in the subjects of chronic alcoholism. Epilepsy may occur in syphilitic subjects; more commonly the convulsive seizures are epileptiform and symptomatic of syphilitic disease of the brain. The general convulsions which occur in pathological primary dentition, or in children at the onset of acute infections, in uræmia, in pregnancy, and in chronic lead poisoning cannot in all instances be distinguished from idiopathic or essential epilepsy.

EXCITING CAUSES. — The first attack sometimes follows fright; sometimes an injury; less frequently one of the acute febrile infections. Masturbation has been regarded as a common cause of epilepsy upon insufficient evidence. The fact that certain local irritants give rise to epilepsy, which ceases upon their removal, is incontestably established. Among them are preputial adhesions, collections of smegma behind the corona, intestinal worms, a foreign body in the ear or nose, an irritable scar, phimosis, and a testicle retained in the inguinal canal. It is necessary to assume a strong predisposition in such cases. In some of them the attacks persist after the correction of the offending condition. Overeating and indigestion are very often followed by a fit, and a seizure may follow trauma, gall-stone. or renal colic, or a trifling surgical operation. Epileptiform convulsions occur in heart block—Stokes-Adams disease.

Symptoms.—The recurring fits are the characteristic and in many cases the only feature of the disease. In the intervals the health of the patient is often excellent; in certain cases and in the later stages it may be much impaired.

1. **Grand Mal; Major Epilepsy.** — The attack is frequently preceded by a localized sensory manifestation called an aura. AURÆ may be: (a) *Psychical.*—The patient experiences a sensation of strangeness or terror, or a feeling of confusion, or he may fall into a vague, dreamy state, or become extremely gay or furious. (b) *Visceral.*—In this form of aura the sensation is referred to various organs. It is described as the pneumogastric aura. Uneasy sensations in the epigastrium are more common. Sometimes the disagreeable sensation may be intestinal. In other cases it is precordial and attended by anxiety and palpitation. (c) *Peripheral.*—The sensation may begin in the hand or in a finger and extend toward the body before consciousness is lost. (d) *Visual.*—The aura may take the form of phosphenes or color sensations or in rare cases of particular objects. (e) *Auditory.*—Noises or ringing in the ear, curious sounds diffi-

cult to describe, musical tones, or voices. (f) *Olfactory or Gustatory.*—These are rare. They consist of strong odors, almost always unpleasant or disagreeable, or foul tastes and the like. The aura is usually of short duration, the attack coming on in a few seconds. In other cases it may be prolonged.

Premonitory Forced Movements.—In some cases the aura does not occur, but in its place there are definite forced movements. The patient turns rapidly or twirls upon his toes, or runs a few steps or even to and fro a number of times.

The Epileptic Cry.—At the onset of the attack the patient very often utters a loud scream or yell.

The attack is instantaneous. Consciousness is at once completely lost and the patient falls as if shot. Slight injuries are common, and grave, even fatal accidents, as fracture of the skull, sometimes occur. The FIT consists of three stages,—(1) tonic spasm, (2) clonic convulsions, and (3) coma.

(1) *Tonic Spasm.*—There is a spastic rigidity of the muscles, including the respiratory muscles, so that respiration is at once arrested. The face is at first pale, then red, and directly bloated and cyanotic; the eyelids are closed or open, the eyes fixed, the pupils dilated, and the iris is irresponsive to light. At the very first there may be a momentary contraction of the pupil, but this soon gives place to dilatation. The head is forcibly extended or turned to one side, the arms are rigidly extended or flexed, the thumbs adducted, and the fingers clinched. The legs may be rigidly extended, or flexed. The tongue may be protruded and caught between the fixed jaws. The fæces and urine may be discharged. This period lasts from a few seconds to half a minute. Toward its close there is tremor, which ushers in the second stage.

(2) *Clonic Convulsions.*—The muscular contractions now intermit. Tremor gives way to rapid and violent spasms. The limbs are tossed about with force. The muscles of the face are violently contracted; the eyes roll from side to side or are turned up, and the eyelids open and close forcibly. The jaw muscles are in violent clonic spasm and the tongue is lacerated by the teeth. A frothy saliva, often stained with blood, is discharged with the violent respiratory movements. Further discharges of urine and fæces may now occur and there is occasionally an ejaculation of semen. This period lasts from one to three or four minutes. The convulsions become less violent and gradually subside and the patient passes into the third stage of the attack.

(3) *Coma.*—The limbs are relaxed and the unconsciousness is profound. The breathing is stertorous and the face flushed but no longer cyanosed. After a time the patient may be aroused, but is dazed and confused and relapses into a deep sleep which lasts for hours.

The following clinical phenomena occur, but are not constant:

Vomiting after the attack. Slight rise of temperature,—one to two degrees of Fahrenheit's scale. In the status epilepticus higher temperatures are observed. Abolition of the reflexes. The conjunctival, corneal, and pupillary reflexes are all abolished during the attack. The deep reflexes may, however, be increased and ankle clonus evoked. Subcutaneous and subconjunctival extravasations of blood; the former chiefly about the face and neck. Albuminuria during the attack; polyuria subsequently; occasionally an increased urea output.

The aura is not always followed by the fit. When it begins in an extremity, particularly in the hand, the attack may be prevented in some cases by immediate compression of the member by a string or ligature or by energetic pulling or rubbing of the part.

2. **Petit Mal; Minor Epilepsy.** — Momentary loss of consciousness is the chief, often the only symptom. The unconsciousness usually is so brief—a few seconds to half a minute—that the patient does not fall, but directly resumes his occupation or conversation as if nothing had occurred to interrupt it. The face usually becomes pale, the eyes are set and staring, and anything in the hands may be dropped. The tongue is not bitten and involuntary discharges do not occur. In many of the cases slight spasmodic movements of the facial muscles may be noticed.

The attack may take the form of transient vertigo, the true nature of which, in the absence of unconsciousness and twitchings of the lips, tongue, or eyelids, cannot be recognized. Following the attack of petit mal there may be slight incoherency or automatic actions, such as beginning to undress, spitting, or rubbing the face or head. The patient may in other cases fall without the occurrence of convulsions. There may be jerking of the limbs, tremor, or sudden visual sensations. The significance of these phenomena is revealed by their occurrence in persons who manifest well-characterized attacks. An aura is rare and many patients are unaware of the occurrence of the attacks. As a rule convulsions gradually develop, and in many of the cases petit mal and grand mal are associated.

3. **Status Epilepticus.**—The patient passes from one convulsive seizure into another, consciousness not being regained in the intervals. The pulse and respiration are rapid, the temperature rises, and the attack frequently terminates in death. Hyperpyrexia is not uncommon and temperatures of 107°–111° F. (41.7°-43.9° C.) have been observed. Status epilepticus may last two or three days.

4. **Psychical Epileptic Equivalents.** — Remarkable psychical disturbances sometimes take the place of the fits or alternate with them. The patients perform extraordinary and apparently premeditated acts of which they have no knowledge or subsequent recollection. They run about, wander away, throw away their clothing, commit violent, even murderous assaults without motive and without self-restraint. These psychical states are not easily distinguished from the maniacal states which sometimes follow the attacks — postepileptic delirium. These conditions may last for hours or for several days. They usually come on suddenly without premonitory symptoms.

Other derangements which are regarded as equivalents are sudden and profuse sweating, sudden sleep (narcolepsy), the automatic repetition of meaningless words or phrases (verbigeration), and attacks of general tremor with impaired consciousness.

5. **Jacksonian Epilepsy; Cortical, Partial, or Symptomatic Epilepsy.** —Consciousness is not at first lost. It may be preserved throughout the attack. The attacks are the result of irritative lesions of the motor

zone, as tumor, inflammatory softening, acute and chronic meningitis, hemorrhage, abscess, and trauma. They occur also in general paresis. The spasm begins in a limited group of muscles of the face, arm, or leg. Numbness or tingling is followed by limited spasm, which extends and involves a limb or the side of the face. The spasms may be localized for a long time; but ultimately tend to become general. Posthemiplegic epilepsy is of the Jacksonian type. The convulsions may for a long time be confined to the paralyzed side, beginning in the hand or foot without unconsciousness.

The mental state of the epileptic is often normal. More commonly there is absence of self-control, associated with depression and irritability. As the disease progresses there is often impairment of intelligence and memory. The seizures may occur daily for a period and then at longer intervals; in other cases they may occur only once in many months. It is common for them to recur at irregular intervals of two or three weeks. The attacks of petit mal may occur many times a day. The attacks are more common by day than at night. Nocturnal epilepsy may go on for a long time without being recognized. In women the attacks frequently occur at or near the menstrual period.

Diagnosis.—DIRECT.—Major epilepsy declares itself by the aura, the cry, the instant loss of consciousness, and the consecutive tonic and clonic spasm followed by coma or stupor. The relaxation of the sphincters and the bitten tongue are distinctive. The recurrence of the fits at irregular periods is an essential feature.

The minor attacks are characterized by momentary loss of consciousness or vertigo. Twitching of the facial muscles is suggestive. Their frequent recurrence and association with grand mal are diagnostic. Status epilepticus occurs as a culminating condition to be known not only by the recurring convulsions and intervening coma, but also by the history of the case. Psychical equivalents of the attack can only be recognized in the light of the anamnesis. There is nothing distinctive in the delirium, mania, or delusions. The diagnosis of Jacksonian epilepsy rests upon its characteristic symptoms, local spasm, and retention of consciousness. The recognition of the peculiar condition of which it is symptomatic is often difficult.

DIFFERENTIAL.—The importance of the distinction between true epilepsy—the so-called idiopathic form—and symptomatic or epileptiform convulsions cannot be over-estimated. To the latter belong convulsions of the Jacksonian type. The diagnosis of epilepsy can never be made from a single attack, particularly when it has not been seen by the physician. *Syncope* may be mistaken by the untrained for epilepsy. The unconsciousness is not so complete as in epilepsy, nor is it preceded by an aura, accompanied by tonic and clonic convulsions, involuntary discharges, or followed by coma or automatic actions. *Ménière's disease* may simulate an epileptic seizure, but aural phenomena are present and the characteristic symptom-complex of epilepsy is absent. *Toxic Conditions.*—The general convulsions of pathological puerperal states, uræmia, lead intoxication, and other toxic conditions are not to be confounded with epilepsy. The underlying causes of these symptomatic seizures are usually plainly manifest. When they occur in individuals previously epileptic, their essential nature may remain in doubt.

General Convulsions of Infancy; Eclampsia.—These are due to direct irritation of the cerebral cortex, reflex irritation, and toxic influences. In early infancy epilepsy is the least obvious diagnosis and should never be made until other causes have been excluded. In older children the seizures may be symptomatic of peripheral irritation or intestinal worms, or they may replace the chill which in adults marks the onset of an acute infectious disease. When they are repeated, only prolonged observation will justify a positive diagnosis. Complete restoration to the usual health in the course of a few hours, especially in a child who presents the stigmata of degeneration, is an important point in favor of a diagnosis of epilepsy.

Coarse Cerebral Lesions.—Cerebral focal disease and meningocortical lesions may cause symptomatic or partial epilepsy. When the convulsions rapidly become general they may closely simulate general epilepsy. In truth the border line between the two forms is not always sharply drawn. The symptomatic convulsive seizures that occur in general paresis are commonly unilateral and not attended by loss of consciousness.

Hysteria.—(See p. 1388.)

Hystero-epilepsy.—The rare forms of major hysteria are attended with recurrent convulsions, which may be readily distinguished from repeated epileptic seizures or the status epilepticus by the emotional prodromes, the hysterogenetic points, globus, contortions, histrionic poses, and hallucinations. There are mixed forms of hysteria and epilepsy and transitional forms. In other words the hysterical person may be also an epileptic.

Simulated Epilepsy.—The normal light reflex, absence of dilatation of the pupil, the absence of instantaneous pallor at the onset of the attack, the lack of the characteristic cyanosis and flushing, and the condition of the patient after the attack are of diagnostic value. The convulsion may be feigned, but the tongue is not bitten and postepileptic coma and mental confusion cannot be imitated.

Prognosis.—In the great majority of cases epilepsy is an incurable disease. No case can be looked upon as having recovered unless there has been complete freedom from the attack for a period of several years. The outlook is less favorable when the disease begins in infancy or childhood than in the cases in which it begins at puberty. When it begins between the twentieth and thirty-fifth years complete recovery is rare. The more frequent the attacks and the longer the period in which they have continued to recur, the more unfavorable the prospect of recovery. The prognosis is unfavorable in degenerates and those suffering from inherited or acquired mental disease. Recovery is more rare in females than in males. The severity of the individual attack has no direct relation to the prognosis, except that the outlook is less favorable when the disease begins as petit mal, and that in the status epilepticus about one-half the cases die in the attack. Epileptics are frequently short lived. The attack in itself is not especially dangerous to life. In very rare instances asphyxia or cardiac rupture may occur. The seizure is attended with the risk of serious, even fatal injury which may result from sudden loss of consciousness. The patient may fall from a height or under a vehicle or into a fire or water. The outlook is more favorable in symptomatic convulsions than in essential epilepsy.

III. HYSTERIA.

Hysteria is a psychoneurosis, in which the mental state induces and dominates a great variety of physical symptoms. Its name, which is derived from the Greek word for the uterus, indicates an error which has prevailed for more than two thousand years, for hysteria has no necessary relation with the womb. It occurs in men and in young children as well as in women. Heredity is a common cause, as was demonstrated by Briquet. Next in importance is trauma, and then mental excitement and moral shock. Toxæmia, metallic poisoning, and acute disease all act as occasional causes.

Pathology.—Hysteria has no recognized pathology. It is a so-called functional disease. The theory that it depends on minute structural changes in the neurons is possibly correct, for all function depends upon structure, but we have no way of ascertaining these changes.

Symptoms.—Hysteria is most common in children and young adults. It rarely appears after middle life.

The French divide the symptoms into two great classes,—the paroxysmal and the interparoxysmal.

The *paroxysm*, or fit, is divided into four periods. Prodromes or auræ may usher in the first period; the former usually are changes in temper and disposition, the latter are the *clavus*, a circumscribed pain in the head, the *globus hystericus*, a sense of a ball rising in the throat, and ovarian hyperæsthesia. Other and more rare prodromes and auræ are seen.

The *first*, or epileptoid, period is marked by a sudden, tonic spasm, in which the patient lies rigid or even in opisthotonus, with hands clenched, eyes fixed or even crossed, and arms extended in the position of a cross; the breath is labored, the pulse slightly accelerated, and consciousness is obtunded but seldom or never entirely lost. The tongue is not bitten, the pupils are not affected, incontinence of urine does not occur, and the patient does not injure herself in falling. It is this stage which most closely resembles epilepsy. But the clonic spasms which supervene are not exactly like those of epilepsy; they are usually more irregular, and often have something of a voluntary aspect. The eyelids present a slight tremor.

The *second* is called by the school of Charcot the period of "clownism." The patient throws herself into grotesque attitudes; she seems as one possessed, and indeed she was believed, in the Middle Ages, to be controlled by a demon. Extreme opisthotonus is one of the commonest positions assumed by the grand hysteric.

In the *third* period the patient seems to act a part; she is dramatic, sometimes pathetic, always extreme. This is the histrionic stage, in which the conduct is evidently the mirror of certain mental states. It has been greatly elaborated by the French school, and by means of hypnotism and suggestion has been not a little overdone.

The *fourth* period is that of delirium—so-called. The patient subsides into a state of weeping and declamation; sometimes there are spells of [illegible].

The [illegible] fit is not al[illegible]ical; there are aberrant or abortive [illegible] epite[illegible] [illegible] whole tableau. The first period is the commonest, with a brief histrionic display, followed by a crisis of weeping and laughter. Among the very rare aberrant forms are ecstasy, somnambulism, catalepsy, and trance. Tourette and Cathelineau have tried to show that the nutrition is affected in a characteristic way; there is loss of weight, with increased excretion of urea, but during the lethargic trance-like stage the urea diminishes. Some of the sensational stories of the dead returning to life, as in the case of Lady Russell, who revived at her own funeral, were doubtless founded on cases of hysterical trance.

The *interparoxysmal* symptoms are motor, sensory, and visceral.

The *motor* symptoms are paralysis, contracture, tremor, and incoördination.

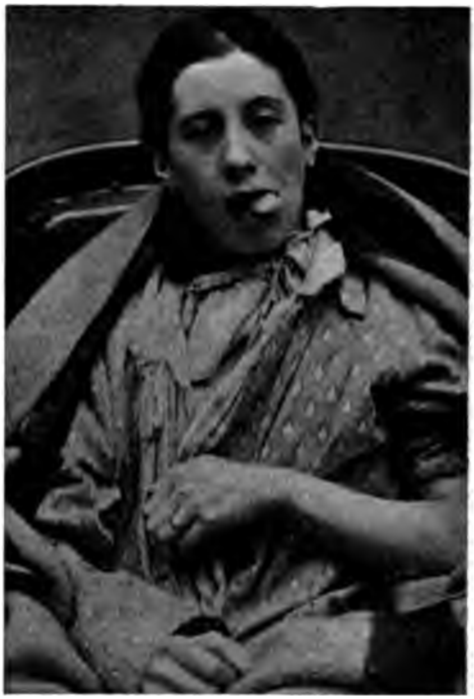

Fig. 402.—Hysterical hemiplegia, showing glossolabio-brachial spasm of left side.—Stewart.

The paralysis may take the form of a monoplegia, a hemiplegia, a paraplegia, or a total palsy. It is sometimes limited to one or a few muscles, as of the hand, arm, face, tongue, pharynx, or larynx. The paralysis in the extremities is likely to be accompanied with contracture; the deep reflexes are not abolished, nor is there true muscular atrophy, or reactions of degeneration. The paralysis is not, as a rule, limited to the distribution of particular nerve-trunks; in other words, it is *central*, not peripheral. The paralyzed part may become œdematous, blue, and mottled, especially in traumatic cases. Hysterical paralysis is usually persistent for long periods, but occasionally it is transitory and recurring; and mild grades may even be transferred from side to side. Sometimes a permanent cure is effected suddenly. In hemiplegia the leg is usually more paralyzed than the arm, and the face and tongue are not affected. In paraplegia there may be anuria but not incontinence. In total paralysis all four limbs are involved, but the face and trunk escape. Chevalier was able to find only 21 authentic cases on record.

Contracture sometimes coexists with paralysis, but the rule is not universal. The paralyzed limb is not always contractured, neither is the contractured limb always paralyzed. The contracture does not always relax during sleep, but it relaxes under ether or chloroform. It may come and go; in some cases it is painful, and it can sometimes be re-established by pressure on the main nerve-trunk. Surprising cases are on record of long-persisting hysterical contractures. They sometimes follow trauma, or the grand convulsion, or sudden shock, and are often accompanied with other stigmata, such as aphonia, anæsthesia, etc.

Tremor is of several types: the most common is that which resembles the intention tremor of multiple sclerosis; of wide amplitude, absent during repose, increased by volition, and likely to be caused by trauma, or by metallic poisoning (lead and mercury). The "type Rendu" closely resembles this tremor, except that it may persist during repose, and is merely aggravated by volition. Dutil has also described a very fine tremor of from 8 to 12 vibrations to the second. Westphal's pseudosclerosis is doubtless a form of hysterical tremor.

Astasia-abasia is one of the curios of hysteria. It consists of a loss of power of standing (astasia) and of walking (abasia). There is no true loss of power or any necessary loss of sensation, and when the patient sits or reclines there is usually no incoördination. Progression on all fours is even possible. The gait consists in a series of wild, incoördinate movements of the legs, with alternate bendings backward and forward of the body. But little progress is made, and the patient requires support on each side. There is sometimes an alternate stiffening and relaxation of the back and legs, causing a tendency to opisthotonus and a rising on the toes. Astasia-abasia is most likely to be caused by trauma and emotion, and is most frequent in young persons.

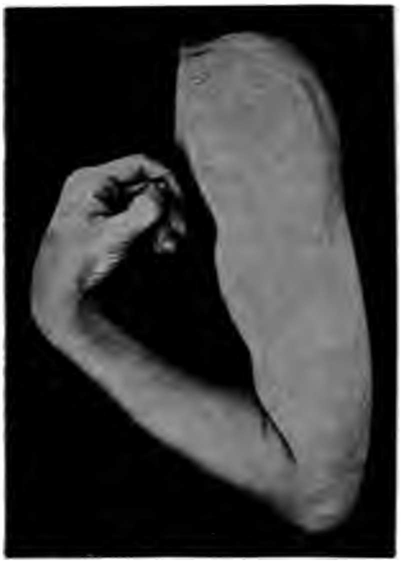

FIG. 403.—Hysterical contracture.—Lloyd.

Sensory changes consist of anæsthesia, hyperæsthesia, and paræsthesia. Anæsthesia is of various kinds, such as hemianæsthesia, segmental anæsthesia of a limb, and anæsthesia in patches. Hemianæsthesia is usually complete; that is, it extends from the crown of the head to the sole of the foot, and is often accompanied with anæsthesia of the mucous membranes of the eye, nose, tongue, mouth, and throat. It is sharply delimited at the median line, and can sometimes be transferred from one side to the other by suggestion. The special senses, sight, hearing, smell, and taste, may be involved on the affected side. Segmental anæsthesia of a limb is not uncommon; the area is sharply delimited above by a transverse boundary line, thus presenting the shape of a stocking or a gauntlet. Irregular anæsthesia in spots and curious geometrical figures, scattered at random over the surface of the body, and changing repeatedly, is quite characteristic. These various sensory stigmata play, and have played, an important rôle in hysteria. In the Middle Ages, during the witchcr[illegible]es, they were known as the "marks of the devil" (*stigmata d*[illegible]thesia of hysteria, in whatever form, is ve[illegible]ven involves the subcutaneous tissues and the nerve-trunks. The patient may not know of its existence, and it requires careful tests for its demonstration. According to Pitres hysterical anæsthesia is never isolated tactile anæsthesia; in other words, one or more of the other forms of anæsthesia—such as analgesia, thermo-anæsthesia, and even electro-anæsthesia—are always present, and in some cases there is a loss of sensibility to all modes of sensation. Loss of muscular sense is a rare phenomenon. The electro-anæsthesia may be preserved, however, when all other modes are lost, but not inevitably. Hyperæsthesia is found in certain zones or territories, as along the spine, and especially in the ovarian region, where pressure may excite a fit or cause other hysterical stigmata. These are the so-called hysterogenous zones. Paræsthesia, consisting of altered sensation, is not so common or significant.

Of the special senses the eyes present the most important changes; there is contraction of the visual fields, and reversal of the color fields, the red being larger in extent than the blue. Hemianopsia is rare. Total blindness or amaurosis has been noted. Blepharospasm, or spasm of the orbicular muscle, is seen, and may be mistaken for paralysis (ptosis), but true paralysis of any of the ocular muscles is extremely rare. Lloyd has seen true iridoplegia in hysteria. Spasm of one or other ocular muscle may cause strabismus, and be mistaken for paralysis of the opposing muscle.

Hysterical deafness, anosmia, and loss of taste are occasionally seen.

Visceral symptoms of various kinds are observed. Anuria is not uncommon; the patient may even require to be catheterized—a bad procedure in hysteria, since it tends to confirm the weakness. Incontinence is not seen. Hysterical vomiting—*anorexia nervosa*—consists of a regurgitation of food, rather than a true vomiting. The food is rejected before it reaches the stomach in most cases. The curious habit known as *merycism*, or chewing the cud, in which the patient regurgitates and remasticates the food, is a closely allied symptom. In hysterical vomiting there is no nausea, but the patient may emaciate and present other hysterical stigmata; in some cases, however, the nutrition is wonderfully preserved. Rapid respiration is sometimes seen, the respirations running as high as seventy or more to the minute; but the pulse is not accelerated, the color remains good, and there is no real dyspnœa. The breathing is shallow or panting. Occasionally, without increased breathing, there is tachycardia, which may even persist in spite of prolonged rest in bed. Persistent cough is sometimes a perplexing and exasperating symptom, as are also bouts of yawning. The cough is unattended with the physical signs of lung disease; and the yawning occurs in paroxysms, much exaggerated and prolonged, but not noisy. Aphonia is not rare. The patient may talk in a whisper, but sometimes is quite speechless, and even voiceless. Instances are reported of the natural voice returning during laughter or even during sleep, but a cure does not necessarily follow. Phantom tumor can be caused by contracture of the abdominal muscles. The French writers describe pyrexia, or pseudopyrexia. Some of the temperatures recorded are quite incredible. The subject requires further study.

The *psychical* state in hysteria is most important, for it is the essential one. To investigate it requires expert knowledge and skill, and it is sufficient here to say that one of its chief features is suggestibility, in which the patient's mind is peculiarly impressionable to outside influences. Frend's theory of "repressed emotions" need not be discussed here.

Diagnosis.—To begin with, the practitioner should disabuse his mind of the vulgar prejudice that hysteria is a simulated disease. The hysterical patient is not a humbug or malingerer. The affection is very real, and these patients are genuine sufferers. Many of them are useful members of society; some of them, to be sure, are weaklings, and a few are even degenerates, but they are none the less entitled to consideration.

It is commonly said that hysteria simulates all diseases; but the truth is that it simulates none exactly. There is always something *sui generis* in the hysterical stigmata. On the other hand, hysteria itself is sometimes simulated by designing persons, especially young women, but the counterfeit is usually detected with ease. No person can simulate successfully, especially for long periods, the paralysis, the contracture, the anæsthesia, or in fact any of the more important stigmata of hysteria. If any one doubts this, let him try to simulate contracture of the arm for a week.

There is often confusion between hysteria and neurasthenia, especially in the traumatic cases; and, in fact, many of the so-called traumatic neuroses are hysterical. The two conditions merge into each other, and the dividing line is not easily determined in some cases. The mental state of suggestibility is highly characteristic of hysteria; also the tendency of symptoms to come and go, and to be influenced by hypnotism. In genuine neurasthenia this is not so marked. Moreover, in neurasthenia we do not see the characteristic permanent stigmata of hysteria, such as the paralyses, anæsthesias, aphonia, anuria, etc.

The hysterical fit can closely simulate that of epilepsy, but there is no biting of the tongue, no frothing at the mouth, no injury to the person, no involuntary passage of urine, and the pupils are not affected. The state of the pupils may be a criterion of great value, for in the epileptic fit the pupils dilate after a momentary contraction in the tonic stage. In mild cases, however, such as petit mal, the pupil in epilepsy may respond to light. The state of the consciousness is appealed to by many as a true test of epilepsy, but it is not always reliable. Doubtless the profound unconsciousness of grand mal is not seen in hysteria, but in petit mal the consciousness is often but momentarily confused, hardly lost, and these are cases that might be simulated by hysteria. Yet hysteria is not usually so momentary as petit mal; and in the hysterical fit, it is true that, as a rule, consciousness is not so completely abolished as in grand mal. The hysterical fit can sometimes be induced by pressure on the ovarian region, and it is likely to be followed by hysterical stigmata, such as anæsthesia or even paralysis. Finally the last three periods of the hysterical fit are determinative, for they are never seen in epilepsy; but neither are they always seen in hysteria. It is important to bear in mind that hysteria and epilepsy can coexist in the same patient and present a confusing picture, but the crises are separate.

The diagnosis of the various permanent stigmata, such as paralysis, tremor, astasia-abasia, anæsthesia, vomiting, phantom tumor, etc., has been indicated in the description of those symptoms. Taken alone, they sometimes closely simulate organic disease, but they are usually associated with other hysterical stigmata, and this fact and the history are determinative. Ovarian pain, hysterical in origin, is not seldom mistaken for evidence of organic disease, and women are thus subjected to operation and needless mutilation. To guard against this too common error the practitioner should study his case well with reference to other hysterical stigmata.

IV. HYPNOTISM.

It is impossible to define hypnotism, or hypnosis, in satisfactory terms. It is a mental state, resembling, but not identical with, sleep, in which consciousness is variously affected, but in which the mind usually remains open to suggestion, especially from the person who stands in relation of hypnotizer to the patient. In the opinion of some good observers hypnotism is merely a form of induced hysteria.

Pathology. — Like all the psychoses, hypnotism cannot be said to have a recognizable pathology. It is common in hysterical patients, but neurotic and imaginative persons are also susceptible. It is rarely seen in the insane.

Symptoms. — We may recognize here the three stages of Charcot, merely premising that they are not sharply defined in all cases, especially the minor cases.

In the *cataleptic* stage the patient assumes a statuesque attitude, with partially opened eyes, blunted sensibility, and a readiness to receive and act upon suggestion. In many cases it is not possible to proceed beyond this stage, which may be regarded as a minor form of hypnotism.

In the *lethargic* stage the patient passes into a more sleep-like state. The special senses and general sensibility are much impaired, the muscular system is relaxed, and the consciousness is deeply affected. Such patients are not as open to suggestion as in the preceding stage. They are usually highly neurotic individuals, and in some cases they may even pass into a trance.

In the *somnambulic* stage we see a state which has often been miscalled "double personality." The patient's special senses are acute, but she is oblivious of much that transpires about her, although open to suggestion from the hypnotizer. The patient seems to be acting a dream.

Hypnotism can usually be excited by fixing the patient's attention on some particular object and keeping the eyes in a strained and fixed position. The French use bright objects, revolving mirrors, etc. The patient gradually becomes drilled and goes into the hypnotic state on the slightest provocation.

Much speculation, and much that is merely fanciful, has been written about hypnotism, but it is sufficient to bear in mind that it is a psychosis, or mental affection, in which the patient exists in a sort of dream-like state, in which suggestion from without can be made to play a prominent part, and in which hysteria is always an important factor.

V. NEURASTHENIA.

Neurasthenia is defined by Savill as a state of irritable weakness of the entire nervous system, characterized by hypersensitiveness, headache, inaptitude for mental work, disturbed sleep, irritability of temper, restlessness, nervousness, vague pains, and affections of the vasomotor and sympathetic systems. The disease has been much exploited in recent years, for it is not uncommon, especially among the overworked populations of our large cities.

Pathology.—There is no recognized pathology. The nerve centres are at fault, and the most plausible explanation is that these centres are not properly nourished. There is, however, a large mental element in these cases, and, as in all psychoses and neuroses, the affection is usually defined as functional. Among the causes, heredity, overwork, trauma, and the excessive use of alcohol and tobacco are the commonest.

Symptoms.—The symptoms are so multiform that only a generalized view will be attempted here for purposes especially of diagnosis.

The cardinal symptoms are mental ones—the despondency, the inability to apply the mind and to work, and the general nervousness. Some patients are not a little hypochondriacal, and in others an hysterical element is present, but the disease is not essentially either hypochondria or hysteria. Introspection and discouragement are prominent. Upon this psychical state are ingrafted some characteristic bodily ailments.

Headache is not uncommon, and a hypersensitive spine is frequent, especially in traumatic cases and in women. There are sensitive points along the spine, and the least exertion aggravates these and causes a sense of exhaustion. Pain may also be transmitted to the limbs, and sometimes there are bodily pains suggestive of visceral disease. Disturbance of sleep is common; there is either insomnia or broken and restless sleep, so that the patient arises in the morning unrefreshed. Ugly dreams are often a feature of the traumatic cases, the patient seeing again the frightful accident through which he has passed. Nutrition is sometimes greatly impaired, the patients emaciating and becoming anæmic; and they are often bedridden, especially if they are women, and present the appearance of extreme illness. Inability to take and digest a sufficient quantity of food is a troublesome feature; and there may even be dilatation of the stomach or gastroptosis. On the other hand, some neurasthenics are remarkably well nourished; these are the fat neurasthenics, who are not the least troublesome patients. Morbid blushing and flushing are sometimes seen; and the heart may be accelerated in spite of prolonged rest. Palpitation annoys these patients on slight exertion. Paralysis is not commonly seen, but in the traumatic cases there may be inhibited or impaired movement on account of pain. Genuine paralysis points either to hysteria or to organic injury. The same is true of anæsthesia; it is rare, and its presence is usually due to an hysterical element. Tremor is occasionally seen, and requires to be carefully distinguished from other tremors, such as those of alcoholism and hysteria. The knee-jerks are usually free, sometimes exaggerated, never lost.

Of the special senses the eyes suffer most; there may be eye-strain and pain on using the eyes. Tinnitus, vertigo, and noises in the head, or a sense of fulness, are present in some cases. Some patients have a strange sense of mental vacuity.

Sexual weakness is not unusual in men, and the sexual act causes profound exhaustion, weakness in the back, and headache, which may endure for a day or so.

In this connection it is well to consider briefly the *traumatic neuroses.* They are in fact largely neurasthenic, although a few are purely hysterical. It is too much to say, however, that all these traumatic cases are purely functional. Some of these patients suffer from organic lesions, such as bruises, sprains, and wrenches of the muscular and tendinous attachments, especially of the spine. The so-called "railway spine" is not always purely neurasthenic, but may be in part the result of shock and sprain. The same may be said of some of the obscure injuries to the hip and other joints; there is a large neurasthenic element, but it is very often not the whole story. These cases are of exceptional importance because they lead so often to litigation; and the controversy over them is frequently acute.

All neurasthenics are quickly and easily fatigued, both by mental and physical exertion. Inability to concentrate the mind is not uncommon; and of other mental phenomena the most important are the so-called *obsessions.* They consist of imperative and inhibitive ideas, and are seen in the state known as *psychasthenia,* which belongs rather to psychiatry than to clinical medicine.

Diagnosis.—Since Beard invented the term, neurasthenia has been used very loosely to cover a wide variety of symptoms. When properly guarded, however, the term has a legitimate use, and, although it is hardly capable of exact definition, it covers a symptom-complex which is fairly recognizable; and this has been described above. The diagnosis must depend upon a careful consideration of those symptoms, and the practitioner must not forget that a neurasthenic state often accompanies other diseases, even grave organic ones. The only rule is to exercise care and judgment, and to go by a process of exclusion.

The most common error is to confuse neurasthenia and hysteria; in fact, some writers in describing the traumatic neuroses do not hesitate to include hysterical symptoms indiscriminately with those of neurasthenia. This is wrong. Hysteria is a much more clearly defined disease than is neurasthenia, and it should be kept apart whenever possible. The mental state, and the various stigmata, such as paralysis, anæsthesia, contracted visual fields, not to mention the convulsions, are enough to distinguish it in most cases. It is often caused by trauma, and frequently figures in court in damage suits, as neurasthenia. There is no doubt, however, that hysteria can coexist with neurasthenia, just as it can coexist with epilepsy.

Hypochondriasis is a state in which there are delusions about the health, rather than real disorders of the health. The hipped state of mind in some neurasthenics suggests a resemblance, but there are not the deep-seated hypochondriacal delusions in the one, nor the genuine sufferings in the other.

In traumatic cases it is important not to overlook organic injury and to call everything neurasthenic. Grave errors have thus been committed.

Secret drug habits, especially the use of morphia, sometimes induce a neurasthenic state, and the true nature of the case may be overlooked. Hence it is important to inquire carefully into the habits. The same is true of alcohol and tobacco, and even of tea and coffee.

Some of the victims of onanism can properly be classed as neurasthenics, but in them there is usually a marked hypochondriacal element. These persons are the easy prey of the advertising charlatans, and their symptoms are sometimes concealed or repressed for fear of exposure.

VI. THE OCCUPATION NEUROSES.

These include a variety of affections which arise from overuse in the course of occupation. They are sometimes called the fatigue neuroses, and are located largely in the neuromuscular apparatus. The commonest forms are scrivener's palsy, telegrapher's cramp, piano-player's hand, and some forms of clergyman's sore throat; and some not so common are seen in fiddlers, bricklayers, and others.

Pathology.—The objection to the term "neurosis" in this connection lies in its implied meaning that the disease is functional. No disease is purely *functional* in the sense that it does not depend on organic change, for there must be some change, however slight and transient. In some of these cases we even see evidences of structural change, such as muscular atrophy, persistent pain, etc.; and we may regard all of them as instances of disordered nutrition both of the nerve-cells and of the muscular attachments. Farther than this it is not possible to go.

Symptoms.—It will be best to consider a few of these affections in regular order.

Scrivener's palsy, or writer's cramp, is marked particularly by spasm, tremor, and incoördination. Pain and vasomotor disorders are sometimes seen. Paralysis and anæsthesia are doubtful symptoms. Three types are noted,—the spasmodic, the paralytic, and the tremulous,—but the distinction is not always clear in practice. The spasm affects chiefly the small muscles engaged in writing, and as a rule is only manifested on attempts at writing—not in other coördinate movements. There are exceptions, however, to this rule. The muscles are held in cramp-like rigidity, wholly preventing the act of writing. In some cases the muscles of the forearm, upper arm, and shoulder are affected, and rare cases are seen in which even distant muscles, as those of the foot, or of the other hand and arm, are involved. The spasm is not a true cramp, for it is non-painful; but exceptions to this rule occur. It is quite involuntary, and may even persist for some moments after the attempt at writing has ceased. Tremor and incoördinate jerky movements are sometimes seen. Paralysis is extremely rare, but a stage of slight paresis may follow the cramp. Pain, or a painful sense of fatigue, is sometimes present, together with painful points on the nerve-trunks. Neuralgic pains are observed in a few cases. Anæsthesia is very rare. It is probably an hysterical symptom. Vasomotor and trophic disorders are not common; turgescence of the limb and flushing of the face are among the former; and such observers as Eulenburg and Gowers claim to have seen muscular atrophy. It has also been seen in a blacksmith, in a saddler, in a tailor, in a dragoon (from holding the reins), in a morocco-worker, and in a player on the bass violin. Atrophy of individual muscles, or small groups of muscles, is seen in some artisans who overuse these muscles, as the small muscles of the hand in locksmiths; and ulnar paralysis and atrophy in glass blowers from pressure rather than overuse.

Telegrapher's cramp is closely allied to the preceding; in fact it is identical with it except that the cause and the seat of the spasm differ. It was first described by Onimus in 1875 and called by him *mal télégraphique.* Fulton studied the movements engaged in using the Morse instrument, which particular instrument has been held most to blame. The letters are made by a series of dots and strokes which require a fine muscular movement, and when the dispatcher is working rapidly and for long stretches the strain on the muscular apparatus is great. Fulton estimated that an operator makes between thirty and forty thousand contractions per hour. The word "occupation," for instance, contains ten letters and requires twenty-five distinct impressions. One of these patients had worked as long as fourteen hours a day. Another said that his first difficulty arose in making the letter B (— - - -); his hand would act involuntarily and make the dash and four dots (— - - - -) which means the numeral 8. Finally his greatest difficulty was experienced in making the letter P (- - - - -), the rapid succession of dots causing a cramp. In another patient the cramp was painful and was located in the extensors, pulling the hand away from the key (Fig. 404). Worry and discouragement are often seen in these operators; it is easy to make mistakes in transmitting, and these may be serious; consequently a neurasthenic state is often present, which is not improved by alcohol and tobacco.

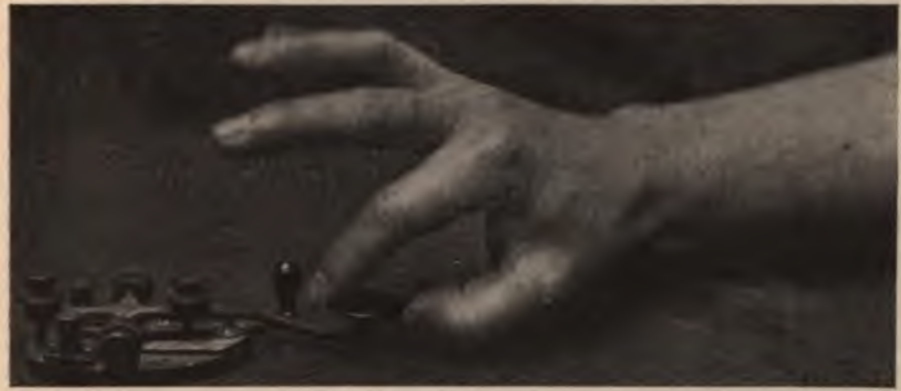

FIG. 404.—Telegrapher's cramp.—Lloyd.

The telegraph operator can train his other hand rather more easily than the scrivener, because the movements are less complex; but in time the second hand is likely to become affected in both cases. It is easy for the disabled telegrapher to become the victim of writer's cramp.

The *dysphonia* and *aphonia* of professional voice-users have many points of similarity to writer's cramp. There is a spasmodic type and a paralytic type. In singers the voice may break down in the midst of song, although the natural voice for speaking may remain, thus showing how a particular *function* is involved. In the so-called clergyman's sore throat there may really be some pharyngitis or laryngitis, but the nervous element too is usually well marked. There is indeed a well-marked hysterical element in many of these cases of aphonia. Thus Mackenzie reported the case of a fish-hawker who lost his voice suddenly while crying his wares; he was voiceless for four months, and was then cured promptly with electricity.

It is needless to describe in detail the long list of the fatigue neuroses. Among the artisans and artists affected are grinders, blacksmiths, engravers, cigarette-rollers, pianists, violinists, bricklayers, tailors, and typesetters. Milker's spasm is seen in this country and is said to be common among the cow-herds of the Tyrol. Miner's nystagmus is a curious form.

Diagnosis.—The history and the symptoms are unmistakable. It is always well to recall that a neurasthenic state is often seen in these patients, and that it may complicate the clinical picture. This is still more so of hysteria. Some of the symptoms seen are distinctly hysterical, as, for instance, paralysis and anæsthesia in writer's cramp, for these two symptoms are probably never seen in pure cases. The same is true of aphonia in habitual voice-users; it usually bears all the marks of an hysterical affection. Still, these facts do not necessarily obscure the diagnosis. Writer's cramp has been confused with other diseases in which writing is affected. Among such diseases are hemiplegia with aphasia and agraphia, general paresis, locomotor ataxia, paralysis agitans, disseminated sclerosis, chorea, progressive muscular atrophy, alcoholic and metallic poisoning, and neuritis.

VII. MIGRAINE.

Hemicrania, migraine, or sick headache, is one of the explosive neuroses. Its chief symptoms are disorders of vision, pain in the head, and vomiting. It is paroxysmal, but has no regular periodicity, as a rule, and it is often hereditary or familial. Möbius claimed that 90 per cent. of the cases show heredity.

Etiology.—This is unknown. English physicians are fond of associating "megrim" with gout, and in France it was Trousseau who said, "migraine and gout are sisters." But these assertions are not proofs. Reflex causes, eye-strain, adenoids, womb disease, and even decayed teeth have been assigned as causes, without much reason. The disease may possibly be due to autoinfection, and gastro-intestinal disorders sometimes excite attacks.

Symptoms.—Migraine usually begins in early life. The initial symptom of the paroxysm is an aura, usually sensory, and in the vast majority of cases visual. The visual auræ consist of attacks of amblyopia in which a part or the whole of the visual fields is obscured; thus scotomata and even hemianopia occur. There are also scintillations, zigzag lines, shaped like a fortification, flashes of light, blazing or flaming splotches, and in very rare cases illusions, as of the forms of animals or terrifying apparitions. Oscillation of the pupil (the so-called *hippus*) is sometimes seen. Other sensory auræ occur, as a numb or tingling feeling in the face, tongue, or one limb. It is characteristic of the aura of migraine to disappear just before the onset of pain.

The pain of migraine is an intense neuralgia in the distribution of the fifth nerve. It is usually described as a hemicrania, but it is not always confined to one side. Occasionally, however, it is strictly localized in one branch of the trigeminus, especially the ophthalmic division. The pain increases gradually, until it reaches its acme, and it may endure only for a few hours or even for a day or two. It is usually terminated by the occurrence of vomiting, which thus constitutes the third or terminal stage of the paroxysm.

Gastric disorder is one of the three characteristic symptoms of migraine. Aversion to food, and even nausea, may occur early in the attack, and sometimes vomiting begins early; but commonly vomiting is a late or terminal symptom. In many cases the patient is promptly relieved by emesis.

Aberrant types of migraine are seen, in which one or other of the cardinal symptoms is wanting. The commonest is the mild form in which the visual aura is followed by slight headache, which is only transient, and the paroxysm aborts without the third stage, or stage of vomiting.

A curious and rare form is the *psychical migraine*, in which mental disorders of various kinds predominate. There is confusion of ideas, with emotional excitement or depression, attending the visual aura, and pain may or may not be marked. Incoherence and even aphasia have been noted, and Liveing attempted to show, with remarkable prescience, that the aphasia always occurs in cases in which the sensory aura, as numb-
ess of the hand, is on the *right* side, thus indicating a *left-sided* cerebral
sion. Genuine *substitutional* attacks have been noted, just as in epilepsy;
s Sir George Airy observed in his own person the attack complicated
transient impairment of speech and memory without either head-
or numbness. Tissot observed a case in which attacks of habitual
ine were at length completely replaced by fits of disordered ideation.
ria doubtless complicates some cases of migraine.

phthalmoplegic migraine is the form in which the paroxysm is com-
d with paralysis of some of the ocular muscles. The muscles
d are those usually supplied by the third nerve; but occasionally
rth or the sixth nerve is affected. The pain is usually severe,
followed quickly by the paralysis, which may endure for days or
weeks. The bout of pain is commonly terminated by a crisis of
ing, as in ordinary migraine, but the paralysis constitutes the
terminal stage, and may be total and complete in the third nerve;
is to say, all the muscles supplied by that nerve are involved and are
mpletely paralyzed. There is ptosis, external strabismus, and the pupil
oes not react to light or on accommodation. In some cases, however, the paralysis is not total or not complete. The duration varies. In Schilling's case the palsy lasted for from four to six weeks, and in Parinaud's case from two to three months; but in many cases the duration

is only for a few days. The paralysis, as a rule, to which there are few exceptions, always occurs in the same eye in successive attacks. In some cases the paroxysms observe a true periodicity; in Suckling's case they occurred every two weeks. Many of these cases date from early childhood. Occasionally a permanent palsy results after repeated seizures. Paralysis of the *fourth* nerve has been noted by Leizenberger; and conjoint paralysis of the third and fourth has also been seen. Paralysis of the *sixth* nerve, either alone or in association with some of the fibres of the third nerve, has been reported. De Schweinitz saw a case of abducent palsy, with migraine, which had begun in early life. A very rare case is that of Rossolimo in which a recurring paralysis of the *seventh* or facial nerve was always ushered in by a migrainous attack. Anæsthesia of the *fifth* nerve has been noted in a very few cases, especially in the first and second divisions, or even in the supra-orbital branch alone. Troemer has reported a case of *ophthalmoplegia interna* following severe attacks of migraine. The pupil was widely dilated and immobile to light, but the other ocular muscles were not affected.

All forms of migraine tend to grow less or even to disappear in middle life.

Diagnosis.—The ordinary migraine is easily recognized; in fact, the patient usually knows well enough himself what he has. The beginning in early life, and the peculiar evolution of the paroxysm, from the aura to the critical vomiting, are unmistakable. Simple neuralgic attacks, which are not uncommon, are known by the absence of the true migrainous symptoms, such as the aura, the explosive onset, and the crisis of vomiting.

Migraine has been likened to epilepsy, especially by Hughlings Jackson and his followers, the resemblance being based largely on the abrupt onset with an aura; but the likeness is superficial. In migraine there are no convulsions, and the disease does not merge into epilepsy. Migraine and epilepsy may however coexist in the same person, but the attacks are separate.

Ophthalmoplegic migraine simulates organic disease, especially tumor and syphilis of the brain. The paroxysmal nature of the attack, however, and the tendency for the paralysis of the third nerve to disappear are against brain tumor, as is also the history of the case. In brain tumor, moreover, there are usually other symptoms, such as choked disk and other and more wide-spread paralysis, and the course is progressive.

Syphilitic meningitis between the peduncles causes paralysis of one third nerve, sometimes of both third nerves, and this paralysis may even be evanescent, with severe headache; but the history is not that of migraine, nor is the onset so abrupt, the cure so complete, and the headache so paroxysmal, with a crisis of vomiting, as in migraine. Moreover, there are likely to be other nerves involved in syphilis, such as the optic nerves, or even a hemiplegia, and the symptoms are irregular. Ophthalmoplegic migraine is always unilateral; syphilis may or may not be so. One of Charcot's cases of migraine had paralysis first of the sixth nerve on one side, then of the third nerve on the other, but the case was unique.

Paroxysmal or recurrent palsy of the oculomotor nerve has been noted also in tubercular meningitis, in otorrhœa, and in nasal catarrh, but the history in these cases and the associated symptoms are against mere migraine.

Some observers claim a relationship between tabes dorsalis and migraine —suggested by the recurrent palsy of the third nerve, sometimes seen in tabes. But it is not scientific to call the recurrent palsy of the third nerve in tabes migrainous; for this palsy is probably due to nuclear disease, and it is to be known by its association with other tabetic symptoms. The same may be said of the association of migraine and general paresis.

Hysteria may unquestionably complicate or simulate migraine, especially in the emotional and psychic symptoms, and in the contraction of the visual fields, which might suggest scotoma. But genuine paralysis of the third nerve is probably never seen in hysteria, although it is sometimes simulated by a blepharospasm.

VIII. PARALYSIS AGITANS.

This affection is usually called a disease of old age; nevertheless it occasionally begins in comparatively early middle life; seldom, however, before the age of forty. It is also called shaking palsy, or Parkinson's disease.

Pathology.—The cause and pathology are obscure. Recently C. D. Camp has made an elaborate study of a series of cases, many of them from the Blockley Clinic in Philadelphia, in which he endeavors to show that the essential changes are in the muscular fibres; and he suggests that there may be alteration in the secretion of the parathyroid glands. The ordinary changes of senility, such as atheroma of the cerebral blood-vessels, are commonly found in these patients, but it does not follow, as Dana and a few observers have suggested, that these changes are causative. Some sclerosis of the posterior and lateral tracts of the cord is occasionally observed; also some atrophy of the cerebral convolutions, pigmentation of the motor neurons, and increase of interstitial tissue.

Symptoms.—The disease is of gradual onset and chronic course, and it is rather more common in men than in women. The chief symptoms are tremor, rigidity, paralysis, and affection of the gait. It usually begins with tremor in the hands, sometimes more marked at first in one hand.

The tremor is a regular to and fro or up and down movement, especially marked in the hands, persisting during repose, and temporarily arrested by voluntary motion. The amplitude varies; sometimes it is slight, at others wide and violent, particularly if the patient is aroused or excited. The arrest on volition is but for a moment; the tremor then returns in spite of the patient, and may even be aggravated for a short period. This is seen on attempts at writing. In the lower limbs the tremor is usually not so marked, and is sometimes even absent. It is seen occasionally in the facial muscles, and in the tongue, especially when it is protruded. The statement that the head is not involved is not correct for all cases. Nystagmus is not present. The tremor ceases during sleep.

Rigidity is usually most marked in the hands, arms, back, neck, and face, and its effect is to give the patient a peculiar attitude and expression.

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Westphal, were doubtless of the same class. In most of these traumatic and hysteroidal cases the tremor is usually of the intention type, that is, it is worse on voluntary movement, and it is rather coarse; moreover the history and progress are suggestive. These cases often occur in young persons; the attitude, gait and expression are not those of Parkinson's disease; and, finally, hysterical stigmata are often present. In an occasional case the tremor may be fine.

The tremor due to metallic poisoning (by lead and mercury especially) is similar to the hysterical tremor.

The tremor of alcoholism is also largely of the intention type and is coarse and irregular; it is easily known by the history.

IX. TETANY.

This disease is characterized by tonic, cramp-like spasms, especially in the fingers, hands, and upper limbs. It occurs in epidemics in some countries, especially in and about Vienna, Northern Italy and Sweden. The cause is obscure. Tetany may be of infectious origin, and it is also seen in ergotism, in the diarrhœa and rickets of young children, in nursing women, after extirpation of the thyroid gland, and in dilatation of the stomach. Chvostek advances the theory that the real cause is a defective action of the parathyroid glands. According to the statistics of Fränkl-Hochwart, shoemakers and tailors seem peculiarly vulnerable. The disease is rare in America.

Pathology.—This is quite unknown, for post-mortem findings have not been uniform or characteristic.

Symptoms.—The tetanic spasms are oftenest seen in the small muscles of the fingers and hands, causing flexion of the basal phalanges, extension of the distal phalanges, and turning in of the thumb. This is sometimes called the "obstetric hand." The wrist and elbow are flexed. The toes may be similarly affected; also the calf muscles.

The spasms are often painful, and usually intermittent. They can sometimes be excited by pressure on the nerve-trunks or main vessels—Trousseau's sign.

There is increase of the mechanical and electrical irritability. Tapping on nerve-trunks causes a lively contraction (Chvostek's sign), often well marked in the face; and the galvanic current, even of mild power, causes active, even tetanic, responses, which may increase, rather than diminish, with repeated stimulation. The nerves may be hypersensitive to pressure.

Other muscles, such as of the trunk, tongue, and respiration, are involved in severe cases, and the ocular muscles, according to Kunn, may be affected. Nystagmus has been seen. Other ocular symptoms noted by a few observers, and which are evidently very rare, if they belong to the disease at all, are neuroretinitis, optic atrophy, unequal pupils, diplopia, cramp of the eye-muscles, especially blepharospasm, reddening of the conjunctivæ, and lachrymation. Slight paresis and anæsthesia (or hypæsthesia) are present in the limbs after severe attacks of the cramps.

There is no affection of consciousness, as a rule, but in severe cases constitutional reaction, such as fever, accelerated pulse, etc., is seen.

Sometimes the disease ends fatally, especially when caused by some of the more serious conditions mentioned above. A patient in the Philadelphia Hospital died of exhaustion and pulmonary œdema; but the prognosis appears to be good in the majority of simple cases.

Diagnosis.—The tetanic symptoms are so characteristic that the diagnosis is easy.

The cramps seen in chronic uræmia are not to be mistaken for tetany.

The French describe an hysterical type of tetany, but it cannot be made to cover all cases; when the disease is truly hysterical the mental state and the presence of other stigmata suggest the right diagnosis, and such signs as Trousseau's and Chvostek's are wanting.

It is probable that a variety of affections cause tetanoid symptoms. and further study is necessary to shed light on these obscure cases, Tetany has no relation to true tetanus, and does not resemble it.

X. THE TICS.

The tics, or *maladie des tics*, or habit spasms, are peculiar motor disorders in which one or more groups of muscles are thrown into regular and oft-repeated contractions, resembling a voluntary act. The muscles most frequently affected are those of the eyes, face, mouth, tongue, and neck, but in some cases the muscles of the limbs are involved.

Pathology.—The French, who have described the tics most carefully, point out the underlying mental state. The tic has a quasi-voluntary character, and in its origin it is volitional or impulsive. Gradually the movement becomes fixed as a motor habit, and can no longer be controlled, or only imperfectly controlled, by the patient, and then at the cost of mental distress and anxiety. Usually there is no very distinct motive or associated idea in the mind; the tic is merely an impulse, which grows into a habit. In proof of its psychic character, however, is the fact that in some rare cases the movements are thus associated with definite ideas in the patient's mind, emotional or otherwise, of which the tic is the facial expression; and in extreme cases there may be obsessions, especially of speech, the patient being impelled to give utterance to some set of words, even an indecency—the so-called coprolalia. In fact, the tics are closely allied to the obsessions; they are due to a sort of imperative motor impulse, and they are usually found in neurotic and degenerate patients.

Symptoms.—As no two cases are exactly alike it is difficult to give a brief description of the tics. Blepharospasm is common, and with it is often seen an associated movement of the face and even of the tongue and larynx. Odd grimaces are the result, and these may even be, or seem to be, the expressions of various mental states, as grief, surprise, pain, or joy. When unilateral, as is often the case, the one-sided expression can best be interpreted by covering the sound side of the face. In many cases, however, there is no such expression, but merely a distortion of the features. When the neck muscles are affected there are various movements simulating torticollis. Sometimes associated movements of the arm, hand, leg, or foot are seen.

The tics occur in regularly recurring bouts, the intervals varying from a few minutes to much longer periods. They can sometimes be controlled for a time by an effort of will, but the effort causes mental discomfort, and the patient seeks relief in what Church has called a "spasmodic debauch," in which for a time the tic is repeated rapidly and frequently.

The tics commonly begin in childhood or in young persons. Occasionally they appear later in life; and blepharospasm may have its origin in some affection of the eye, and gradually become fixed as a habit.

Diagnosis.—As already said, the tics are allied to the obsessions and to various neuroses. They are easily recognized by the conspicuous motor disorder; yet they have sometimes been confused with forms of epidemic hysteria, such as in the "jumpers" of Maine and Canada, and the disease known as "latah" among the Malays, and "myreachit" in Siberia, in which imitation and suggestion are prominent factors. But the tics are not a form of hysteria; they do not occur in epidemics; they are but little influenced by suggestion; and they are too apt to be incurable. It is likewise an error to describe this disease, as some authors do, under the head of chorea. There is nothing choreic about it. In young children these habit spasms in the face, neck, and shoulders, at the very beginning, may suggest St. Vitus's dance, but the tic is more localized, more habitual, and more persistent than the movements in chorea, and there is always a volitional element in it. There is a mild grade of tic in children which tends to recover.

The true tics are to be distinguished from the spasmodic form of tic douloureux, which is a form of facial neuralgia in which the facial muscles are thrown into spasm by the action of pain. In the habit tics pain is absent.

Stammerers sometimes develop a kind of associated spasm in the face or even in a limb. When the embarrassment of speech is great the facial muscles are contorted, and even the hand and arm may be moved spasmodically. This cannot be called a tic in the proper sense of the word.

The tics differ from mere spasm by the psychic element in them. In pure spasm the affection is in the neuromuscular apparatus, and there is no mental collaboration. Such a spasm, purely local, oft repeated, and not involving consciousness, is sometimes seen in some isolated muscle-groups, as, for instance, in the head and neck, constituting the disease known as torticollis. It is usually not controllable by the patient's will, and the cause and pathology are obscure.

VASOMOTOR AND TROPHIC DISEASES.

I. RAYNAUD'S DISEASE.

This is a trophic disease in which the extremities, especially the fingers and toes, are the seat of recurring pallor, congestion, and even gangrene. The affection was first described by Raynaud in 1862. It is apparently of vasomotor origin, and has three types or stages—local syncope, local asphyxia, and local gangrene.

Pathology.—The disease seems to be essentially due to vasomotor disorder. Neuritis and arterial sclerosis, or endarteritis obliterans, are [illegible] progress, although they may be present as sec- [illegible] stages. This whole subject of the pathology, [illegible]

Symptoms.—[illegible] the parts become blanched, shrivelled [illegible] similar to that caused by exposure to cold. [illegible] dead fingers." The parts affected are usually the [illegible] the affection is symmetrical. The fingers feel numb, [illegible] anæsthesia and analgesia, but as a rule no loss of the sense [illegible]. The affection is most common in cold weather, and it is [illegible] and recurrent, the attacks lasting from a few minutes to [illegible]. There is no real paralysis, but the fingers may be awkward [illegible] from the numbness and stiffness. There is usually no [illegible] tingling and numbness.

[illegible] *asphyxia* the reverse of the preceding picture is seen; it is a [illegible] apparently, although it is not necessarily preceded by a [illegible] stage of syncope. The skin becomes dusky, red, purplish, [illegible] almost black; the parts, as the fingers, are congested; the surface [illegible] the tactile sense is impaired; and the members may be most pain- [illegible]. The radial pulse remains unchanged. As in syncope, the attacks are paroxysmal and recurrent, and even occur in cycles. The affected fingers are usually involved in turn, and the congestion disappears first from one part and then another. Occasionally the nose, ears, and face are invaded.

The local or *symmetrical gangrene* may follow either the syncopal stage or the asphyxial stage. The parts are cold, usually shrivelled, and bullæ form; these break and reveal black spots, which result in localized destruction. The ends of the fingers are destroyed, or the under parts of the toes. The spots sometimes heal, leaving a healthy scar. The pain is often severe.

Among accessory symptoms should be mentioned hæmoglobinuria, which, being associated with chill and slight fever, and having a paroxysmal course, has led to the suspicion of malaria; an idea which seems to [illegible] some favor with Barlow. Amblyopia is also occasionally seen, and may depend on alteration in the calibre of the retinal vessels, as verified by Galezowski. Temporary changes in the joints have been noted, also hemiplegia and aphasia, and in rare cases even mental symptoms. Epilepsy, [illegible] attacks suspiciously like it, has been reported.

Diagnosis.—There are various forms of gangrene which must not be mistaken for Raynaud's disease. The disorder closely resembles chilblain or frost bite, but the history is sufficient to prevent error. Raynaud's disease is a recurring affection, whereas frost bite is a simple and solitary accident, and by no means always symmetrical.

[illegible] resembles ergotism, but the history alone should distinguish [illegible].

[illegible] the gangrene of diabetes, in which the glycosuria and the [illegible] character of the gangrene are significant.

Erythromelalgia resembles the asphyxial stage of Raynaud's disease. [illegible] the former the affected limb is hot, pulsating, and more uniformly [illegible] the latter; moreover, it does not present sensory changes [illegible] gangrene does not result. Nevertheless, the two affections have some affinity. It has been claimed that they are identical.

Morvan's disease closely simulates the late or gangrenous stage of Raynaud's disease; but the former is closely allied to syringomyelia or central gliomatosis, and there is a peripheral neuritis and thickening of the arterial coats. Hence in Morvan's disease there is usually seen muscular atrophy in the extremities, anæsthesia of all the modes of sensation, changes in the deep reflexes, and possibly scoliosis or kyphosis. The history is not that of recurring paroxysms, and the whitlows are painless.

Injury to a nerve, especially the median nerve, may cause gangrene of the finger-tips, as in a case lately recorded by Sneve, but the history usually is clear, the affection is unilateral, and the paralysis and anæsthesia are characteristic.

Local gangrene may result from the obstruction of an artery, as in the condition called endarteritis obliterans, the pathology of which is obscure.

We do not agree with Oppenheim in confusing the various trophic lesions of tabes, syringomyelia, and even hysteria with those of Raynaud's disease. The associated symptoms of these diseases are sufficient to distinguish them.

Barker has recently called attention to the subject of *acrocyanosis* in which there is anæsthesia with gangrene of the toes. It seems to be a vasomotor affection and may have to be distinguished from Raynaud's disease by the difference in the state of sensation.

II. ERYTHROMELALGIA.

The affection to which Weir Mitchell gave this name in 1878 is described by that author as a chronic disease in which a part or parts of the body, usually one or more extremities, suffer with pain, flushing, and local fever, made far worse if the parts hang down.

Pathology.—The disease has often been ascribed to vasomotor disorder. Lately, however, the tendency has been to attribute it to a peripheral neuritis; although careful examination has not always supported this view. In most cases changes in the blood-vessels, as atheroma and arterial sclerosis, have been present. Finally it has been suggested that there is a central or cord lesion, and cases have been reported by Collier in multiple sclerosis, tabes, and myelitis. It is also seen in hemiplegia. The disease has been observed not infrequently in workmen who do heavy labor and stand for long periods; and Sturgis suggests that it has points of resemblance to the occupation neuroses, such as writer's cramp. On the whole the question of the pathology is still an open one. The weight of evidence seems in favor of a conjoint arterial sclerosis and peripheral neuritis; while a central influence may also act.

Symptoms.—When the limb hangs down it becomes congested and rose-red, or even of a dark violaceous hue; the arteries throb; and the local temperature of the skin rises. Pain is usually present, and may be severe, of a burning or neuralgic kind, worse on pressure, and generally relieved when the limb is again elevated; but sometimes it persists in a minor degree even then. It is worse in summer, and is relieved by the application of cold. Sensation of all kinds is preserved. The disease may be asymmetrical, and is not followed by gangrene, although a somewhat similar condition has been seen to precede senile gangrene. The symptoms, when in the feet, are usually provoked by walking and standing. It may be questioned whether erythromelalgia is a distinct disease, or anything more than a symptom of various diseases.

Diagnosis.—The distinction from senile gangrene is sufficiently shown in the account of the symptoms. In erythromelalgia the painful swelling is not constant, but is aggravated by the dependent position, and it does not lead to destruction of tissue.

Raynaud's disease is not dependent on position; it begins as an ischæmia; pain is inconstant; there is anæsthesia, analgesia, and lowered temperature; and there is a local, and usually a symmetrical, gangrene. But Voorhees, in a recent paper, maintains that the two diseases are merely different phases of the same condition.

The disease has some resemblance to neuritis and injury to the nerves, but there are not the objective symptoms, such as paralysis, anæsthesia, and trophic changes; when these occur it is doubtful whether the case is one of pure erythromelalgia.

It is well to recall, however, that some of the above data for differentiation are not entirely reliable, for pallor, ischæmia, sensory changes, etc., have been noted in cases closely resembling erythromelalgia, and gangrene has even followed in some of them, as in one of Mitchell's cases.

The blue œdema of hysteria is usually confined to one limb; it is likely to be associated with loss of power, and it is often non-painful; moreover, the history and other hysterical symptoms are significant. These hysterical cases usually follow trauma.

III. ANGIONEUROTIC ŒDEMA.

This is a sort of pseudo-urticaria, occurring usually in weakened or neurotic persons. It is also called giant urticaria, or Quincke's disease.

Pathology.—The affection is probably of vasomotor origin, although the essential cause and its mode of action are obscure. Several instances are given, as by Osler and by Milroy, of hereditary transmission through five and six generations.

Symptoms.—Localized swellings occur on the skin in various regions. These are somewhat like wheals, or hives, but they are painless, and do not itch. They are at first, as a rule, slightly pale, but flushing soon follows. The subcutaneous tissue is involved in the œdema, and sometimes the mucous membranes are invaded. Thus croupy symptoms have been caused, and death has even been ascribed to angioneurotic œdema of the glottis. Gastro-intestinal symptoms, such as colic and vomiting, are sometimes seen.

Cerebral symptoms are caused by these angioneuroses. Thus we sometimes see flushing of the face and head, palpitation, throbbing in the temples, tinnitus, dimness of vision, confusion, and emotional disorder. Such cases are not uncommon in neurasthenic and hysterical patients.

Diagnosis.—The disease looks a little like urticaria, but there is no burning or itching. Some authors, however, incline to identify the two, and claim that itching occurs, but this is doubtful of real cases.

When marked cerebral symptoms occur, as in the cases of "rush of blood to the head," there may be some remote resemblance to epilepsy, but the history of the case and the well-marked vasomotor disorder should prevent error.

Similar phenomena are sometimes seen in old alcoholic cases, and in exophthalmic goitre. Purpura, with urticaria and hæmoglobinuria, is doubtless a different disease.

IV. HEMIFACIAL ATROPHY.

This is an affection in which, as the name signifies, atrophic changes on one side of the face, brow, and skull are seen.

Pathology.—Autopsies have been rare; probably the most instructive was one by Mendel, in which he found neuritis of the left trigeminal nerve, most marked in the second division, with atrophy of the descending root and of the *substantia ferruginea.* Homen found a dural tumor compressing the Gasserian ganglion. The disease is a trophic one, probably depending on changes in the fifth nerve, although operations on the Gasserian ganglion have not fully sustained this theory. Some ascribe it to changes in the sympathetic nerves. The subject is still obscure.

Symptoms.—The changes are in the skin, subcutaneous tissue, and bone. The muscles supplied by the seventh nerve escape.

The initial symptom is changed color in a limited area of the skin; this may spread until it involves the entire side of the face. The atrophy of the skin follows, but it may be very slow. Atrophy of the bones is more rare. The color of the hair of the beard, eyebrows, and head is sometimes changed to white. The affected part does not sweat in some cases; and some writers have observed lowered temperature ($\frac{6}{10}$ of a degree). Taste is sometimes involved; hearing seldom; sight never. The facial muscles are not paralyzed, but the masticatory muscles, supplied by the motor branch of the fifth nerve, have been found wasted, according to some authors.. Twitching of the facial muscles is sometimes seen.

The above are the essential features of the disease. In some cases trigeminal neuralgia is present, and there may be paræsthesia, as numbness and tingling, but objective anæsthesia is uncommon.

In a few cases hemifacial atrophy has appeared in the chronic insane; and in a few instances a hemilingual atrophy has appeared along with a hemiplegia.

Diagnosis.—This presents no difficulty. The appearance of the atrophied tissue is unmistakable.

In morphœa the bones are not involved; and in scleroderma, according to Duhring, there is an hypertrophy rather than atrophy, and the tissue is hardened.

In Bell's palsy the facial muscles are atrophied, and there is paralysis with reactions of degeneration, but the skin and bones are not affected. Turner says that the faradic excitability of the facial muscles in hemiatrophy is increased and that this arises from lessened resistance owing to the disappearance of the subcutaneous fat. The history in Bell's palsy is usually clear the affection is acute; and the muscles alone are involved.

So, too, in progressive muscular atrophy and in muscular dystrophy, when the face is invaded, the muscles alone are involved, the affection is bilateral, and the course and appearance are entirely different.

V. OSTEITIS DEFORMANS: PAGET'S DISEASE.

Definition.—A rare disease of the bones, characterized by the absorption and new formation of bone tissue, which remains for a time uncalcified and leads to curvatures, over-growth and other deformities of the skeleton.

Up to 1900 only 66 undoubted cases had been reported and in 1902 only 11 cases had been observed in North America.

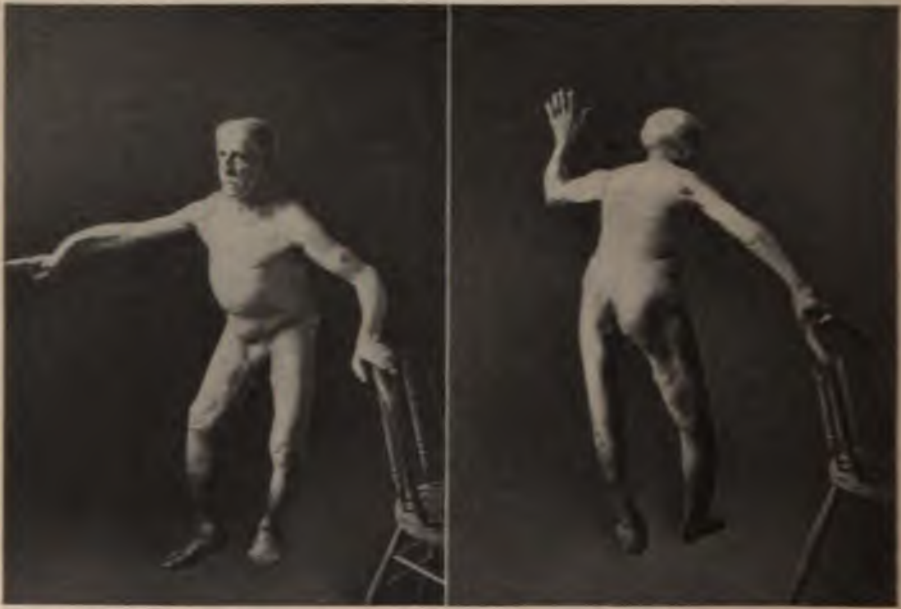

FIG. 405.—Osteitis deformans.—Jefferson Hospital.

Etiology. — PREDISPOSING INFLUENCES. — Both sexes are liable to the disease. Of the reported cases about twice as many occurred in males as in females. Age is more important. The first symptoms have commonly shown themselves after the fortieth year. The onset of the disease in one instance occurred about the age of twenty-one. As the disease is chronic and progressive and in most instances unattended by subjective symptoms, the cases have usually come under observation at a period more or less remote from the time of onset. In the majority of instances the first symptoms have been observed in middle rather than in advanced life. The influence of heredity is uncertain. In three instances, however, cases have occurred in two members of the same family. Occupation is altogether without influence in predisposing to the disease.

Association with Other Diseases.—It has been assumed that there is some causal relation between lesions of the nervous system and osteitis deformans. No constant relationship has, however, been established and in the greater number of the cases there has been an entire absence of phenomena indicating nervous or visceral disease. Arthritis deformans has in a few instances coexisted with osteitis deformans. This association appears to have been a coincidence and there is no reason to believe that there is any causal relationship or interdependence between the two affections.

The EXCITING CAUSE of the disease remains wholly unknown.

Symptoms.—The manifestations of osteitis deformans are chiefly objective. The onset is insidious, sometimes involving a single bone or a limited number of bones, but in the course of time showing a tendency to symmetrical involvement of the skeleton. Individuals suffering from this disease present as the result of definite skeletal deformities a remarkable resemblance to each other. There is thickening of the bones of the skull and an alteration in its shape. The calvarium becomes flattened, the brow broad, the parietal regions prominent. The general circumference is increased so that the patient has to wear a larger hat than formerly. The face is irregu-arly egg-shaped or triangular, the base being at the forehead and the apex at the chin. The head is carried forward with the chin sunk upon the reast. There is cervicodorsal kyphosis, flattening of the thorax at the pper part, spreading at its base, the abdomen is diamond-shaped and ows a deep transverse sulcus, the hips are increased in width and the wer extremities markedly curved outward and forward, while owing to e decrease in height amounting in some instances to several inches, the ms appear disproportionately long—like those of the anthropoid apes.

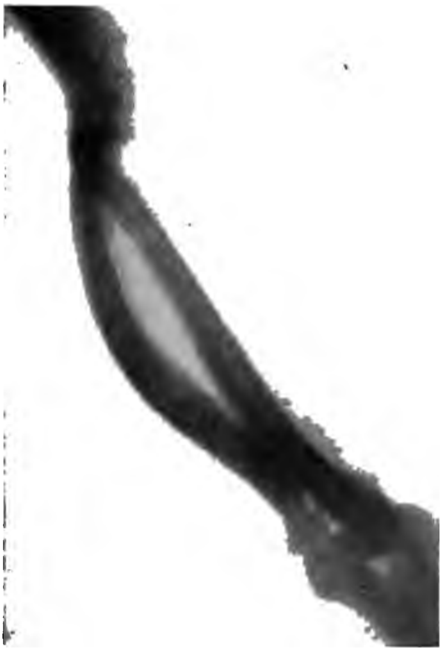

Fig. 406.—Skiagram showing deformity of radius and ulna.

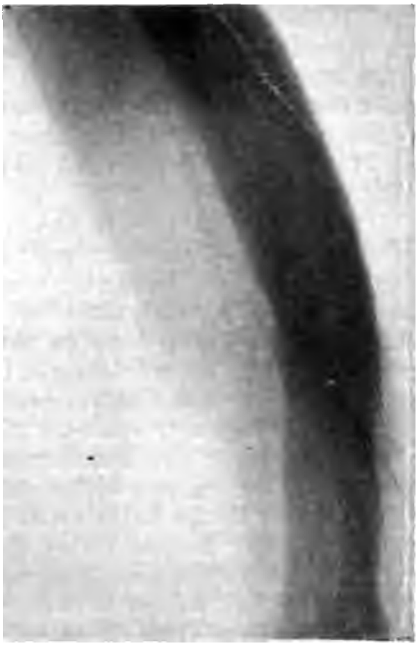

Fig. 407.—Skiagram showing deformity of tibia and fibula.

pulmonary affections. This deforming affection of the osseous system bears only a remote resemblance to osteitis deformans. The absence of changes in the cranium, the escape of the shafts of the long bones and the constant presence of intrathoracic lesions constitute points of radical difference.

Prognosis.—Osteitis deformans is slowly progressive and requires a number of years, varying from five to fifteen, to attain its maximum development. It has little influence upon the general health and is not a direct cause of death.

VI. ACHONDROPLASIA: CHONDRODYSTROPHIA FŒTALIS.

Definition.—A form of dwarfism characterized by micromyelia and macrocephalia, the trunk being of about the normal size. This remarkable disease of fetal life was first described by Virchow (1858) who regarded it as a form of fetal or congenital rhachitis. Parrot (1878) suggested the name *achondroplasia.* The most satisfactory account is that of P. Marie (1900).

Etiology.—Predisposing influences and the exciting cause are alike wholly unknown.

Pathology.—There is a dystrophy of the epiphyseal cartilages from the earliest period of osteogenesis. The growth of the bones of the extremities at the epiphyseal cartilages is defective or arrested so that they do not normally increase in length. The bones which develop from a primitive membranous matrix, without passing through the stage of cartilaginous formation, as the clavicle, ribs and certain of the cranial bones, are not involved in the dystrophy, a fact which accounts for the great difference in the development of the extremities and that of the trunk and head. The dystrophy has been ascribed to the action of an unknown toxic agent circulating in the blood to which the epiphyseal cartilage substance is peculiarly obnoxious—perhaps a vitiated internal secretion having to do with the regulation of the normal growth of the body.

Clinical Characteristics.—The disease is essentially an affection of fetal life. By far the greater number of cases die *in utero* or shortly after birth. Most of those who survive die in childhood. A few reach adult life. In a limited number of cases the disease has appeared to commence shortly after birth. These constitute the dwarfs known as micromelic.

The deformities are characteristic. Both the upper and the lower extremities are symmetrically shortened. The arm is shorter than the forearm; the thigh shorter than the leg. The lower limbs are bent, an exaggeration of normal curves. The development of the trunk is about normal. The enlargement of the head is characteristic. It may suggest hydrocephalus. The head is not only large; it is also rounded with exaggerated parietal and frontal bosses. The features are large and coarse, especially the nose, which is depressed at its root and rounded at its point with flaring nostrils. There is lordosis affecting the lumbar vertebræ and in females contraction of the pelvis. The hand is small and square, the fingers short, of about the same length and spreading—a deformity which has been designated the *trident hand.* The scapulæ are short. The muscles are usually well developed. The genital organs are normal. Many of the subjects of this disease are obese. The mental powers are as a rule good; in some of the reported cases defective. Comby thinks it probable that the court dwarfs were achondroplasiacs as they are apt to be very intelligent.

The direct diagnosis of achondroplasia is unattended with difficulty.

It is to be differentiated from *rickets*, of which it was at one time regarded as a prenatal type by the skeletal changes which do not involve the trunk and are wholly dissimilar in the extremities and from cretinism, of which it was formerly considered a fetal form by the higher degree of intelligence, the peculiar deformities of the long bones which are shortened and of relatively great thickness in the shaft and by the fact that improvement does not follow the administration of thyroid extract.

XV.

THE DIAGNOSIS OF DISEASES OF THE MUSCLES.

I. MYOSITIS.

Definition.—Inflammation of the skeletal muscles.

Primary inflammation of the muscles may occur as an acute, subacute or chronic disease. The following forms are recognized:

1. Infectious Myositis.—This form is very common in Japan. It may affect one or many muscles. Of 32 cases studied bacteriologically by Miyake, 2 yielded negative results; 27 yielded a pure culture of the staphylococcus pyogenes aureus; 2 the cultures of the albus and aureus and 1 the streptococcus. The onset is sudden. There is fever often high, great depression, induration of the muscles followed by abscesses and, unless the pus is completely evacuated, sepsis.

2. Dermatomyositis.—Many muscles are usually affected. The overlying skin is inflamed and œdematous. The muscles are tender, painful and stiff and upon palpation feel inelastic and dough-like. The affection progressively involves new groups of muscles. Upon post-mortem examination, the muscle substance is firm but fragile and the seat of serous infiltration, fatty degeneration and proliferation of the interstitial connective tissue. This form of myositis resembles trichinosis, from which it cannot be differentiated except by the microscopical examination of a bit of the affected muscle.

3. Neuromyositis.—This name was suggested by Senator for a group of cases characterized by marked disorders of sensation.

4. Polymyositis Hæmorrhagica.—These cases present the clinical phenomena of dermatomyositis but to these are added grave circulatory symptoms due to the implication of the myocardium in the process. The muscles show more or less extensive interstitial hemorrhages. This variety is extremely rare.

5. Myositis Ossificans.—Two forms are described, a local and a progressive. The first affects a limited muscle mass and is stationary. The second develops early in life, advances by irregularly recurring attacks and progressively involves many muscle groups. The number

of boys who suffer from this affection appears to be five times as great as that of girls. It is commonly first recognized in late infancy or childhood. The muscles of the neck, trunk and upper extremities are usually involved, the hands and lower extremities escaping. The masseters are sometimes affected. The other muscles of the face escape. The process by which the ossification occurs consists in cyanotic congestion in a localized area of the tissues which upon pressure are found to be of doughy consistency and very painful. After repeated attacks, bony nodules are found within the muscles at the seat of the trouble and exostoses develop. Impairment of function follows and muscular movements are greatly restricted. The general health is not usually disturbed. There is no fever. Developmental defects are common. They include microdactylia, ankylosis of the interphalangeal articulations, hallus valgus and other deformities of the great toe and malformations of the genital organs. Warren Walker, to whose article I am indebted for some of the above facts, has recently reported a most interesting case. The disease is very rare.

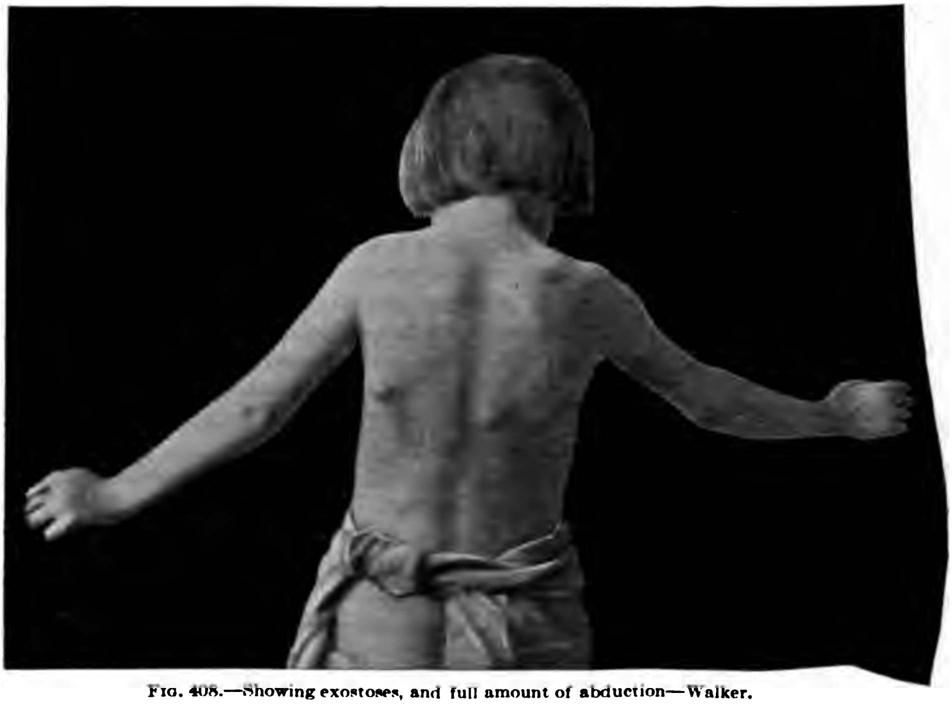

FIG. 408.—Showing exostoses, and full amount of abduction—Walker.

II. THE MYOPATHIES.

By this term we understand essential changes in the muscles, marked by atrophy and loss of power, and not depending on changes in the central or peripheral nervous system.

Pathology.—In the pure myopathies the changes are confined to the muscles. There is atrophy of the muscular fibres, sometimes associated with or preceded by swelling in some of them. The nuclei of the fibres may increase, and there is proliferation of the fibrous tissue. Fatty deposits occur, and ultimately the muscular fibres show splitting and longitudinal striations, with the formation of vacuoles. The cause is not definitely known, but there is probably a congenital defect of development or of nutrition of the essential elements of the muscles. As a rule the spinal cord and nerves are normal, although is some cases the motor nerve-endings are involved; and a question arises whether indeed there is not a juvenile type which is not a pure myopathy, but depends upon a peripheral neuritits or nerve dystrophy. The form known as the Charcot-Marie-Tooth type is apparently not a pure myopathy at all, but rather a muscular atrophy dependent upon a degeneration of the peripheral nerves—a so-called primary neuritic atrophy, as shown in autopsies made by Virchow and others. A well-marked type is that in which some of the muscles undergo an increase in bulk, but this is a pseudohypertrophy, in which the over-growth is not in the muscular elements proper. Various other types of the affection occur, which are rather clinical than anatomical; in fact, these types are probably only varieties of the same degenerative process. It is proper to note that some observers believe that most of these cases are dependent upon a dystrophy of the nerve-endings. The pathology of the myopathies is still somewhat obscure.

Symptoms.—The onset usually occurs early in life, and may be hereditary or familial. The trunk muscles are affected early in the disease, and the arms and thighs also suffer. There is an absence of fibrillary twitching in the muscles, and the complete reactions of degeneration are not seen. Much refinement of description has been indulged in. The commonest types are as follows:

A juvenile type, or scapulohumeral form, which appears in children, and in which the muscles of the shoulder and arm are first affected.

The facio-scapulo-humeral type, in which, as the name implies, the muscles of the face, shoulder and arm are especially involved. It differs little from the former, chiefly, in fact, in the affection of the face.

The peroneal or leg type, in which the lower extremities, especially the peroneal muscles, are implicated.

Pseudomuscular hypertrophy, in which some of the muscles, especially those of the calf, are enlarged, while others, especially of the back and arms, are atrophied. The enlargement of the calf muscles is not a true hypertrophy, but is due largely to a deposit of fat.

The Charcot-Marie-Tooth type, or primary neuritic atrophy, in which the distal muscles of the arms and legs especially suffer, and in which there is fibrillation along with the atrophy, often with preservation of the knee-jerks, without contractures and occurring as a familial affection. As already said, this type is not a pure myopathy.

The results of these various atrophies, or dystrophies, are various forms of paralysis. Wasting may become extreme, with consequent complete loss of power. The trunk muscles are so affected in some cases that extreme lordosis occurs. When the face is attacked we see the so-called "myopathic face," in which the oral muscles are especially involved. The muscles of mastication and deglutition, as well as of the eye, are not affected. When the arm and shoulder muscles are involved there results great weakness in the upper extremities, with some deformities of the hands. In the lower limbs the thigh muscles may be so wasted and weakened that the patient cannot stand, much less walk; and in the legs the destruction of the muscles causes various forms of club-foot. In extreme cases no particular "type" is presented, but the patient has extensive atrophy of all the limbs and of many of the trunk muscles, and may become a so-called "living skeleton." In the pseudohypertrophic form the enlarged calves stand out conspicuously, and are hard and brawny to the touch; but the patient usually presents wasting of the arms and trunk. He shows a special difficulty in rising from the floor, climbing with his two hands, as it were, up his legs. In the myopathies there is no involvement of sensation, nor of the bladder or bowel, nor of the mental faculties. The affection is chronic and incurable.

Diagnosis.—The student or practitioner need not be so much concerned to make out "types" or varieties as to establish a general diagnosis of muscular dystrophy. For this purpose the history of the case is first considered, and then the peculiar atrophy, associated perhaps in some muscles with pseudohypertrophy, without fibrillation, reactions of degeneration, sensory changes or incontinence. These points, some or all, serve to distinguish the affection from the progressive muscular atrophy of adults, or from acute multiple neuritis, or from myelitis as the case may be. From infantile paralysis or acute anterior poliomyelitis the disease is distinguished by the slow onset, its progressive character and wide extent, the preservation of the electrical responses to an advanced stage, and the general history.

The primary neuritic atrophy of Charcot, Marie, and Tooth presents some special points which give it a place almost unique. It somewhat resembles progressive muscular atrophy, also multiple neuritis. It is distinguished, however, by being often a familial affection, beginning rather early in life, very chronic, and the wasted muscles are the distal groups of the extremities. Fibrillation is common and foot-drop is seen. The knee-jerks may be preserved, and sensory symptoms are wanting.

In the cerebral palsies of children we see hemiplegia, diplegia, paraplegia and rarely, monoplegia. The paralysis is spastic, with exaggerated tendon reflexes and without true muscular atrophy; and in some cases there are cerebral symptoms, as epilepsy and various grades of idiocy.

III. THOMSEN'S DISEASE: MYOTONIA.

This is a bizarre affection, seen by but few persons, and described by most writers in terms exactly alike. It was first reported by a Dr. Thomsen, for whom it is named, and in whose family it seems to have prevailed to an unprecedented extent. As it is claimed to be congenital, hereditary, and familial it is sometimes called *myotonia congenita*.

Pathology.—The disease has no pathology that anyone has yet discovered. But little significance is to be attached to such slight changes as are reported by Erb, Dejerine, and a few others, and which seem to consist in nothing but a little increase in size of the muscle fibres.

Symptoms.—Muscular rigidity, or cramp, occurring on voluntary movements or attempts at such movements, is the chief symptom. A condition of tonic spasm sets in when the patient, especially after a long rest, attempts to use certain muscles or groups of muscles. These spasms do not seem painful; at least, pain is not insisted on by some writers. In some cases a few muscles only are involved; in others almost the whole musculature is thrown into tonic spasms. There is no loss of consciousness.

Some effort has been made to show that the muscles present a special or so-called "myotonic reaction" to electricity. This consists in a state, more or less transient, of slight contracture and tonic spasm, which varies somewhat with the kind of current used and with its strength. With faradism there is a tonic contracture of long duration; with galvanism only labile currents produce contractures, which are sluggish in character. There is increase of mechanical irritability, shown by the muscle giving a sluggish tonic contraction on percussion, especially at the point struck.

Diagnosis.—This is not difficult, for the disease is like none other. An attempt should always be made to exclude hysteria.

IV. PARAMYOCLONUS MULTIPLEX.

Friedreich, in 1881, described a disease which has been given this cumbersome name. Few seem to have seen it, or anything like it, and some good authorities deny its existence. It has no recognized cause or morbid anatomy.

Symptoms.—As described by most writer s,the disease is manifested by a series of short, quick, irregular, shock-like contractions of the muscles of the extremities and trunk. The face usually escapes. The contractions are not unlike those caused by electric shocks. The intelligence, the sensory system, and the sphincters are not involved.

Diagnosis.—Hysteria is not to be ignored. Lloyd refers to aggravated cases of hysterical tremor, with coarse irregular jerkings, which suggested to him the picture of paramyoclonus as drawn in some books. These are probably the patients who get well.

The *chorea electrica* of Bergeron, Henoch and others is probably a true chorea, although cases of it have been described as paramyoclonus multiplex. Dubini's chorea, also called electrical, is very rare, and is said to be associated with fever, muscular atrophy, and paralysis. It is an infectious disease, seen mostly in Italy.

V. MYASTHENIA GRAVIS.

The disease to which Jolly gave this name may be said to be still on trial. Both clinically and pathologically it is as yet a subject of dispute. It may be defined as a syndrome in which there is rapid exhaustion of the muscles supplied by the bulb, as well as of some of the eye muscles and muscles of the extremities. The tendency is towards a fatal termination. The disease is also called *asthenic bulbar palsy.*

Pathology.—Almost by unanimous consent the Germans, who have done most to secure the recognition of this syndrome, declare that the anatomical examinations in the brain and cord are negative. Maier claimed to have found alterations in the anterior roots of the spinal nerves, and Marinesco and Widal reported changes in the ganglion cells, but Oppenheim denies the validity of these findings. In America, studies have been made by Hun, Burr and McCarthy, and others, and there is a disposition to see changes in the thymus gland and lymphoid infiltration in the muscles as the true causes of the disease. The subject is still far from settled.

Symptoms.—Rapid exhaustion of certain muscles or muscle groups seems to be the cardinal symptom. The patient may begin using the muscles with normal vigor, but they rapidly exhaust, sometimes with alarming results. Thus there occur dysarthria, dysphonia, dysphagia, dyspnœa, ptosis, in short, the evidences of paralysis of muscles supplied by the 3d, 5th, 7th, 10th, and 12th nerves. Along with this the extremities are often involved, and the respiratory muscles weakened, so that the prostration may be extreme and dangerous. These attacks may be paroxysmal, excited especially by voluntary use, and in fact the progress of the disease is often by stages.

Jolly observed the "myasthenic reaction" to faradic stimulation, *i.e.*, the rapid exhaustion of the muscles by the faradic current. This reaction is claimed by some to be pathognomonic. There is no muscular atrophy; no fibrillation; no involvement of sensations; no optic atrophy; no paralysis of the bladder or bowel; no abolition of the tendon reflexes (as a rule); no affection of consciousness. Hun's patient, however, had attacks of extreme weakness, or collapse, in which the mind was not clear and the heart's action was depressed. The attacks resembled *angina sine dolore*, or even heart block.

Cases vary in the distribution of the symptoms. In some patients bulbar symptoms are most marked; in others exhaustion is more wide-spread. Some cases continue for years, and the disease may have marked remissions and exacerbations. A fatal result is to be apprehended.

Diagnosis.—The disease resembles Landry's paralysis, except in its tendency to remission and in the absence of evidence of organic central disease. It is probable, however, that some reported cases are allied to, if not identical with, Landry's disease.

In acute anterior poliomyelitis the evidence of organic central disease is clear; as for instance, a flaccid paralysis with loss of the tendon reflexes and with the reactions of degeneration. The disease is ushered in as an acute febrile affection; it is not one of remissions and exacerbations, and the paralysis is obstinately located, as a rule, in one limb and even in one set of muscles. Bulbar symptoms are not common, although not unnoted especially in epidemic anterior poliomyelitis.

From true bulbar palsy the asthenic form is distinguished especially by the history and the absence of evidence of nuclear disease. In the former there is an insidious onset, a slow course, and the affected muscles slowly waste, and present fibrillation and gradual loss of power.

Hysteria may closely simulate myasthenia gravis. The exhaustion and the myasthenic reaction to faradism should distinguish the two affections. Bulbar symptoms are rare, although there may be aphonia, globus, œsophagismus, and retching. Hysterical ptosis is not unheard of.

It is necessary to utter a warning against placing too much confidence in the exhaustion symptom. Something very like it can be seen in neurasthenia; and, in fact, a weakened muscle from whatever cause (as in neuritis or dystrophy) exhausts rapidly on being used.[1]

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[1] See Campbell and Bramwell for a critical digest of myasthenia gravis, Brain, 1900.

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